Annotated Bibliography on Diamhoeal Diseases:

Review Articles and Selective Studies



Annotated Bibliography on Diarrhoeal Diseases: Review Articles and Selective Studies



INTERNATIONAL CENTRE FOR DIARRHOEAL DISEASE RESEARCH, BANGLADESH G P O BOX 128, DHAKA 1000 BANGLADESH EDITOR - IN - CHIEF:

COMPILATION AND DOCUMENTATION:

Prof Roger Eeckels

M Shamsul Islam Khan, Malik M Abdul Quader, M Motasem Ali and M M Hassan

MANAGING EDITOR:

PRODUCTION:

M Shamsul Islam Khan

Cover design: Asem Ansari

EDITORS:

Publication and printing:

Arifuzzaman Khan Nishat Chowdhury Hasan S Ahmed and M Nurul Huda

Manuscript typing:

ABSTRACTORS:

M Mahfuzul Hassan and

Ekramul Hassan

Iftekharul Islam Mahua Khair

Price

a SAARC countries, except Bangladesh ... US\$ 25.-*
(Bhutan, India, Maldives, Nepal,
Pakistan, Sri Lanka) and Myanmar (Burma)

b Bangladesh ... Tk 200.-

c Other countries ... US\$ 40.-*

All payments should be made in the form of cheque or bank draft, drawn on any bank in Bangladesh, USA, or UK, in favour of the "International Centre for Diarrhoeal Disease Research, Bangladesh". All correspondence regarding price should be addressed to: Managing Editor, Specialized Bibliography Series; ICDDR,B-DISC, G P O Box 128, Dhaka 1000, Bangladesh.

Copyright © 1989.

International Centre for Diarrhoeal Disease Research, Bangladesh G P O Box 128, Dhaka 1000, Bangladesh.

Cable: Cholera Dhaka; Telex: 675612 ICDD BJ; Fax: 880 - 2 - 883116

Telephone: (880-2) - 600171 through (880-2) - 600178.

^{*}Price includes air mail postage cost.

PREFACE

The Specialized Bibliography Series is a part of the larger effort to facilitate exchange of information and to establish an information network in the field of diarrhoeal diseases — an effort being carried out by the Diarrhoeal Diseases Information Services Centre (DISC) of the ICDDR,B. The present issue, the thirteenth of the series, includes citations of 665 papers (385 abstracted) on review articles and selective studies on diarrhoeal diseases.

The "Annotated Bibliography on Diarrhocal Diseases: Review Articles and Selective Studies" has been prepared as per the recommendation made in the Workshop on Diarrhocal Disease Information and Documentation Service, held in Bangkok, on 12 June 1985, organised by the ICDDR,B, in conjunction with the 3rd Asian Conference on Diarrhocal Diseases.

This is a selective bibliography on the topic. The bibliography was compiled from the resource available at the DISC of ICDDR,B, and it is possible that inadvertent omissions may have occurred.

We hope the present bibliography will contribute towards generating greater interest and awareness in this field, and will facilitate user access to existing knowledge. Most of the published papers cited in this bibliography are available from the DISC of ICDDR,B to interested persons/organisations. We will consider this attempt successful if the bibliography helps diarrhoeal disease researchers and practitioners' interest.

Prof D Habte

Director International Centre for Diarrhocal Disease Research, Bangladesh

ACKNOWLEDGEMENTS

The activities, services and programmes of the Diarrhoeal Diseases Information Services Centre (DISC) are supported by the International Development Research Centre (IDRC), Canada, Swiss Development Co – operation (SDC), Switzerland, and the International Centre for Diarrhoeal Disease Research, Bangladesh (ICDDR,B). ICDDR,B is supported by countries and agencies which share its concern about the impact of diarrhoeal diseases on the developing world. Current major donors giving assistance to ICDDR,B are: the Aga Khan Foundation, Arab Gulf Fund, Australia, Bangladesh, Belgium, Canadian International Development Agency (CIDA), Canadian International Development Research Centre (IDRC), Danish International Development Agency (DANIDA), France, the Ford Founda – tion, Japan, the Netherlands, Norwegian Agency for International Development (NORAD), SAREC (Sweden), Swiss Development Co – operation (SDC), United Kingdom, United Nations Development Programme (UNDP), United Nations Children's Fund (UNICEF), United Nations Capital Development Fund (UNCDF), United States Agency for Interna – tional Development (USAID), World Health Organization (WHO), and World University Services of Canada (WUSC).

Publication of this bibliography was made possible by a special grant from the International Development Research Centre, Canada.

CONTENTS

User's Guide	•••	•••	***	i
Bibliography			•••	1
Subject Section	***	•••		1
Acquired Immunodeficiency	Syndrome			1
Adenovirus and Adenovirus	Infections			1
Aeromonas	***	•••	•••	1
Aluminium and Decontamir	nation		***	2
Amebiasis			•••	1 2 2 3 3 3 3 5 5 6 6
Antacids	•••	***	***	3
Anthelmintics	***	•••	***	3
Antibodies, Monoclonal	***		***	3
Antidiarrheal, Antimicrobial	and Antis	secretory A	gents	3
Antigens, Bacterial			***	ž
Antigens, Immune Response	e	•••		Ž
Antigens, Protozoan	•••	***	***	Ď
Antitoxins	***	***	***	6
Arsenic	***	***		6
Arthritis			***	6
Ascariasis	***		***	6 6 7 7 7 8 9
Astrovirus and Astrovirus In	nfections	•••	***	7
Bacillus subtilis	•••	***	144	7
Bacteria and Bacterial Infec	ctions	***	***	7
Bacterial Adhesion			•••	8
Bacterial Proteins	***		•••	9
Bacterial Toxins	•••	***		9
Bicarbonates	•••	•••	•••	9
Bile Acids and Salts			***	10
Biometry				10
Biopsy		***	•••	10
Blind Loop Syndrome	•••			10
Breast Feeding	•••	***		10
Caloric Intake		***	•••	10
Campylobacter and Campylo	o <i>bacter</i> Info		***	11
Carbohydrates			•••	12
Cataract		***	•••	12
Celiac Disease		***	***	12
Chemotactic Factors		***		12
Cholera	•••	***		12
Drug Therapy	•••	***	•••	13
History	•••			13
Immunology	•••	.,,	•••	13
Microbiology	•••			13
Occurrence	•••	•••	***	13
Oral Therapy	•••	•••		14
Physiopathology	•••	•••	•••	14
Prevention and Control	•••	•••		15
Transmission		***		15

Cholera Toxin				16
Cholera Vaccine		•••	•••	16
Chromatography, Gas	***	***	***	
Clostridium		***	***	17
Colitis and Enterocolitis	***	•••	1**	17
Colonic Diseases, Function	no1	***	***	17
Communicable Diseases	liai	***	н	19
	***	***	***	20
Community Participation	•••	144	***	20
Copper	***	***		20
Coronaviridae	***	***	***	20
Cost – benefit Analysis				20
Crohn Disease	111	٠,-		21
Cryptosporidium and Crypt	osporidiosis		***	21
Cytomegatoviruses	***		111	22
Cytotoxins	***	.,,	***	22
Dairy Products			***	22
Dehydration	•••	•••	•••	22
Diabetes Mellitus	***		•••	23
Diagnosis, Laboratory		•••	•••	23
Diarrhea	•••	•••	***	24
Chemically Induced	***	•••	***	
Complications	•	***	***	25
Congenital	***			25
Diagnosis	***	***	144	26
Diat Theren	***	***	•••	26
Diet Therapy	***		***	26
Drug Therapy	•••	***		27
Etiology		***	•••	28
Fluid Therapy	***		***	30
Immunology	***	***	***	30
Metabolism	•••	***	***	31
Microbiology	***	***	***	32
Mortality	***			32
Occurrence			•••	33
Oral Therapy				33
Parasitology	•••	•••	***	35
Pathology	•••	,	144	35
Physiopathology		***	1**	
Prevention and Control	***	•••	•••	35
Therapy	•••	***	***	39
Transmission	***	***	***	41
Veterinary	***	*-*	•••	41
Diggrhag Agusa	***	***	***	41
Diarrhea, Acute	•••	***	•••	42
Complications	***	***	***	42
Diagnosis	***	***	***	42
Diet Therapy	***	***	***	43
Drug Therapy	•••	•••	***	43
Etiology Fluid Therapy		,	414	43
Fluid Therapy		1	•••	44
Metadonsm			***	44
Microbiology	•••		***	44
Mortality —				44
Oral Therapy	•••	***	•••	44
Physiopathology		•••	***	45
Prevention and Control	•••	•••	*14	45
Therapy	***	***	***	45 46
<i></i>	•••	***	•••	40

Diarrhea, Chronic	***	***	•••	46
Complications	***	***	***	47
Diagnosis	•••	***	•••	47
Diet Therapy	***	***	•••	47
Drug Therapy	•••	•••	***	48
Etiology	•••		***	48
Fluid Therapy	•••		•••	48
Metabolism			•••	48
Oral Therapy	•••	***	•••	48
Physiopathology		•••		49
Prevention and Control		***		49
Therapy				49
Veterinary	***			50
Diarrhea, Infantile	•	•••		50
Chemically Induced	•••	•••	•••	51
Complications	•••		•••	51
Diagnosis	•••	.,,	***	51
	•••	***	***	51
Diet Therapy	***	•••		52
Drug Therapy	***	***		52
Etiology		***	***	52
Fluid Therapy	141	***	***	52
Metabolism	***	***	н1	53
Microbiology	***	***	144	53
Mortality	445	***	144	53
Occurrence	•••	***	***	54
Oral Therapy	***	***	144	54
Pathology		***	***	
Physiopathology	•••	•••	***	54
Prevention and Control	•••	•••	•••	55
Therapy	***	***	***	55
Dientamoeba fragilis	***	144	***	55
Diets and Diet Therapy	***	***	***	55
Digestion				56
Digestive System Diseases	144		***	57
Disaccharidases	***	***		57
Disease		***		57
Disease Models, Animal		***		57
Disease Outbreaks and Tra	nsmission			58
DNA, Bacterial	***	***	***	59
Doxycycline	***	***	***	59
Drug Resistance, Microbial			***	59
Drug - Evaluation, Interac	ctions and	Therapeutic	: Use	59
Dysentery, Amebic	,,,	*		61
Dysentery, Bacillary	***	***	144	62
Eicosanoic Acids		,.,	***	63
Electrolytes		***		63
Endotoxins	***	***	***	64
Entamoeba histolytica				64
Enteral Feeding				64
Enteritis	*		41-	64
Enterobacteriaceae		•••		65
Enterotoxins			***	66
Enteroviruses	***	•••		66
Environmental Pollution	***		•••	66
	***	•••	***	

Eosinophils		***	•••	66
Epidemiology and Epidemio	ologic Metho	ds	•••	66
Escherichia coli and Escheri	chia coli Infe	ections		69
Escherichia coli. Enteropath	ogenic	•••		70
Escherichia coli, Enterotoxia	genic			71
Feeding Behavior				71
Fiber				71
Fluid Therapy		•••	***	71
Food Hypersensitivity	***	***		72
Fungi	•••			72
Gastric Emptying			***	72
Gastritis and Peptic Ulcer	•••	•••		72
Gastroenteritis		•••	***	72
Gastrointestinal and Colonic	c Motility		144	73
Gastrointestinal Diseases			***	74
Genetics, Microbial	***	***	***	75
Giardia and Giardiasis		•••	•••	75
Growth and Growth Disord	i	***	***	76
Health, Health Education, 1	icis Haadwachina	and Ungions	***	76
Helminths and Helminthiasi	iranuwasining		* ***	
Hemolysins	15	***	•••	77
	•••	•••	144	77
Hepatitis, Viral	•••	***	•••	77
Hormones	***	***	***	78
Host - parasite Relations		•••	***	78
Hybridization Hypernation	***	***		78
Hypernatremia	***	•••	***	78
Ileostomy	***	•••	***	78
Immune Response and Tole	erance	•••	•••	79
Immunity	***	•••	***	79
Immunoassay	***	***	•••	80
Immunologic Deficiency Syn	idromes	***	***	80
Impact and Evaluation Stud	ies	***	***	80
Infant, Low Birth Weight	***	***	***	81
Infant Mortality	***	***	***	81
Infant Nutrition and Food	***	***		81
Infant Nutrition Disorders		***	***	82
Infections	***	***	***	83
Inflammation	1**	***	***	83
Inflammatory Bowel Disease	;		***	84
Interventions			***	84
Intestinal Absorption		•••	***	86
Intestinal Diseases	***	***	***	86
Intestinal Diseases, Parasitic				88
Intestinal Secretions			***	88
Ions		•••	•••	89
Iron				89
Islet Cell Tumor	***			89
Isospora belli	***	***		8 9
Knowledge, Attitudes and P	ractice		•••	89
Liver Abscess, Amebic		•••		9ó
Malabsorption Syndromes	***	***	•••	9ŏ
Malignant Carcinoid Syndroi				91
Measles		***	***	91
Membranes	***	•••		92

Metabolism, Inborn Errors		•••		92
Microscopy		***	•••	92
Milk		•••	•••	92
Morbidity and Mortality		•••	***	92
Mycobacterium Infections				93
Narcotics		•••		93
Nematode and Nematode Inf	notions	•••	***	93
	echons		•••	94
Neoplasms	••	***		
Norwalk Agent			***	94
Nutrition, Nutrition Disorders	and Nutrit	ional Requir	ements	94
Oral Rehydration			***	96
Parasites and Parasitic Diseas	es		***	98
Parenteral Hyperalimentation		/	***	99
	••	***	***	99
Plague	••	***	***	99
Plasmide		•••	***	99
Plesiomonas .			•••	100
Primary Health Care .		,,.	***	100
D-autoclassicae				101
Protein - calorie Malnutrition		***	***	101
		***	***	102
Protein - losing Enteropathy		•••		102
Proteins				. = -
Protozoa and Protozoan Infe	ctions	•••	•••	102
		***	***	102
Public Health .		***	***	103
Receptors .		***	***	103
Reiter's Disease .	••	***	•••	103
Risk Factors .		***	***	103
Rotavirus and Rotavirus Infe-	ctions	•••	***	104
Salmonella and Salmonella Ir	ifections			104
Sanitation and Sewage .			•••	105
Cahictacamiasis	••	***		107
Saratimina	••	•••	***	107
Savuelly Transmitted Discosor	••	•••		107
Sexually Transmitted Diseases	8			107
Shigella .			***	7 = 4
		***	***	108
Shigella flexneri .		***		108
	.,	***		109
Socioeconomic and Socioculti	ural Factors		***	109
Spirochaetales .		***	411	109
Splenia Diceases		***	***	109
Starch	**			109
Surgery				109
Thiogulfotae	**	•••	•••	110
Toroviridae		•••		110
Toxins .	**		***	110
		•••	•••	110
Trace Elements .		•••	***	110
Trichostrongyloidiasis		***		
Trichuris and Trichuriasis .		***		110
· I		***		110
Tropical Climate and Disease	es	***	***	111
Typhoid .	·		***	111
Typhoid - paratyphoid Vaccin	es		***	111
Vaccines, Vaccination, Immu	nization and	Vaccine De	evelopment	112
			1	

-

Vasoactive Intestinal Pept	ide		•••	113
Vasoactive Intestinal Pept Vibrio and Vibrio Infection	ns		***	114
Vibrio cholerae	***	,,,	414	114
Vibrio parahaemolyticus	•••			115
Virulence	144	***	117	115
Virus and Viral Diseases			•••	116
Vitamin A Deficiency and	d Blindness.	/Xerophtha	lmia	117
Water - electrolyte Balanc	e and Imba	lance	•••	117
Water Pollution				118
Water Supply	•••	***		119
Weaning and Weaning Ed		***		120
Yersinia and Yersinia Infe	ctions		***	121
Zinc	•		***	121
Zoonoses	***		***	121
Author Section	***			122

.

•

USER'S GUIDE

The Specialized Bibliography Series includes abstracts and citations of currently available literature from sources worldwide.

The bibliography is divided into subject and author sections. In the Subject Section, citations are arranged alphabetically by the first author under specific headings. The citation sometimes is followed by a sign (**), indicating that an abstract of the cited paper appears in the Author Section.

The Author Section contains citations arranged alphabetically by the first author and then by the title of paper. Co-authors' names also appear in alphabetical order along with a cross-reference to the first author (e.g. Abbott SL see Janda JM). This will facilitate a search by co-authors' names.

Efforts have been made to present abstracts with all available information regarding the study's nature and objective, method(s) used, and the major findings and conclusions.

The bibliography is in English. A title in parenthesis indicates that the paper is in another language.

ANNOTATED BIBLIOGRAPHY ON DIARRHOEAL DISEASES: REVIEW ARTICLES AND SELECTIVE STUDIES

SUBJECT SECTION

ACQUIRED IMMUNODEFICIENCY SYNDROME

Diarrhea and malabsorption associated with the acquired immunodeficiency syndrome (AIDS). Nutr Rev 1985 Aug;43(8):235-7

Haverkos HW. Factors associated with the pathogenesis of AIDS. J Infect Dis 1987 Jul; 156(1):251-7

Ho DD, Pomerantz RJ, Kaplan JC. Pathogenesis of infection with human immunodeficiency virus. N Engl J Med 1987 Jul 30;317(5):278-86

Rogers MF. AIDS in children: a review of the clinical, epidemiologic and public health aspects. Pediatr Infect Dis 1985 May-Jun;4(3):230-6

ADENOVIRUS AND ADENOVIRUS INFECTIONS

Albert MJ. Enteric adenoviruses: brief review. Arch Virol 1986;88(1-2):1-17 **

Hyypia T, Pettersson U. Spot hybridization for the detection of adenoviruses and enteroviruses. Clin Lab Med 1985 Sep;5(3):491-501

Wood DJ. Adenovirus gastroenteritis. Br Med J 1988 Jan 23;296(6617):229-30

AEROMONAS

Ewing WH, Hugh R, Johnson JG. Studies on the Aeromonas group. Atlanta, Ga.: U S Department of Health and Human Services, $1981.\ 37$ p.

Gracey M, Burke V. Characteristics of <u>Aeromonas</u> species and their association with human diarrhoeal disease. J Diarrhoeal Dis Res 1986 Jun;4(2):70-3 **

Holmberg SD, Farmer JJ, 3d. <u>Aeromonas hydrophila</u> and <u>Plesiomonas shigelloides</u> as causes of intestinal infections. Rev Infect Dis 1984 Sep-Oct;6(5):633-9 **

Ljungh A, Wadstrom T. Aeromonas and Plesiomonas as possible causes of diarrhoea. Infection 1985 Jul-Aug;13(4):169-73

Ljungh A, Wadstrom T. Aeromonas toxins. Pharmacol Ther 1982;15(3):339-54 **

Ljungh A, Eneroth P, Wadstrom T. Cytotonic enterotoxin from Aeromonas hydrophila. Toxicon 1982;20(4):787-94 **

^{**}indicates an abstract appears with the citation in the author section.

Schoch PE, Cunha BA. Aeromonas. Infect Control 1984 Nov;5(11):542-4 **

Trust TJ, Chipman DC. Clinical involvement of <u>Aeromonas hydrophila</u>. Can Med Assoc J 1979 Apr 21:120(8):942-6 **

von Graevenitz A. <u>Aeromonas</u> and <u>Plesiomonas</u> as agents of diarrhea. <u>In: Ellner PD, ed. Infectious diarrheal diseases;</u> current concepts and <u>laboratory procedures</u>. New York: Marcel, 1984;59-75

von Graevenitz A. Aeromonas and Plesiomonas. In: Lennette EH, Balows A, Hausler WJ, Jr., Truant ∂P , eds. Manual of clinical microbiology. 3d ed. Washington, D.C.: American Society for Microbiology, 1980:220-5

von Graevenitz A, Mensch AH. The genus <u>Aeromonas</u> in human bacteriology: report of 30 cases and review of the literature. N Engl 'J Med 1968 Feb;278 (5): 245-9 **

Wadstrom T. Aeromonas and Plesiomonas as possible causes of diarrhoea. Third Meeting of the Scientific Working Group on Bacterial Enteric Infections, Geneva, 12-14 Sep 1984. Geneva: World Health Organization, 1984. 10 p. (Unpublished document)

ALUMINIUM AND DECONTAMINATION

Ahmad K, Jahan K, Huq I. Decontamination of drinking water by alum for the preparation of oral rehydration solution. Food Nutr Bull 1984 Jun;6(2):54-7 **

Khan MU, Khan MR, Hossain B, Ahmed QS. Alum potash in water to prevent cholera [letter]. Lancet 1984 Nov 3;2(8410):1032 **

AMEBIASIS

Datta T. Intestinal amoebiasis and giardiasis in children. Indian J Pediatr 1985 Mar-Apr;52(415):184-5 **

Diamond LS. Amebiasis: nutritional implications. Rev Infect Dis 1982 Jul-Aug;4(4):843-50 **

Harries J. Amoebaisis: a review. J R Soc Med 1982 Mar;75(3):190-7

Holtan NR. Amebiasis: the ancient scourge is still with us. Postgrad Med 1988 Jun;83(8):65-72 **

Knight R. Hepatic amebiasis. Semin Liver Dis 1984 Nov;4(4):277-92

Martinez-Palomo A. The pathogenesis of amoebiasis. Parasitol Today 1987 Apr; 3(4):111-8

Martinez-Palomo A, Martinez-Baez M. Selective primary health care: strategies for control of disease in the developing world. X. Amebiasis. Rev Infect Dis 1983 Nov-Dec; 5(6):1093-1102 **

Mirelman D. Ameba-bacterium relationship in amebiasis. Microbiol Rev 1987 Jun:51(2):272-84 **

Patterson M, Schoppe LE. The presentation of amoebiasis. Med Clin North Am 1982 May;66(3):689-705 **

Ravdin JI, Guerrant RL. A review of the parasite cellular mechanisms involved in the pathogenesis of amebiasis. Rev Infect Dis 1982 Nov-Dec;4(6): 1185-1207 **

Sepulveda 8. Amebiasis: host-pathogen biology. Rev Infect Dis 1982 Nov-Dec;4 (6):1247-53 **

Sepulveda B. Progress in amebiasis. Scand J Gastroenterol 1982;17(suppl 77): 153-64 **

ANTACIDS

Kivilaakso E. Antacids and bile salts. Scand J Gastroenterol 1982;(suppl 75):

ANTHELMINTICS

Gracey M. Antibiotic and antiparasitic therapy in chronic diarrhea. <u>In:</u> Lebenthal E, ed. Chronic diarrhea in children. New York: Raven Press, 1984:469-76

Prichard RK. Interaction of host physiology and efficacy of antiparasitic drugs. Vet Parasitol 1985 Aug;18(2):103-10

ANTIBODIES, MONOCLONAL

Polin RA. Monoclonal antibodies against microorganisms. Eur J Clin Microbiol 1984 Oct;3(5):387-98

ANTIDIARRHEAL, ANTIMICROBIAL AND ANTISECRETORY AGENTS

Awouters F, Niemegeers CJE, Janssen PAJ. Pharmacology of antidiarrheal drugs. Annu Rev Pharmacol Toxicol 1983;23:279-301 **

Bennet J. A review of antidiarrhoeal compounds. <u>In</u>: Gough D, ed. The control of diarrhoea in clinical practice. London: The Royal Society of Medicine, 1978:1-8 (Royal Society of Medicine International Congress and Symposium series. 5)

Black RE. The prophylaxis and therapy of secretory diarrhea. Med Clin North Am 1982 May; 66(3): 611-21 ·

Bradshaw MJ, Harvey RF. Antidiarrhoeal agents: clinical pharmacology and therapeutic use. Curr Ther 1983 Feb:65-73 **

Coupar IM. Opioid action of the intestine: the importance of the intestinal mucosa. Life Sci 1987 Aug 24;41(8):917-25 **

Danhof IE. Pharmacology, toxicology, clinical efficacy, and adverse effects of calcium polycarbophil, and enteral hydrosorptive agent. Pharmacotherapy 1982 Jan-Feb;2(1):18-28 **

DiJoseph JF, Taylor JA, Mir GN. Alpha-2 receptors in the gastrointestinal system: a new therapeutic approach. Life Sci 1984 Sep 3;35(10):1031-42

DuPont HL, Ericsson CD, Johnson PC, Cabada FJ. Antimicrobial agents in the prevention of travelers' diarrhea. Rev Infect Dis $1986\ May-Jun; 8(suppl\ 2); 5167-71\ **$

DuPont HL, Ericsson CD, Reves RR, Galindo E. Antimicrobial therapy for travelers' diarrhea. Rev Infect Dis 1986 May-Jun;8(suppl 2):S217-22 **

DuPont HL, Steele JH. Use of antimicrobial agents in animal feeds: implications for human health. Rev Infect Dis 1987 May-Jun;9(3):447-60

Fedorak RN, Field M. Antidiarrheal therapy: prospects for new agents. Dig Dis Sci 1987 Feb; 32(2): 195-205 **

Fettman MJ, Rollin RE. Antimicrobial alternatives for calf diarrhea: iron chelators or competitors. J Am Vet Med Assoc 1985 Oct 1;187(7):746-8

Fingl E, Freston JW. Antidiarrhoeal agents and laxatives: changing concepts. Clin Gastroenterol 1979 Jan:8(1):161-85

George WL. Antimicrobial agent-associated colitis and diarrhea: historical background and clinical aspects. Rev Infect Dis 1984 Mar-Apr;6(suppl 1): \$208-13 **

Gotz VP, Rand KH. Medical management of antimicrobial-associated diarrhea and colitis. Pharmacotherapy 1982 Mar-Apr;2(2):100-9 **

Greenough WB, III, Rabbani GH. Antisecretory and antimicrobial drugs for treating diarrhoea. <u>In</u>: Holmgren J, Lindberg A, Mollby R, eds. Development of vaccines and drugs against diarrhea; proceedings of the 11th Nobel Conference, Stockholm, 1985. Lund: Studentilitteratur, 1986:270-7 **

Gyr K. Infectious diarrhoea and gastrointestinal hormones: potential therapeutic implications. Scand J Gastroenterol 1983;18(supp) 84):135-40 **

Joiner KA, Gorbach SL. Antimicrobial therapy of digestive diseases. Clin Gastroenterol $1979 \, \mathrm{Jan}; 8(1):3-35$

Lambert HP. Antimicrobial agents in diarrhoeal disease. Clin Gastroenterol 1979 Sep;8(3):827-33 **

Levine MM. Antimicrobial therapy for infectious diarrhea. Rev Infect Dis 1986 May-Jun;8(suppl 2):S207-16 **

McHenry MC, Weinstein AJ. Antimicrobial drugs and infections in ambulatory patients. Some problems and perspectives. Med Clin North Am 1983 Jan;67(1): 3-16

May JT. Antimicrobial properties and microbial contaminants of breast milk - an update. Aust Paediatr J 1984 Nov:20(4):265-9 **

Murray BE. Resistance of <u>Shigella</u>, <u>Salmonella</u>, and other selected enteric pathogens to antimicrobial agents. Rev Infect Dis 1986 May-Jun;8(suppl 2):S172-81 **

O'Brien TF. Resistance of bacteria to antibacterial agents: report of Task Force 2. Rev Infect Dis 1987 May-Jun:9(suppl 3):S244-60

Ooms L. Alterations in intestinal fluid movement. Scand J Gastroenterol 1983:18(suppl 84):65-77 **

Read NW. Speculations on the role of motility in the pathogenesis and treatment of diarrhoea. Scand J Gastroenterol 1983;18(suppl 84):45-63 **

Sack RB. Antimicrobial prophylaxis of travelers' diarrhea: a selected summary. Rev Infect Dis 1986 May-Jun;8(suppl 2):S160-6 **

Sack RB. Antimicrobial prophylaxis of travellers' diarrhoea: a summary of studies using doxycycline or trimethoprim and sulphamethoxazole. Scand J Gastroenterol 1983;18(suppl 84):111-7 **

Turnberg LA. Antisecretory activity of opiates in vitro and in vivo in man. Scand J Gastroenterol 1983;18(suppl 84):79-83 **

ANTIGENS, BACTERIAL

Evans DJ, Jr., Evans DG. Classification of pathogenic <u>Escherichia coli</u> according to serotype and the production of virulence factors, with special reference to colonization-factor antigens. Rev Infect Dis 1983 Sep-Oct;5 (suppl 4):S692-701 **

Simmons DAR, Romanowska E. Structure and biology of Shigella flexneri O antigens. J Med Microbiol 1987 Jun;23(4):289-302 **

ANTIGENS. IMMUNE RESPONSE

Dougan G, Hormaeche CE, Maskell DJ. Live oral <u>Salmonella</u> vaccines: potential use of attenuated strains are carriers of heterologous antigens to the immune system. Parasite Immunol 1987 Mar; 9(2):151-60**

Elson CO, Kagnoff MF, Fiocchi C, Befus AD, Targan S. Intestinal immunity and inflammation: recent progress. Gastroenterology 1986 Sep;91(3):746-68

Freier S, Kuperman O. Immune regulation in the gastrointestinal tract. J Pediatr Gastroenterol Nutr 1983;2(suppl 1):S310-4 Mitchell GF. Injection versus infection: the cellular immunology of parasitism. Parasitol Today 1987 Apr;3(4):106-11

Rowley D, La Brooy J. Intestinal immune responses in relation to diarrhoeal diseases. J Diarrhoeal Dis Res 1986 Mar;4(1):1-9 **

Targan SR, Kagnoff MF, Brogan MD, Shanahan F. Immunologic mechanisms in intestinal diseases. Ann Intern Med 1987 Jun;106(6):853-70 **

Trissl D. Immunology of Entamoeba histolytica in human and animal hosts. Rev Infect Dis 1982 Nov-Dec; 4(6):1154-84

ANTIGENS, PROTOZOAN

Pritchard DI. Antigens of gastrointestinal nematodes. Trans R Soc Trop Med Hyg 1986;80(5):728-34 **

ANTITOXINS

Alouf JE. [Anti-toxin vaccines]. Ann Inst Pasteur Microbiol 1985 Nov-Dec; 136B(3):309-21

ARSENIC

Riviere JE, Boosinger TR, Everson RJ. Inorganic arsenic toxicosis in cattle. Mod Vet Pract 1981 Mar;62(3):209-11 **

ARTHRITIS

Catterall RD. Clinical aspects of Reiter's disease. Br J Rheumatol 1983 Nov:22(4 suppl 2):151-5 **

Keat A. Reiter's syndrome and reactive arthritis in perspective. N Engl J Med 1983 Dec 29:309(26):1606-15

Neumann V, Wright V. Arthritis associated with bowel disease. Clin Gastroenterol 1983 Sep;12(3):767-95

ASCARIASIS

Arfaa F. Selective primary health care: strategies for control of disease in the developing world. XII. Ascariasis and trichuriasis. Rev Infect Dis 1984 May-Jun;6(3):364-73 **

ASTROVIRUS AND ASTROVIRUS INFECTIONS

Nazer H. Astrovirus gastroenteritis. J Trop Pediatr 1985 Apr;31(2):67-71 **

BACILLUS SUBTILIS

Piggot PJ, Hoch JA. Revised genetic linkage map of <u>Bacillus subtilis</u>. Microbiol Rev 1985 Jun;49(2):158-79

BACTERIA AND BACTERIAL INFECTIONS

Adegbola RA. Review: Bacterial adhesion and pathogenicity. Afr J Med Med Sci 1988 Jun;17(2):63-9 **

Barrett EL, Clark MA. Tetrathionate reduction and production of hydrogen sulfide from thiosulfate. Microbiol Rev 1987 Jun;51(2):192-205 **

Buchino JJ, Suchy FJ, Snyder JW. Bacterial diarrhea in infants and children. Perspect Pediatr Pathol 1984 Summer;8(2):163-80 **

Evans N. Pathogenic mechanisms in bacterial diarrhoea. Clin Gastroenterol 1979 Sep;8(3):599-623

Formal SB, Hale TL, Sansonetti PJ. Invasive enteric pathogens. Rev Infect Dis 1983 Sep-Oct;5(suppl 4):S702-7 **

Freter R, Jones GW. Models for studying the role of bacterial attachment in virulence and pathogenesis. Rev Infect Dis 1983 Sep-Oct;5(suppl 4):S647-58 **

Germanier R. Oral vaccination against enteric bacterial infections: an overview. Infection 1984 Mar-Apr;12(2):138-42 **

Gracey M. Bacterial diarrhoea. Clin Gastroenterol 1986 Jan; 15(1):21-37 **

Gracey MS. Nutrition, bacteria and the gut. Br Med Bull 1981 Jan;37(1): 71-5 **

Greenough WB, III. Bacterial diarrhoeal diseases: current concepts on etiology and pathogenesis. Southeast Asian J Trop Med Public Health 1982 Sep;13(3):319-24 **

Greenough WB, III. Protozoal, bacterial and viral diarrhoea: a common mechanism [editorial]. J Diarrhoeal Dis Res 1984 Jun;2(2):68

Isaacs PET, Kim YS. Blind loop syndrome and small bowel bacterial contamination. Clin Gastroenterol 1983 May;12(2):395-414

Keusch GT, Solomons NW. Microorganisms, malabsorption, diarrhea and dysnutrition. J Environ Pathol Toxicol Oncol 1985 Jul;5(6):165-209

Lang W. Progress in the pathogenesis and therapy of viral and bacterial

diseases. Scand J Infect Dis 1982; (suppl 36):7-11

Levine MM, Kaper JB, Black RE, Clements ML. New knowledge on pathogenesis of bacterial enteric infections as applied to vaccine development. Microbiol Rev $1983 \, \text{Dec}_{\,}(47(4);510-50) \, **$

Mathias JR, Clench MH. Review: pathophysiology of diarrhea caused by bacterial overgrowth of the small intestine. Am J Med Sci 1985 Jun; 289(6): 243-8 **

Miller DP, Everett ED. Bacterial enteritis. Missouri Med 1983 May;80(5): 241-8 **

Old DC. Bacterial adherence. Med Lab Sci 1985 Jan;42(1):78-85 **

Ordal GW. Bacterial chemotaxis: biochemistry of behavior in a single cell. CRC Crit Rev Microbiol 1985;12(2):95-130

Polin RA. Monoclonal antibodies against microorganisms. Eur J Clin Microbiol 1984 Oct;3(5):387-98

Rennels MB, Levine MM. Classical bacterial diarrhea: perspectives and update - Salmonella, Shigella, Escherichia coli, Aeromonas and Plesiomonas. Pediatr Infect Dis 1986 Jan;5(1):591-100 **

Ruiz-Palacious GM. Norfloxacin in the treatment of bacterial enteric infections. Scand J Infect Dis 1986; (suppl 48):55-63 **

Shuval HI, Yekutiel P, Fattal B. An epidemiological model of the potential health risk associated with various pathogens in wastewater irrigation. Wat Sci Technol 1986;18(10):191-8 **

Shuval HI, Fattal B, Yekutiel P. State of the art review: an epidemiological approach to the health effects of wastewater reuse. Wat Sci Technol 1986;18 (9):147-62 **

Simon GL, Gorbach SL. Intestinal microflora. Med Clin North Am 1982 May;66 (3):557-74

Sparling PF. Bacterial virulence and pathogenesis: an overview. Rev Infect Dis 1983 Sep-Oct;5(suppl 4):S637-46 **

Thimann KV. The life of bacteria: their growth, metabolism, and relationships. 2d ed. New York: MacMillan, 1963:258-88

BACTERIAL ADHESION

Adegbola RA. Review: Bacterial adhesion and pathogenicity. Afr J Med Med Sci $1988 \ \mathrm{Jun}; 17(2): 63-9 \ **$

Candy DCA, Leung TSM, Phillips AD, Harries JT, Marshal WC. Models for studying the adhesion of enterobacteria to the mucosa of the human intestinal tract. In: Elliott K, O'Connor M, Whelen J, eds. Adhesion and microorganisms pathogenicity. London: Pitman, 1981:72-93. (Ciba Foundation symposium, 80)

Freter R, Jones GW. Models for studying the role of bacterial attachment in virulence and pathogenesis. Rev Infect Dis 1983 Sep-Oct;5(suppl 4):S647-58 **

Klemm P. Fimbrial adhesions of <u>Escherichia</u> <u>coli</u>. Rev Infect Dis 1985 May-Jun:7(3):321-40

. Old DC. Bacterial adherence. Med Lab Sci 1985 Jan:42(1):78-85 **

Zhalko-Titarenko VP, Bondarenko VM, Grigoryev AV, Kupchinsky LG, Rybalko SL. Dynamics of the interaction of <u>Shigellae</u> with the epithelium in the process of infection. Zh Mikrobiol Epidemiol Immunobiol 1986 Apr: (4):21-4 **

BACTERIAL PROTEINS

Wadstrom T, Baloda SB, Yuk YR. Cytotoxic and cytolytic proteins of enteropathogenic Escherichia coli and Salmonella: new concepts on possible role in intestinal colonization. Zentralbl Bakteriol Mikrobiol Hyg [A] 1986;(suppl 15):153-60

BACTERIAL TOXINS

Cantey JR. Shiga toxin--an expanding role in the pathogenesis of infectious diseases. J Infect Dis 1985 May: 151(5): 766-71 **

Cunha BA. The toxigenic diarrheas. Intern Med 1987 Feb;8(2):92-110 **

Eidels L. Proia RL, Hart DA. Membrane receptors for bacterial toxins. Microbiol Rev 1983 Dec;47(4):596-20

Keusch GT, Donohue-Rolfe A, Jacewicz M. Shigella toxin(s); description and role in diarrhea and dysentery. Pharmacol Ther 1982;15(3):403-38 **

Keusch GT, Donohue-Rolfe A, Jacewicz M. Shigella toxin and the pathogenesis of shigellosis. Ciba Found Symp 1985;112:193-214 **

Ljungh A, Wadstrom T. Aeromonas toxins. Pharmacol Ther 1982;15(3):339-54 **

Moriarty KJ, Turnberg LA. Bacterial toxins and diarrhoea. Clin Gastroenterol 1986 Jul:15(3):529-43 **

O'Brien AD, Holmes RK. Shiga and Shiga-like toxins. Microbiol Rev 1987 Jun; 51(2):206-20 **

Sanyal SC. NAG Vibrio toxin. Pharmacol Ther 1983;20(2):183-201

BICARBONATES

Elliott EJ, Walker-Smith JA, Farthing MJG. The role of bicarbonate and base precursors in treatment of acute gastroenteritis. Arch Dis Child 1987 Jan;62 (1):91-5 **

BILE ACIDS AND SALTS

Balistreri WF, Heubi JE, Suchy FJ. Bile acid metabolism: relationship of bile acid malabsorption and diarrhea. J Pediatr Gastroenterol Nutr 1983;2(1):105-21

Fromm H, Malavolti M. Bile acid-induced diarrhoea. Clin Gastroenterol 1986 Jul:15(3):567-82 **

Kivilaakso E. Antacids and bile salts. Scand J Gastroenterol 1982;17(suppl 75):16-9 **

BIOMETRY

Shanmugan R. An intervened Poisson distribution and its medical application. Biometrics $1985\ \mathrm{Dec};41(4):1025-9$

BIOPSY

Goldman H. Acute versus chronic colitis: how and when to distinguish by biopsy [editorial]. Gastroenterology 1984 Jan;86(1):199-201 **

BLIND LOOP SYNDROME

Isaacs PET, Kim YS. Blind loop syndrome and small bowel bacterial contamination. Clin Gastroenterol 1983 May:12(2):395-414

BREAST FEEDING

Behar M. The role of feeding and nutrition in the pathogeny and prevention of diarrheic processes. Bull Pan Am Health Organ 1975;9(1):1-9 **

Feachem RG, Koblinsky MA. Interventions for the control of diarrhoeal diseases among young children: promotion of breast-feeding. Bull WHO 1984;62(2): 271-91 **

Research on improving infant feeding practices to prevent diarrhoea or reduce its severity: Memorandum from a JHU/WHO meeting. Bull WHO 1989;67(1):27-33 **

Rowland MGM. The weanling's dilemma: are we making progress? Acta Paediatr Scand 1986;(suppl 323):33-42 **

CALORIC INTAKE

Hoerr RA, Young VR. Alterations in nutrient intake and utilization caused by disease. Ann NY Acad Sci 1987;499:124-31

Molla AM, Molla A, Sarker SA, Rahaman MM. Food intake during and after recovery from diarrhoea in children. <u>In</u>: Chen LC, Scrimshaw NS, eds. Diarrhea and malnutrition: interactions, mechanisms, and interventions. New York: Plenum, 1983:113-23 **

Nicholl CG, Polak JM, Bloom SR. The hormonal regulation of food intake, digestion, and absorption. Annu Rev Nutr 1985;5:213-39

CAMPYLOBACTER AND CAMPYLOBACTER INFECTIONS

Alexandrescu M, Coman G, Ene L, Manuca M. [Human infection with <u>Campylobacter</u> jejuni/coli]. Rev Ig [Bacteriol] 1984 Jul-Sep;29(3):245-57

Blaser MJ, Reller LB. <u>Campylobacter</u> enteritis. N Engl J Med 1981 Dec 10;305 (24):1444-52 **

Blaser MJ, Taylor DN, Feldman RA. Epidemiology of <u>Campylobacter jejuni</u> infections. Epidemiol Rev 1983:5:157-76 **

Blaser MJ. Gastric <u>Campylobacter-like</u> organisms, gastritis, and peptic ulcer disease. Gastroenterology 1987 Aug;93(2):371-83

Butzler JP, Skirrow MB. <u>Campylobacter</u> enteritis. Clin Gastroenterol 1979 Sep:8(3):737-65 **

Butzler JP, Dekeyser P, Detrain M, Dehaen F. Related <u>Vibrio</u> in stools. J Pediatr 1983 Mar;82(3):493-5 **

Chowdhury MNH. <u>Campylobacter jejuni</u> enteritis: a review. Trop Geogr Med 1984 Sep;36(3):215-22 **

Fox JG. Campylobacteriosis - a "new" disease in laboratory animals. Lab Anim Sci 1982 Dec: 32(6):625-37 **

King EO. Human infections with <u>Vibrio</u> fetus and a closely related <u>Vibrio</u>. Unfect Dis 1957 Sep-Oct; 101:119-28 **

Marshall 8J. <u>Campylobacter pyloridis</u> and gastritis. J Infect Dis 1986 Apr; 153(4):650-7 **

Pal SC, Nair GB. Epidemiology of campylobacteriosis in developing countries. ICMR Bull 1984 Jul;14(7):1-4 **

Penner JL. The genus <u>Campylobacter</u>: a decade of progress. Clin Microbiol Rev 1988 Apr;1(2):157-72

Sakai S, Ito T. [Campylobacter infections]. Nippon Saikingaku Zasshi 1985 May;40(3):563-80

Shane SM, Montrose MS. The occurrence and significance of Campylobacter jejuni in man and animals. Vet Res Commun 1985 Jul;9(3):167-98 **

Sharp JCM. Infections associated with milk and dairy products in Europe and

North America, 1980-85. Bull WHO 1987:65(3):397-406 **

Walker RI, Caldwell MB, Lee EC, Guerry P, Trust TJ, Ruiz-Palacios GM. Pathophysiology of <u>Campylobacter</u> enteritis. Microbiol Rev 1986 Mar;50(1): 81-94 **

Weber A. [Occurrence of Campylobacter jejuni in animals and its significance for the human]. Tierarztl Prax 1985;13(2):151-7

CARBOHYDRATES

Caspary WF. Diarrhoea associated with carbohydrate malabsorption. Clin Gastroenterol 1986 Jul;15(3):631-55

Gracey M, Burke V. Sugar-induced diarrhoea in children. Arch Dis Child 1973 May;48(261):331-6 **

Ravich WJ, Bayless TM. Carbohydrate absorption and malabsorption. Clin Gastroenterol 1983 May;12(2):335-56

CATARACT

The nutritional origin of cataracts. Nutr Rev 1984 Nov;42(11):377-9

CELIAC DISEASE

Auricchio S. Gluten-sensitive enteropathy and infant nutrition. J Pediatr Gastroenterol Nutr 1983;2(suppl 1):S304-9

Falchuk ZM. Gluten-sensitive enteropathy. Clin Gastroenterol 1983 May:12(2): 475-94 **

CHEMOTACTIC FACTORS

Ordal GW. Bacterial chemotaxis: biochemistry of behavior in a single cell. CRC Crit Rev Microbiol 1985;12(2):95-130

CHOLERA

Aggarwal P, Misra BS, Singh J, Basu RN. Review of cholera in Delhi: a 14 year study. J Commun Dis 1986 Mar;18(1):17-21 **

Kobari K. Recent trends of cholera. Jpn J Trop Med Hyg 1985 Mar;13(1):53 **
Rabbani GH. Cholera. Clin Gastroenterol 1986 Jul:15(3):507-28

Diarrhoeal Diseases 13

Recent advances in cholera research: Memorandum from a WHO meeting. Bull WHO 1985;63(5):841-9 **

Drug Therapy

Black RE. The prophylaxis and therapy of secretory diarrhea. Med Clin North Am 1982 May:66(3):611-21

History

Morris RJ. Religion and medicine: the cholera pamphlets of Oxford, 1832, 1849 and 1854. Med Hist 1975 Jul;19(3):256-70 **

Takeda Y. "Second century of <u>Vibrio cholerae</u>" [editorial]. J Diarrhoeal Dis Res 1984 Mar:2(1):1-2

Immunology

Kaper JB, Lockman HA, Baldini MM, Levine MM. Development of live oral cholera vaccine candidates through recombinant DNA techniques. In: Kuwahara S, Pierce NF, eds. Advances in research on cholera and related $\overline{\text{diarrheas}}$. Tokyo: KTK Scientific Publishers, 1986:181-91

Levine MM, Kaper JB, 81ack RE, Clements ML. New knowledge on pathogenesis of bacterial enteric infections as applied to vaccine development. Microbiol Rev $1983\ \text{Dec};47(4):510-50$ **

Manning PA. Involvement of cell envelope components in the pathogenesis of Vibrio cholerae: targets for cholera vaccine development. Vaccine 1987 Jun;5(2):83-7

Svennerholm A-M, Jertborn M, Gothefors L, Karim A, Sack D, Holmgren J. Secretory immunity to <u>Vibrio cholerae</u> bacteria and cholera toxin: prospects for an improved cholera <u>vaccine</u>. <u>In</u>: Takeda Y, Miwatani T, eds. Bacterial diarrheal diseases. Tokyo: KTK Scientific Publishers, 1985:169-74 **

Microbiology

Guidolin A, Manning PA. Genetics of $\underline{\text{Vibrio}}$ cholerae and its bacteriophages. Microbiol Rev 1987 Jun;51(2):285-98 **

Occurrence

Briscoe J. The role of water supply in improving health in poor countries (with special reference to Bangladesh). Am J Clin Nutr 1978 Nov;31(11); 2100-13 **

Feachem RG. Environmental aspects of cholera epidemiology, I. A review of selected reports of endemic and epidemic situations during 1961-1980. Trop Dis Bull 1981 Aug;78(8):675-98 **

Monsur KA. How this happened? [editorial]. J Diarrhoeal Dis Res 1983 Mar;1 (1):3-4

Morger H, Steffen R, Schar M. Epidemiology of cholera in travellers, and conclusions for vaccination recommendations. Br Med J 1983 Jan 15;286(6360): 184-6 **

Mosley WH, Khan MU. Cholera epidemiology - some environmental aspects. ProgWater Technol 1979:11(1-2):309-16 **

Steffen R. Epidemiologic studies of travelers' diarrhea, severe gastrointestinal infections, and cholera. Rev Infect Dis 1986 May-Jun;8(suppl 2):S122-30 **

Oral Therapy

Black RE. The prophylaxis and therapy of secretory diarrhea. Med Clin North Am 1982 May; 66(3):611-21

Chatterjee HN. Control of vomiting in cholera and oral replacement of fluid. Lancet 1953 Nov 21;2(6795):1063 **

Greenough WB, III. Principles and prospects in the treatment of cholera and related dehydrating diarrheas. <u>In:</u> Ouchterlony O, Holmgren J, eds. Cholera and related diarrheas: molecular aspects of a global health problem. Basel: Karger, 1980:211-8 (43rd Nobel Symposium) **

Mahalanabis D, Choudhuri AB, Bagchi NG, Bhattacharya AK, Simpson TW. Orał fluid therapy of cholera among Bangladesh refugees. Johns Hopkins Med J 1973 Apr;132(4):197-205 **

Molla AM, Sarker SA, Hossain M, Molla A, Greenough WB, III. Rice-powder electrolyte solution as oral therapy in diarrhoea due to <u>Vibrio cholerae</u> and <u>Escherichia coli</u>. Lancet 1982 Jun 12;1(8285):1317-9 **

Tulloch J. Burton P. Global access to oral rehydration saits and use of oral rehydration therapy. World Health Stat Q 1987:40(2):10-5

Physiopathology

Carpenter CCJ. The pathophysiology of secretory diarrheas. Med Clin North Am 1982 May;66 (3):597-610 **

Finkelstein RA, Dorner F. Cholera enterotoxin (choleragen). Pharmacol Ther 1985;27(1):37-47

Gyr K. Toxin receptors and their pathogenetic significance. Acta Histochem 1984;(suppl 29):S95-102 **

Holmgren J. Pathogenesis and prevention of cholera. Scand J Infect Dis 1982; (suppl 36):58-64

Levine MM, Kaper JB, Black RE, Clements ML. New knowledge on pathogenesis of

Diarrhoeal Diseases 15

bacterial enteric infections as applied to vaccine development. Microbiol Rev 1983 Dec:47(4):510-50 **

Mekalonos JJ. Cholera toxin: genetic analysis, regulation, and role in pathogenesis. Curr Top Microbiol Immunol 1985:118:97-118

van Heyningen S. Cholera toxin: review. Biosci Rep 1982:2:135-46 **

Prevention and Control

Black RH. Invited discussion of Dr R M Glasse's paper. <u>In:</u> Proceedings of the Cholera Research Symposium, Honolulu, Hawaii, 24-29 Jan 1965. Washington, D.C.: U S Government Printing Office, 1965:340 **

Briscoe J. The role of water supply in improving health in poor countries (with special reference to Bangladesh). Am J Clin Nutr 1978 Nov;31(11): 2100-13 **

Feachem RG. Environmental aspects of cholera epidemiology. III. Transmission and control. Trop Dis Bull 1982 Jan:79(1):1-47 **

Holmgren J. Pathogenesis and prevention of cholera. Scand J Infect Dis 1982; (suppl 36):58-64 **

Khan MU, Khan MR, Hossain B, Ahmed QS. Alum potash in water to prevent cholera [letter]. Lancet 1984 Nov 3;2(8410):1032 **

Kuo C. Measures to control diarrhoeal diseases—environmental sanitation. Regional Meeting on Cholera and Diarrhoeal Diseases, Alexandria, 1-5 Jun 1978. Alexandria: Regional Office for the Eastern Mediterranean, World Health Organization, 1978. 6 p. **

Oral cholera vaccines [editorial]. Lancet 1986 Sep 27:2(8509):722-3

Svennerholm A-M, Holmgren J. Oral combined B subunit - whole cell cholera vaccine. In: Holmgren J, Lindberg A, Mollby R, eds. Development of vaccines and drugs against diarrhea; proceedings of the 11th Nobel Conference, Stockholm, 1985. Lund: Studentlitteratur, 1986:33-43 **

Transmission

Black RH. Invited discussion of Dr R M Glasse's paper. <u>In: Proceedings of the Cholera Research Symposium, Honolulu, 24-29 Jan 1965. Washington, D.C.: U S Government Printing Office, 1965:340 **</u>

Briscoe J. Intervention studies and the definition of dominant transmission routes. Am J Epidemiol 1984 Sep;120(3):449-55 **

Briscoe J. The role of water supply in improving health in poor countries (with special reference to Bangladesh). Am J Clin Nutr 1978 Nov:31(11):2100-13 **

Feachem RG. Environmental aspects of cholera epidemiology. III. Transmission

16 Diarrhoeal Diseases

and control. Trop Dis Bull 1982 Jan:79(1):1-47 **

Mosley WH, Khan MU. Cholera epidemiology – some environmental aspects. Prog Water Technol 1979;11(1-2):309-16 **

CHOLERA TOXIN

Finkelstein RA, Dorner F. Cholera enterotoxin (choleragen). Pharmacol Ther 1985;27(1):37-47 **

Mekalonos JJ. Cholera toxin: genetic analysis, regulation, and role in pathogenesis. Curr Top Microbiol Immunol 1985;118:97-118

Svennerholm A-M, Jertborn M, Gothefors L, Karim A, Sack D, Holmgren J. Secretory immunity to <u>Vibrio cholerae</u> bacteria and cholera toxin: prospects for an improved cholera vaccine. <u>In:</u> Takeda Y, Miwatani T, eds. Bacterial diarrheal diseases. Tokyo: KTK Scientific Publishers, 1985:169-74 **

van Heyningen S. Cholera toxin: review. Biosci Rep 1982;2:135-46 **

CHOLERA VACCINE

Germanier R. Oral vaccination against enteric bacterial infections: an overview. Infection 1984 Mar-Apr;12(2):138-42 **

Kaper JB, Lockman HA, Baldini MM, Levine MM. Development of live oral cholera vaccine candidates through recombinant DNA techniques. In: Kuwahara S, Pierce NF, eds. Advances in research on cholera and related diarrheas. Tokyo: KTK Scientific Publishers. 1986:181-91

La Brooy J, Rowley D. Cholera vaccine \sim recent progress. In: Easmon CSF, Jeljasewicz, eds. Medical Microbiology. v. 2. London: Academic Press, 1983:157-76

Levine MM, Kaper JB, Black RE, Clements ML. New knowledge on pathogenesis of bacterial enteric infections as applied to vaccine development. Microbiol Rev $1983\ \text{Dec}_3(7(4)):510-50$ **

Manning PA. Involvement of cell envelope components in the pathogenesis of <u>Vibrio cholerae</u>: targets for cholera vaccine development. Vaccine 1987 Jun;5(2):83-7

Morger H, Steffen R, Schar M. Epidemiology of cholera in travellers, and conclusions for vaccination recommendations. Br Med J 1983 Jan 15;286(6360): 184-6 **

Oral cholera vaccines [editorial]. Lancet 1986 Sep 27:2(8509):722-3

Svennerholm A-M, Holmgren J. Oral combined B subunit - whole cell cholera vaccine. In: Holmgren J, Lindberg A, Mollby R, eds. Development of vaccines and drugs against diarrhea; proceedings of the 11th Nobel Conference.

Stockholm, 1985, Lund: Studentlitteratur, 1986:33-43 **

Svennerholm A-M, Jertborn M, Gothefors L, Karim A, Sack D, Holmgren J. Secretory immunity to <u>Vibrio cholerae</u> bacteria and cholera toxin: prospects for an improved cholera vaccine. <u>In:</u> Takeda Y, Miwatani T, eds. Bacterial diarrheal diseases. Tokyo: KTK Scientific Publishers, 1985:169-74 **

CHROMATOGRAPHY, GAS

Brooks JB. Review of frequency-pulsed electron-capture gas-liquid chromatography studies of diarrheal diseases caused by members of the family Enterobacteriaceae, Clostridium difficile, and rotavirus. J Clin Microbiol 1986 Nov:24(5):687-91 **

CLOSTRIDIUM

Georye WL. Antimicrobial agent-associated colitis and diarrhea: historical background and clinical aspects. Rev Infect Dis 1984 Mar-Apr;6(suppl 1): \$208-13 **

Meyer-Kawohl R, Bockermuhl J. <u>[Clostridium difficile-induced enterocolitis: pathogenesis, clinical course, epidemiology and laboratory diagnosis]. Immun Infekt 1986 Apr:14(2):63-7</u>

Rolfe RD. Diagnosis of <u>Clostridium difficile</u>-associated intestinal disease. CRC Crit Rev Clin Lab Sci 1986;24(3):235-61 **

Silva J. Jr., Fekety R. <u>Clostridia</u> and antimicrobial enterocolitis. Annu Rev Med 1981:32:327-33

Trnka YM, Lamont JT. <u>Clostridium</u> <u>difficile</u> colitis. Adv Intern Med 1984;29: 85-107 **

COLITIS AND ENTEROCOLITIS

Altman DF. Gastrointestinal cryptosporidiosis and cytomegalovirus enterocolitis. Front Radial Ther Oncol 1985;19:88-90

Bartlett JG. Antibiotic-associated colitis. DM 1984 Dec;30(15):1-54

Burdon DW. Treatment of pseudomembranous colitis and antibiotic-associated diarrhoea. J Antimicrob Chemother 1984 Dec;14(suppl D):103-9 **

Cheromcha DP, Hyman PE. Neonatal necrotizing enterocolitis: inflammatory bowel disease of the newborn. Dig Dis Sci 1988 Mar;33(suppl 3):785-845 **

de Louvois J. Necrotising enterocolitis. J Hosp Infect 1986 Jan;7(1):4-12

Farmer RG. Nonspecific ulcerative proctitis. Gastroenterol Clin North Am

1987 Mar; 16(1): 157-74

Fortson WC, Tedesco FJ. Drug-induced colitis: a review. Am J Gastroenterol 1984 Nov;79(11):878-83 **

George WL. Antimicrobial agent-associated colitis and diarrhea: historical background and clinical aspects. Rev Infect Dis 1984 Mar-Apr;6(suppl 1): \$208-13 **

Goldman H. Acute versus chronic colitis: how and when to distinguish by biopsy [editorial]. Gastroenterology 1984 Jan;86(1):199-201 **

Gotz VP, Rand KH. Medical management of antimicrobial-associated diarrhea and colitis. Pharmacotherapy 1982 Mar-Apr;2(2):100-9 **

Gross MH. Management of antibiotic-associated pseudomembranous colitis. Clin Pharm 1985 May-Jun; 4(3):304-10

Karmali MA. Infection by verocytotoxin-producing <u>Escherichia</u> <u>coli</u>. Clin Microbiol Rev 1989 Jan;2(1):15-38 **

Kliegman RM, Fanaroff AA. Necrotizing enterocolitis. N Engl J Med 1984 Apr 26;310(17):1093-1103 **

Kosloske AM. Pathogenesis and prevention of necrotizing enterocolitis: a hypothesis based on personal observation and a review of the literature. Pediatrics $1984\ Dec; 74(6): 1086-92$ **

Lennard-Jones JE. Medical treatment of ulcerative colitis. Postgrad Med J 1984 Nov;60(709):797-802

Mani V. Idiopathic ulcerative colitis - clinical problems, controversies and what's new. Trop Gastroenterol 1986 Oct-Dec;7(4):147-56

Mayberry JF. Some aspects of the epidemiology of ulcerative colitis. Gut $1985 \, \mathrm{Sep}; 26(9): 968-74$

Meyer-Kawohl R, Bockermuhl J. [Clostridium difficile-induced enterocolitis: pathogenesis, clinical course, epidemiology and laboratory diagnosis]. Immun Infekt 1986 Apr;14(2):63-7

Mosalova NM. [Cytochemical characteristics of lymphocytes in patients with chronic enterocolitis]. Vrach Delo 1985 Mar;(3):81-3

Nime FA, Burek JD, Page DL, Holscher MA, Yardley JH. Acute enterocolitis in a human being infected with the protozoan <u>Cryptosporidium</u>. Gastroenterology 1976 Apr;70(4):592-8 **

O'Connor TW. Pseudomembranous enterocolitis: a historical and clinical review. Dis Colon Rectum 1981 Sep;24(6):445-8 **

Persson PG, Ahlbom A, Hellers G. Crohn's disease and ulcerative colitis. A review of dietary studies with emphasis on methodologic aspects. Scand J Gastroenterol 1987 May;22(4):385-9

Diarrhoeal Diseases 19

Rampton DS, Hawkey CJ. Prostaglandins and ulcerative colitis. Gut 1984 Dec; 25(12):1399-413

Rams H, Rogers AI, Ghandur-Mnaymneh L. Collagenous colitis. Ann Intern Med 1987 Jan;106(1):108-13 **

Rolfe RD. Diagnosis of <u>Clostridium difficile</u>-associated intestinal disease. CRC Crit Rev Clin Lab Sci 1986;24(3):235-61 **

Silva J, Jr., Fekety R. <u>Clostridia</u> and antimicrobial enterocolitis. Annu Rev Med 1981;32:327-33

Tedesco FJ. Pseudomembranous colitis: pathogenesis and therapy. Med Clin North Am 1982 May;66(3):655-64

Topalian SL, Ziegler MM. Necrotizing enterocolitis: a review of animal models. J Surg Res 1984 Oct;37(4):320-36

Trnka YM, Lamont JT. <u>Clostridium</u> <u>difficile</u> colitis. Adv Intern Med 1984;29:

Wormann B, Hochter W, Othenjiann R. [Drug-induced colitis]. Dtsch Med Wochenschr 1985 Sep 27;110(39):1504-9

COLONIC DISEASES, FUNCTIONAL

Corazza GR, Gasbarrini G. Defective splenic function and its relation to bowel disease. Clin Gastroenterol 1983 Sep;12(3):651-69

Drossman DA, Lowman BC. Irritable bowel syndrome: epidemiology, diagnosis and treatment. Clin Gastroenterol 1985 Jul;14(3):559-73 **

Krag E. Irritable bowel syndrome: current concepts and future trends. Scand J Gastroenterol 1985;20(suppl 109):107-15 **

Misiewicz JJ. Human colonic motility. Scand J Gastroenterol 1984;19(suppl 93):43-51 **

Neumann V, Wright V. Arthritis associated with bowel disease. Clin Gastroenterol 1983 Sep;12(3):767-95

Read NW. The relationships between colonic motility and transport. Scand J Gastroenterol 1984;19(suppl 93):35-42 **

Thompson JS, Rikkers LF. Surgical alternatives for the short bowel syndrome. Am J Gastroenterol 1987 Feb;82(2):97-106 **

Thompson WG. The irritable bowel. Gut 1984 Mar; 25(3):305-20 **

Tucker H, Schuster MM. Irritable bowel syndrome: newer pathophysiologic concepts. Adv Intern Med 1982;27:183-204

Weber J, Ducrotte P. Colon motility in health and disease. Dig Dis 1987;5(1):1-12

COMMUNICABLE DISEASES

Baker RW, Peppercorn MA. Enteric diseases of homosexual men. Pharmacotherapy 1982 Jan-Feb; 2(1):32-42

The Child Day Care Infectious Disease Study Group. Considerations of infectious diseases in day care centers. Pediatr Infect Dis 1985 Mar-Apr;4 (2):124-36

Ellencweig AY, Slater PE. Demographic and socio-economic patterns of hospitalization for infectious diseases in Israel. Eur J Epidemiol 1986 Jun; 2(2):83-9 **

Elzouki AY, Vesikari T. First international conference on infections in children in Arab countries. Pediatr Infect Dis 1985 Sep-Oct;4(5):527-31

Poppensiek GC, Kahrs RF. Twenty-five years of progress in understanding major infectious diseases of dairy cattle. J Dairy Sci 1981 Jun;64(6):1443-64

Sever JL. Infectious diseases and immunizations. Rev Infect Dis 1982 Jan-Feb; 4(1):136-46

Wolfe MS. Diseases of travelers. Clin Symp 1984;36(2):2-32

COMMUNITY PARTICIPATION

Feachem RG. Rural water and sanitation; community participation in appropriate water supply and sanitation technologies: the mythology for the decade. Proc R Soc Lond (B) 1980 Jul:209(1174):15-29

Sarkar U. Community participation in the control of diarrhoea. J Indian Med Assoc 1987 Jul;85(7):210-2

COPPER

Tasman-Jones C, Kay RG, Lee SP. Zinc and copper deficiency, with particular reference to parenteral nutrition. Surg Annu 1978;10:23-52 **

CORONAVIRIDAE

Torres-Medina A, Schläfer DH, Mebus CA. Rotavirual and coronaviral diarrhea. Vet Clin North Am (Food Anim Pract) 1985 Nov;1(3):471-93 **

COST BENEFIT ANALYSIS

Creese AL. Cost effectiveness of potential immunization interventions against

diarrhoeal disease. Soc Sci Med 1986;23(3):231-40 **

Cvjetanovic B, Chen L, Kronmall R, Rohde C, Suskind R. Measuring and evaluating diarrhea and malabsorption in association with village water supply and sanitation: a review of the Food Wastage/Sanitation Cost Benefit Methodology Project (Guatemala). Arlington, Virginia: Water and Sanitation for Health Project, 1981. 36 p. (WASH technical report, 12)

Cvjetanovic B. Sanitation versus immunization in control of enteric and diarrhoeal diseases. Prog Water Technol 1979;11(1-2):81-7 **

CROHN DISEASE

Cello JP. Inflammatory and malignant diseases of the small bowel causing malabsorption. Clin Gastroenterol 1983 May:12(2):511-32

Chiodini RJ. Crohn's disease and the mycobacterioses: a review and comparison of two disease entities. Clin Microbiol Rev 1989 Jan;2(1):90-117 **

Doe WF, Hapel AJ. Intestinal immunity and malabsorption. Clin Gastroenterol 1983 May;12(2):415-35

Drossman DA, Lowman BC. Irritable bowel syndrome: epidemiology, diagnosis and treatment. Clin Gastroenterol 1985 Jul;14(3):559-73 **

Krag E. Irritable bowel syndrome: current concepts and future trends. Scand J Gastroenterol 1985;20(suppl 109):107-15 **

Persson PG, Ahlbom A, Hellers G. Crohn's disease and ulcerative colitis. A review of dietary studies with emphasis on methodologic aspects. Scand J Gastroenterol 1987 May;22(4):385-9

CRYPTOSPORIDIUM AND CRYPTOSPORIDIOSIS

Altman DF. Gastrointestinal cryptosporidiosis and cytomegalovirus enterocolitis. Front Radial Ther Oncol 1985;19:88-90

Angus KW. Cryptosporidiosis in man, domestic animals and birds: a review. J R Soc Med 1983 Jan:76:62-9

Armstrong M. Cryptosporidiosis. Med Lab Sci 1987 Jul;44(3):280-4 **

Burchard GD. [Clinical importance, epidemiology and laboratory diagnosis of intestinal cryptosporidia infection]. Immun Infekt 1986 Apr;14(2):51-7

Casemore DP, Sands RL, Curry A. Cryptosporidium species a "new" human pathogen. J Clin Pathol 1985 Nov; 38(11): 1321-36**

Fayer R, Ungar BLP. <u>Cryptosporidium</u> spp. and cryptosporidiosis. Microbiol Rev 1986 Dec;50(4):458-83 **

Janoff EN, Reller LB. <u>Cryptosporidium</u> species, a protean protozoan. J Clin

Microbiol 1987 Jun;25(6):967-75

Navin TR, Juranek DD. Cryptosporidiosis: clinical, epidemiologic, and parasitologic review. Rev Infect Dis 1984 May-Jun;6(3):313-27 **

Navin TR. Cryptosportdiosis in humans: review of recent epidemiologic studies. Eur J Epidemiol 1985 Jun;1(2):77-83 **

Nime FA, Burek JD, Page DL, Holscher MA, Yardley JH. Acute enterocolitis in a human being infected with the protozoan <u>Cryptosporidium</u>. Gastroenterology 1976 Apr;70(4):592-8 **

Rolston KV, Fainstein V. Cryoptosporidiosis. Eur J Clin Microbiol 1986 Apr;5(2): 135-7

Tzipori S. Cryptosporidiosis in animals and humans. Microibol Rev 1983 Mar; 47(1):84-96 **

CYTOMEGALOVIRUSES

Altman DF. Gastrointestinal cryptosporidiosis and cytomegalovirus enterocolitis. Front Radial Ther Oncol 1985:19:88-90

CYTOTOXINS

Karmali MA. Infection by verocytotoxin-producing Escherichia coli. Clin Microbiol Rev 1989 Jan;2(1):15-38 **

Ljungh A. Eneroth P. Wadstrom T. Cytotonic enterotoxin from <u>Aeromonas</u> hydrophila. Toxicon 1982;20(4):787-94 **

DAIRY PRODUCTS

Sharp JCM. Infections associated with milk and dairy products in Europe and North America, 1980-85. Bull WHO 1987:65(3):397-406 **

DEHYDRATION

Darrow DC, Pratt EL, Flett J, Jr., Gamble AH, Wiess HF. Disturbances of water and electrolytes in infantile diarrhea. Pediatrics 1949 Feb;3(2):129-56 **

Darrow DC, Pratt EL. Fluid therapy: relation to tissue composition and the expenditure of water and electrolyte. JAMA 1950 May 27:143(4):365-73

Darrow DC, Pratt EL. Fluid therapy: relation to tissue composition and the expenditure of water and electrolyte. JAMA 1950 Jun 3;143(5):432-9

Feld LG, Kaskel FJ, Schoeneman MJ. The approach to fluid and electrolyte

Diarrhoeal Diseases 23

therapy in pediatrics. Adv Pediatr 1988;35:497-536

Gottlieb RP. Dehydration and fluid therapy. Emerg Med Clin North Am 1983 Apr:1(1):113-23

Greenough WB, III. Principles and prospects in the treatment of cholera and related dehydrating diarrheas. <u>In</u>: Ouchterlony O, Holmgren J, eds. Cholera and related diarrheas: molecular aspects of a global health problem. Basel: Karger. 1980:211-8 (43rd Nobel Symposium) **

Mahalanabis D, Choudhuri AB, Bagchi NG, Bhattacharya AK, Simpson TW. Oral fluid therapy for cholera among Bangladesh refugees. Johns Hopkins Med J 1973 Apr:132(4):197-205 ***

Nalin DR. Oral replacement of water and electrolyte losses due to travellers' diarrhoea. Scand J Gastroenterol 1983:18(suppl 84):95-8 **

Paneth N. Hypernatremic dehydration of infancy: an epidemiologic review. Am J Dis Child 1980 Aug:134(8):785-92 **

Sunoto. Oral rehydration salts: a. simple and appropriate tool against dehydration due to diarrhoea. Paediatr Indones 1981 Mar-Apr;21(3-4):90-100 **

DIABETES MELLITUS

Atkinson M, Hosking DJ. Gastrointestinal complications of diabetes mellitus. Clin Gastroenterol 1983 Sep:12(3):633-50

Feldman M, Schiller LR. Disorders of gastrointestinal motility associated with diabetes mellitus. Ann Intern Med 1983 Mar:98(3):378-84 **

DIAGNOSIS, LABORATORY

Burchard GD. [Clinical importance, epidemiology and laboratory diagnosis of intestinal cryptosporidia infection]. Immun Infekt 1986 Apr;14(2):51-7

Echeverria P, Seriwatana J, Sethabutr O, Taylor DN. DNA hybridization in the diagnosis of bacterial diarrhea. Clin Lab Med 1985 Sep;5(3):447-62

Guerrant RL, Shields DS, Thorson SM, Schorling JB, Groschel DHM. Evaluation and diagnosis of acute infectious diarrhea. Am J Med 1985 Jun 28;78(suppl 6B):91-8 **

Guerrant RL, Saver KT. Selective use of microbiological procedures for identification of ethologic agents of diarrheal illness. J Food Saf 1981;3: 145-64 **

Hyypia T, Pettersson U. Spot hybridization for the detection of adenoviruses and enteroviruses. Clin Lab Med 1985 Sep;5(3):491-501

Mahmoud AA. A physician's guide to the diagnosis of common parasitic infections. Med Clin North Am 1983 Jan;67(1):253-8

Rapid laboratory techniques for the diagnosis of viral infections. WHO Tech Rep Ser 1981;(661):1-60

Rolfe RD. Diagnosis of Clostridium difficile-associated intestinal disease. CRC Crit Rev Clin Lab Sci $\frac{1986;24(3):235-61}{1986;24(3):235-61}$

Schmidt NJ. Rapid viral diagnosis. Med Clin North Am 1983 Sep;67(5):953-72 **

Tanowitz HB, Weiss LM, Wittner M. Diagnosis and treatment of protozoan diarrheas. Am J Gastroenterol 1988 Apr;83(4):339-50

Yolken RH, Leggiadro RJ. Immunoassays for the diagnosis of viral enteric pathogens. Diagn Microbiol Infect Dis 1986 Mar; 4(suppl 3): S61-9

DIARRHEA

Axon AT. Functional diarrhoea. Int J Color Dis 1986 Jan;1(1):49-53

Black RE. Viral diarrheas. <u>In</u>: Strickland GT, ed. Hunter's Tropical medicine. 6th ed. Philadelphia: Saunders, 1984:124-31

Butler TC. Viral diarrhoeas [editorial perspective]. J Diarrhoeal Dis Res 1984 Sep;2(3):137-41 **

Chakraborty B. Diarrhoeal diseases in children [editorial]. J Indian Med Assoc 1987 Jul;85(7):193-5

Chandra RK, Greenough WB, Guerrant RL, Martorell R, Mata LJ, Warren KS, Wu C-C. Diarrhea and malnutrition: research priorities. <u>In</u>: Chen LC, Scrimshaw NS, eds. Diarrhea and malnutrition: interactions, mechanisms, and interventions. New York: Plenum. 1983:305-8 **

Chowdhury AMR, D'Souza S. A design and field methods for monitoring impact on mortality of an oral therapy programme. Dhaka: International Centre for Diarrhoeal Disease Research, Bangladesh, 1982. 23 p. (ICDDR,B working paper 27) **

Cooper BT. Diarrhoea as a symptom. Clin Gastroenterol 1985 Jul;14(3):599-613 **

Cvjetanovic B. Epidemiological models of diarrhoeal diseases [editorial]. J Diarrhoeal Dis Res 1985 Jun;3(2):63-4

Diarrhoea. Clin Gastroenterol 1986 Jul;15(3):477-744

DuPont HL. Diarrheal diseases: an overview. Am J Med 1985 Jun 28;78(suppl 6B):63-4

Feldman M. Southwestern internal medicine conference: traveler's diarrhea. Am J Med Sci 1984 0ct;288(3):136-48 **

Kean 8H. Travelers' diarrhea: an overview. Rev Infect Dis 1986 May-Jun;8 (suppl 2):S111-6 **

Kimmey M. Infectious diarrhea. Emerg Med Clin North Am 1985 Feb;3(1):127-42

Kluge RM. Infectious diarrhea: an update. Compr Ther 1983 Nov;9(11):26-30

MacIntyre PB. The short bowel. Br J Surg 1985 Sep;(suppl 72):S92-3

Metcalf AM, Phillips SF. [leostomy diarrhoea. Clin Gastroenterol 1986 Jul;15 (3):705-22

Miller DP, Everett ED. Bacterial enteritis. Missouri Med 1983 May;80(5): 241-8 **

Nye FJ. Travelers' diarrhoea. Clin Gastroenterol 1979 Sep;8(3):767-81

Quinn TC, Bender BS, Bartlett JG. New developments in infectious diarrhea. DM 1986 Apr;32(4): 165-244 **

Rahaman MM. Diarrhoea in Bangladesh: an overview of research conducted between 1962-1984. In: Tzipori S, Barnes G, Bishop R, Holmes I, Robins-Browne R, eds. Infectious $\overline{\text{diarrhoea}}$ in the young: strategies for control in humans and animals; proceedings of an International Seminar on Diarrhoeal Disease in South East Asia and the Western Pacific Region, Geelong, 10-15 Feb 1985. Amsterdam: Elsevier, 1985:69-72 **

Sattherwhite TK, DuPont HL. Infectious diarrhea in office practice. Med Clin North Am 1983 Jan;67(1):203-20

Sen PC, Banerjee H. Diarrhoea - the great killer of infants and children. Your Health 1983 Jan;32(1):9-14 **

Wolfe MS. Diseases of travelers. Clin Symp 1984;36(2):2-32

Chemically Induced

George WL. Antimicrobial agent-associated colitis and diarrhea: historical background and clinical aspects. Rev Infect Dis 1984 Mar-Apr;6(suppl 1): \$208-13 **

Gotz VP, Rand KH. Medical management of antimicrobial-associated diarrhea and colitis. Pharmacotherapy 1982 Mar-Apr;2(2):100-9 **

Gracey M, Burke V. Sugar-induced diarrhoea in children. Arch Dis Child 1973 May:48(261):331-6 **

Riviere JE, Boosinger TR, Everson RJ. Inorganic arsenic toxicosis in cattle. Mod Vet Pract 1981 Mar;62(3):209-11 **

Rocha MP, Burrichter PJ, Blodgett RC. Effect of chrysotherapy on the lower gastrointestinal tract: a review. Semin Arthritis Rheum 1987 May:16(4):294-9

Complications

Atkinson M. Hosking DJ. Gastrointestinal complications of diabetes mellitus.

Clin Gastroenterol 1983 Sep; 12(3):633-50

Axton JH. Measles and the state of nutrition. S Afr Med J 1979 Jan 27;55(4):125-6 **

Diarrhea and malabsorption associated with the acquired immunodeficiency syndrome (AIDS). Nutr Rev 1985 Aug;43(8):235-7

Guillozet N. Measles in Africa: a deadly disease. Some personal comments. Clin Pediatr (Phila) 1979 Feb;18(2):95-100 **

Kubba R. Gastrointestinal manifestations of skin diseases: a review. Trop Gastroenterol 1983 Apr-Jun;4(2):67-78

Pirie A. Vitamin A deficiency and child blindness in the developing world. Proc Nutr Soc 1983 Jan;42(1):53-64

Rambaud J-C. Small intestinal lymphomas and alpha-chain disease. Clin Gastroenterol 1983 Sep;12(3):743-66

Rosenberg IH, Solomons NW, Schneider RE. Malabsorption associated with diarrhea and intestinal infections. Am J Clin Nutr 1977 Aug;30(8):1248-53 **

Congenital

Holmberg C. Congenital chloride diarrhoea. Clin Gastroenterol 1986 Jul;15 (3):583-602

Diagnosis

Brooks JB. Review of frequency-pulsed electron-capture gas-liquid chromatography studies of diarrheal diseases caused by members of the family Enterobacteriaceae, Clostridium difficile, and rotavirus. J Clin Microbiol 1986 Nov;24(5):687-91 **

Echeverria P, Seriwatana J, Sethabutr O, Taylor DN. DNA hybridization in the diagnosis of bacterial diarrhea. Clin Lab Med 1985 Sep;5(3):447-62 **

Shiau YF. Clinical and laboratory approaches to evaluate diarrheal disorders. CRC Crit Rev Clin Lab Sci 1987;25(1):43-69 **

Diet Therapy

Banwell JG. Treatment of travelers' diarrhea: fluid and dietary management. Rev Infect Dis 1986 May-Jun;8(suppl 2):S182-7 **

Feeding during diarrhea. Nutr Rev 1986 Mar: 44(3):102

Jelliffe EFP, Jelliffe DB, Feldon K, Ngokwey N. Traditional practices concerning feeding during and after diarrhoea (with special reference to acute dehydrating diarrhoea in young children). World Rev Nutr Diet 1987;53:218-95

Rohde JE, Cash RA, Guerrant RL, Mahalanabis D, Molla AM, Valyasevi A.

Diarrhoeal Diseases 27

Therapeutic interventions in diarrhea. $\underline{1n}$: Chen LC, Scrimshaw NS, eds. Diarrhea and malnutrition: interactions, mechanisms, and interventions. New York: Plenum, 1983:287-95 **

Drug Therapy

Bennet J. A review of antidiarrhoeal compounds. In: Gough D, ed. The control of diarrhoea in clinical practice. London: The Royal Society of Medicine, 1978:1-8 (Royal Society of Medicine International Congress and Symposium series, 5)

Black RE. The prophylaxis and therapy of secretory diarrhea. Med Clin North Am 1982 May:66(3):611-21

Bradshaw MJ, Harvey RF. Antidiarrhoeal agents: clinical pharmacology and therapeutic use. Curr Ther 1983 Feb:65-73 **

Burdon DW. Treatment of pseudomembranous colitis and antibiotic-associated diarrhoea. J Antimicrob Chemother 1984 Dec;14(suppl D):103-9 **

de Zoysa I, Feachem RG. Interventions for the control of diarrhoeal diseases among young children: chemoprophylaxis. Bull WHO 1985;63(2):295-315

Donowitz M, Wicks J, Sharp GWG. Drug therapy for diarrheal diseases: a look ahead. Rev Infect Dis 1986 May-Jun;8(suppl 2):S188-201 **

Donowitz M, Wicks J, Cusolito S, Sharp GW. Pharmacotherapy of diarrheal diseases: an approach based on physiologic principles. KROC Found Ser 1984;17:329-59

OuPont HL, Ericsson CD, Johnson PC, Cabada FJ. Antimicrobial agents in the prevention of travelers' diarrhea. Rev Infect Dis 1986 May-Jun;8(suppl 2): S167-71 **

DuPont HL, Ericsson CD, Reves RR, Galindo E. Antimicrobial therapy for travelers' diarrhea. Rev Infect Dis 1986 May-Jun;8(suppl 2):S217-22 **

Ericsson CD, DuPont HL, Johnson PC. Nonantibiotic therapy for travelers' diarrhea. Rev Infect Dis 1986 May-Jun;8(suppl 2):S202-6 **

Fedorak RN, Field M. Antidiarrheal therapy: prospects for new agents. Dig Dis Sci 1987 Feb;32(2):195-205 **

Fekety R. Recent advances in management of bacterial diarrhea. Rev Infect Dis 1983 Mar-Apr;5(2):246-57 **

Gotz VP, Rand KH. Medical management of antimicrobial-associated diarrhea and colitis. Pharmacotherapy 1982 Mar-Apr;2(2):100-9 **

Greenough WB, III, Rabbani GH. Antisecretory and antimicrobial drugs for treating diarrhoea. \underline{In} : Holmgren J, Lindberg A, Mollby R, eds. Development of vaccines and drugs against diarrhea; proceedings of the 11th Nobel Conference, Stockholm, 1985. Lund: Studentilitteratur, 1986:270-7

Johnson PC. DuPont HL. Ericsson CD. Chemoprophylaxis and chemotherapy of

travelers' diarrhea in children. Pediatr Infect Dis 1985 Nov-Dec;4(6):620-1

Lambert HP. Antimicrobial agents in diarrhoeal disease. Clin Gastroenterol 1979 Sep;8(3):827-33 **

Levine MM. Antimicrobial therapy for infectious diarrhea. Rev Infect Dis 1986 May-Jun:8(suppl 2):S207-16 **

Netchvolodoff CV, Hargrove MD, Jr. Recent advances in the treatment of diarrhea. Arch Intern Med 1979 Jul;139(7):813-6 **

Powell DW. Enterotoxigenic diarrhea: mechanisms and prospects for therapy. Pharmacol Ther 1984:23(3):407-16 **

Sack RB. Antimicrobial prophylaxis of travelers' diarrhea: a selected summary. Rev Infect Dis 1986 May-Jun;8(suppl 2):S160-6 **

Sack RB. Antimicrobial prophylaxis of travellers' diarrhoea: a summary of studies using doxycycline or trimethoprim and sulphamethoxazole. Scand J Gastroenterol 1983;18(suppl 84):111-7 **

Steffen R, Heusser R, DuPont HL. Prevention of travelers' diarrhea by nonantibiotic drugs. Rev Infect Dis 1986 May-Jun;8(suppl 2):S151-9 **

Turner AC. Travellers' diarrhoea: prevention by chemoprophylaxis. Scand J Gastroenterol 1983;18(suppl 84):107-10 **

Etiology

Black RE. Pathogens that cause travelers' diarrhea in Latin America and Africa. Rev Infect Dis 1986 May-Jun;8(suppl 2):S131-5 **

Booth CC. Diarrhoea due to intestinal malabsorption. Proc R Soc Med 1963 Dec;56(12):1068-70 **

Burman D. Iron deficiency in infancy and childhood. Clin Haematol 1982 Jun; 11 (2):339-51

Caspary WF. Diarrhoea associated with carbohydrate malabsorption. Clin Gastroenterol 1986 Jul;15(3):631-55

Feachem RG. Vitamin A deficiency and diarrhoea: a review of interrelation-ships and their implications for the control of xerophthalmia and diarrhoea. Trop Dis Bull 1987 Mar;84(3):R1-16 **

Gracey M, Burke V. Characteristics of Aeromonas species and their association with human diarrhoeal disease. J Diarrhoeal Dis Res 1986 Jun;4(2):70-3 **

Gracey MS. Nutrition, bacteria and the gut. Br Med Bull 1981 Jan;37(1): 71-5 **

Greenough WB, III. Bacterial diarrhoeal diseases: current concepts on etiology and pathogenesis. Southeast Asian J Trop Med Public Health 1982 Sep;13(3):319-24 **

Diarrhoeal Diseases 29

Holmberg SD, Farmer JJ, 3d. Aeromonas hydrophila and Plesiomonas shigelloides as causes of intestinal infections. Rev Infect Dis 1984 Sep-Oct;6(5):633-9 **

Keusch GT, Solomons NW. Microorganisms, malabsorption, diarrhea and dysnutrition. J Environ Pathol Toxicol Oncol 1985 Jul;5(6):165-209

Keusch GT, Donowitz M. Pathophysiological mechanisms of diarrhoeal diseases: diverse aetiologies and common mechanisms. Scand J Gastroenterol 1983;18 (suppl 84):33-43 **

Kivilaakso E. Antacids and bile salts. Scand J Gastroenterol 1982;17(suppl 75):16-9 **

Ljungh A, Wadstrom T. <u>Aeromonas</u> and <u>Plesiomonas</u> as possible causes of diarrhoea. Infection 1985 Jul-Aug;13(4):169-73 **

Ma P, Kaufman D. <u>Isospora belli</u> diarrheal infection in homosexual men. AIDS Res 1984;1(5):327-38 **

McClain CJ. Trace metal abnormalities in adults during hyperalimentation. JPEN 1981 Sep-Oct:5(5):424-9 **

Nelson JD. Etiology and epidemiology of diarrheal diseases in the United States. Am J Med 1985 Jun 28;78(suppl 6B):76-80 **

The nutritional origin of cataracts. Nutr Rev 1984 Nov;42(11):377-9

Pirie A. Vitamin A deficiency and child blindness in the developing world. Proc Nutr Soc 1983 Jan;42(1):53-64

Ramband JC, Hautefeuille M, Ruskone A, Jacquenod P. Diarrhoea due to circulating agents. Clin Gastroenterol 1986 Jul;15(3):603-29

Reddy V. Interaction between malnutrition and diarrhoea with particular reference to pediatric practice. Indian J Gastroenterol 1985 Jul;4(3):183-6 **

San Joaquin VH, Marks MI. New agents in diarrhea. Pediatr Infect Dis 1982 Jan-Feb;1(1):53-6b

Saw SP. The causes and economic significance of enteric infections in domestic animals. In: Tzipori S, Barnes G, Bishop R, Holmes I, Robins-Browne R, eds. Infectious diarrhoea in the young: strategies for control in humans and animals; proceedings of an International Seminar on Diarrhoeal Disease in South East Asia and the Western Pacific Region, Geelong, 10-15 Feb 1985. Amsterdam: Elsevier, 1985:160-2 **

Sunoto. Diarrhoeal problems in Southeast Asia. Southeast Asian J Trop Med Public Health 1982 Sep;13(3):306-18 **

Tasman-Jones C, Kay RG. Lee SP. Zinc and copper deficiency, with particular reference to parenteral nutrition. Surg Annu 1978;10:23-52 **

Taylor DN, Echeverria P. Etiology and epidemiology of travelers' diarrhea in Asia. Rev Infect Dis 1986 May-Jun;8(suppl 2):S136-41 **

Tharavanij S. Pathogenesis of diarrhoea caused by parasites, Southeast Asian

J Trop Med Public Health 1982 Sep;13(3):331-8 **

Trust TJ, Chipman DC. Clinical involvement of <u>Aeromonas hydrophila</u>. Can Med Assoc J 1979 Apr 21;120(8):942-6 **

von Graevenitz A. <u>Aeromonas</u> and <u>Plesiomonas</u> as agents for diarrhea. <u>In:</u> Ellner PD, ed. Infectious diarrheal diseases; current concepts and laboratory procedures. New York: Marcel, 1984:59-75

Wadstrom T. <u>Aeromonas</u> and <u>Plesiomonas</u> as possible causes of diarrhoea. Third Meeting of the <u>Scientific</u> Working Group on Bacterial Enteric Infections, Geneva, 12-14 Sep 1984. Geneva: World Health Organization, 1984. 10 p. (Unpublished document)

Ziegler MM. Short bowel syndrome in infancy: etiology and management. Clin Perinatol 1986 Mar;13(1):163-73

Zollinger RM. Islet cell tumors of the pancreas and the alimentary tract. Am J Surg 1975 Feb; 129(2):102-10 **

Fluid Therapy

Banwell JG. Treatment of travelers' diarrhea: fluid and dietary management. Rev Infect Dis 1986 May-Jun;8(suppl 2):S182-7 **

Darrow DC, Pratt EL. Fluid therapy: relation to tissue composition and the expenditure of water and electrolyte. JAMA 1950 May 27;143(4):365-73

Darrow DC, Pratt EL. Fluid therapy: relation to tissue composition and the expenditure of water and electrolyte. JAMA 1950 Jun 3;143(5):432-9

Gottlieb RP. Dehydration and fluid therapy. Emerg Med Clin North Am 1983 Apr;1(1):113-23

Immunology

Arbo A, Santos JI. Diarrheal diseases in the immunocompromised host. Pediatr Infect Dis 1987~Oct; 6(10):894-906

Feachem RG, Koblinsky MA. Interventions for the control of diarrhoeal diseases among young children: promotion of breast-feeding. Bull WHO 1984;62(2):271-91 **

Kapikian AZ, Flores J, Hoshino Y, Glass RI, Midthun K, Gorziglia M, Chanock RM. Rotavirus: the major etiologic agent of severe infantile diarrhea may be controllable by a "Jennerian" approach to vaccination. J Infect Dis 1986 May; 153(5):815-22

Levine MM, Kaper JB, Black RE, Clements ML. New knowledge on pathogenesis of bacterial enteric infections as applied to vaccine development. Microbiol Rev $1983\ \text{Dec};47(4):510-50$

Levine MM, Black RE, Brinton CC, Jr., Clements ML, Fusco P, Hughes TP,

O'Donnell S, Robins-Browne R, Wood S, Young CR. Reactogenicity, immunogenicity, and efficacy studies of <u>Escherichia coli</u> type 1 somatic pili parenteral vaccine in man. Scand J Infect Dis 1982; (suppl 33):83-95

Levine MM. Travellers' diarrhoea: prospects for successful immunoprophylaxis. Scand J Gastroenterol 1983:18(suppl 84):121-34 **

Rowley D, La Brooy J. Intestinal immune responses in relation to diarrhoeal diseases. J Diarrhoeal Dis Res 1986 Mar:4(1):1-9 **

Saif LJ, Smith KL. Enteric viral infections of calves and passive immunity. J Dairy Sci 1985 Jan:68(1):206-28 **

Shiner M. Autoimmunity in gastrointestinal diseases. Arq Gastroenterol 1986 Apr-Jun:23(2):99-103 **

Metabolism.

Awouters F, Niemegeers CJE, Janssen PAJ. Pharmacology of antidiarrheal drugs. Annu Rev Pharmacol Toxicol 1983;23:279-301 **

Balistreri WF, Heubi JE, Suchy FJ. Bile acid metabolism: relationship of bile acid malabsorption and diarrhea. J Pediatr Gastroenterol Nutr 1983;2(1): 105-21

Bennet J. A review of antidiarrhoeal compounds. <u>In:</u> Gough D, ed. The control of diarrhoea in clinical practice. London: The Royal Society of Medicine, 1978:1-8 (Royal Society of Medicine International Congress and Symposium series, 5)

Bradshaw MJ, Harvey RF. Antidiarrhoeal agents: clinical pharmacology and therapeutic use. Curr Ther 1983 Feb:65-73 **

Caspary WF. Diarrhoea associated with carbohydrate malabsorption. Clin Gastroenterol 1986 Jul:15(3):631-55

Chang EB, Field M. Intestinal electrolyte transport and diarrheal disease. In: Kern \cdot F, Jr., Blum AL, eds. The gastroenterology annual, 1/1983. Amsterdam: Eisevier, 1983:148-80

da Rocha JM. [Disaccharidase disorders]. Bol Inst Puericult 1963 Apr;20(1):
311-22 **

Donowitz M, Wicks J, Cusolito S, Sharp GW. Pharmacotherapy of diarrheal diseases: an approach based on physiologic principles. KROC Found Ser 1984;17:329-59

Keusch GT, Donowitz M. Pathophysiological mechanisms of diarrhoeal diseases: diverse aetiologies and common mechanisms. Scand J Gastroenterol 1983;18 (suppl 84):33-43 **

Love AHG. Metabolic response to malnutrition: its relevance to enteral feeding. Gut 1986 Nov;27(suppl 1):9-13 **

Mahalanabis D. Patra FC. In search of a super oral rehydration solution: can

optimum use of organic solute-mediated sodium absorption lead to the development of an absorption promoting drug? J Diarrhoeal Dis Res 1983 Jun;1 (2):76-81 **

Mathan VI. Small intestine failure. <u>In</u>: Taylor TG, Jenkins NK, eds. Proceedings of the XIII International Congress of Nutrition. London: Libbey, 1986:671-4 **

Milla PJ. Disorders of electrolyte absorption. Clin Gastroenterol 1982 Jan;11(1):31-46

Ooms L. Alterations in intestinal fluid movement. Scand J Gastroenterol 1983;18(suppl 84):65-77 **

Ooms L, Degryse A. Pathogenesis and pharmacology of diarrhea. Vet Res Commun 1986 Sep; 10(5):355-97

Wagh MG, Ghooi RB, Shetty RK. Lactose intolerance; physiological, clinical and therapeutic considerations. Indian J Pediatr 1984 Nov-Dec;51(413):671-81

Microbiology

Brooks JB. Review of frequency-pulsed electron-capture gas-liquid chromatography studies of diarrheal diseases caused by members of the family Enterobacteriaceae, Clostridium difficile, and rotavirus. J Clin Microbiol 1986 Nov.24(5):687-91 **

Buchino JJ, Suchy FJ, Snyder JW. Bacterial diarrhea in infants and children. Perspect Pediatr Pathol 1984 Summer;8(2):163-80 **

Guerrant RL, Sauer KT. Selective use of microbiological procedures for identification of etiologic agents of diarrheal illness. J Food Saf 1981;3: 145-64 **

Simon GL, Gorbach SL. Intestinal flora in health and disease. Gastroenterology 1984 Jan;86(1):174-93

Simon GL, Gorbach SL. Intestinal microflora. Med Clin North Am 1982 May;66 (3):557-74

Mortality

Ashworth A. International differences in child mortality and the impact of malnutrition. Hum Nutr Clin Nutr 1982;36C(4):279-88 **

Briscoe J, Feachem RG, Rahaman MM. Measuring the impact of water supply and sanitation facilities on diarrhoea morbidity: prospects for case-control methods. Geneva: World Health Organization, 1985. 71 p. (WHO/CWS/85.3; CDD/OPR/85.1) **

Feachem RG, Koblinsky MA. Interventions for the control of diarrhoeal diseases among young children: measles immunization. Bull WHO 1983;61(4): 641-52 **

Feachem RG. The role of water supply and sanitation in reducing mortality in

China, Costa Rica, Kerala State (India) and Sri Lanka. <u>In</u>: Halstead SB, Walsh JA, Warren KS, eds. Good health at low cost; proceedings of a conference, held at the Bellagio Conference Center. Italy, 29 Apr-3 May 1985:191-8

Sunoto. Diarrhoeal problems in Southeast Asia. Southeast Asian J Trop Med Public Health 1982 Sep;13(3):306-18 **

Occurrence

MacDonald KL, Cohen ML. Epidemiology of travelers' diarrhea: current perspectives. Rev Infect Dis 1986 May-Jun;8(suppl 2):S117-21 **

Monsur KA. Epidemiology of Escherichia coli - an important but neglected field [editorial]. J Diarrhoeal Dis Res 1985 Sep;3(3):128-30

Nelson JD. Etiology and epidemiology of diarrheal diseases in the United States. Am J Med 1985 Jun 28;78(suppl 6B):76-80 **

Nunoue T. Epidemiological aspect of viral diarrhea in tropical area. Jpn J Trop Med Hyg 1985 Mar;13(1):51 **

Pickering LK. Infections in day care. Pediatr Infect Dis 1987 Jun;6(6): 614-7 **

Saran M, Dabral M, Srivastava RN, Sharma VK. Epidemiology of human rotavirus diarrhoea -- a review. J Indian Assoc Commun Dis 1982 Sep-Dec;5(3-4):50-7 **

Shane SM, Montrose MS. The occurrence and significance of Campylobacter jejuni in man and animals. Vet Res Commun 1985 Jul; 9(3):167-98

Steffen R. Epidemiologic studies of travelers' diarrhea, severe gastrointestinal infections, and cholera. Rev Infect Dis 1986 May-Jun;8(suppl 2):S122-30 **

Steffen R. Epidemiology of travellers' diarrhoea. Scand J Gastroenterol 1983:18(suppl 84):5-17 **

Sunoto. Diarrhoeal problems in Southeast Asia. Southeast Asian J Trop Med Public Health 1982 Sep;13(3):306-18 **

Taylor DN, Echeverria P. Etiology and epidemiology of travelers' diarrhea in Asia. Rev Infect Dis 1986 May-Jun;8(suppl 2):S136-41 **

van Zijl WJ. Studies on diarrhoeal diseases in seven countries by the WHO Diarrhoeal Diseases Advisory Team. Bull WHO 1966;35(2):249-61 **

Oral Therapy

Abed FH. Household teaching of oral rehydration therapy in rural Bangladesh. J Indian Med Assoc 1987 Jul;85(7):205-9

Ahmed HS, Molla AM. Rice-based oral rehydration. J Diarrhoeal Dis Res. 1987 Mar; 5(1):1-6 **

Banwell JG. Treatment of travelers' diarrhea: fluid and dietary management. Rev Infect Dis 1986 May-Jun; $8(\sup 2)$: $8(\sup 2)$:

Black RE. The prophylaxis and therapy of secretory diarrhea. Med Clin North Am 1982 May;66(3):611-21

Carpenter CCJ. Oral rehydration: is it as good as parenteral therapy? [editorial]. N Engl J Med 1982 May 6;306(18):1103-4 **

Chowdhury AMR, D'Souza S. A design and field methods for monitoring impact on mortality of an oral therapy programme. Dhaka: International Centre for Diarrhoeal Disease Research, Bangladesh, 1982. 23 p. (ICDDR,B working paper 27) **

Feachem RG. Preventing diarrhoea: what are the policy options? Health Policy Plann 1986;1(2):109-17 **

Ghatikar KN. Oral rehydration therapy--an overview. Q Med Rev 1980 Oct;31 (4):1-12

Gopalan C. Oral rehydration therapy - the need for a proper perspective. Food Nutr Bull 1986 Sep;8(3):69-70 **

Greenough WB, III. Principles and prospects in the treatment of cholera and related dehydrating diarrheas. In: Ouchterlony O, Holmgren J, eds. Cholera and related diarrheas: molecular aspects of a global health problem. Basel: Karger, 1980:211-8 (43rd Nobel Symposium) **

Greenough WB, III. "Super ORT" [editorial]. J Diarrhoeal Dis Res 1983 Jun;1 (2):74-5 **

Leung AKC, Darling P, Auclair C. Oral rehydration therapy: a review. J R Soc Health 1987;107(2):64-7 **

Mahalanabis D, Patra FC. In search of a super oral rehydration solution: can optimum use of organic solute-mediated sodium absorption lead to the development of an absorption promoting drug? J Diarrhoeal Dis Res 1983 Jun;1 (2):76-81 **

Mahalanabis D, Merson MH, Barua D. Oral rehydration therapy - recent advances. World Health Forum 1981;2(2):245-9

Meeuwisse GW. High sugar worse than high sodium in oral rehydration solutions. Acta Paediatr Scand 1983 Mar;72(2):161-6 **

Molla AM, Sarker SA, Hossain M, Molla A, Greenough WB, III. Rice-powder electrolyte solution as oral therapy in diarrhoea due to Vibrio cholerae and Escherichia coli. Lancet 1982 Jun 12;1(8285):1317-9 **

Nalin DR. Oral replacement of water and electrolyte losses due to travellers' diarrhoea. Scand J Gastroenterol 1983;18(suppl 84):95-8 **

Netchvolodoff CV, Hargrove MD, Jr. Recent advances in the treatment of diarrhea. Arch Intern Med 1979 Jul;139(7):813-6 **

Rohde JE, Cash RA, Guerrant RL, Mahalanabis D, Molla AM, Valyasevi A,

Therapeutic interventions in diarrhea. <u>In</u>: Chen LC, Scrimshaw NS, eds. Diarrhea and malnutrition: interactions, mechanisms, and interventions. New York: Plenum, 1983:287-95 **

Samadi AR, Islam MR, Aziz KMS. ICDDR,B model for treatment of diarrhoeal diseases. Dhaka: International Centre for Diarrhoeal Disease Research, Bangladesh, Jul 1982. 12 p. (ICDDR,B special publication no. 19) **

Sunoto. Oral rehydration salts: a simple and appropriate tool against dehydration due to diarrhoea. Paediatr Indones 1981 Mar-Apr;21(3-4):90-100 **

Tulloch J, Burton P. Global access to oral rehydration salts and use of oral rehydration therapy. World Health Stat Q 1987;40(2):110-5

Parasitology

Armstrong M. Cryptosporidiosis. Med Lab Sci 1987 Jul;44(3):280-4 **

Casemore DP, Sands RL, Curry A. <u>Cryptosporidium</u> species a "new" human pathogen. J Clin Pathol 1985 Nov;38(11):1321-36 **

Fayer R, Ungar BLP. <u>Cryptosporidium</u> spp. and cryptosporidiosis. Microbiol Rev 1986 Dec;50(4):458-83 **

Janoff EN, Reller LB. <u>Cryptosporidium</u> species, a protean protozoan. J Clin Microbiol 1987 Jun;25(6):967-75

Navin TR, Juranek DD. Cryptosporidiosis: clinical, epidemiologic, and parasitologic review. Rev Infect Dis 1984 May-Jun;6(3):313-27 **

Navin TR. Cryptosporidiosis in humans: review of recent epidemiologic studies. Eur J Epidemiol 1985 Jun;1(2):77-83 **

Tharavanij S. Pathogenesis of diarrhoea caused by parasites. Southeast Asian J Trop Med Public Health 1982 Sep;13(3):331-8 **

Wright SG. Parasites and travellers' diarrhoea. Scand J Gastroenterol 1983; 18(suppl 84):25-9 **

Pathology -

Cooper BT. Diarrhoea as a symptom. Clin Gastroenterol 1985 Jul;14(3): 599-613 **

Physiopathology

Cantey JR. Infectious diarrhea. Pathogenesis and risk factors. Am J Med 1985 Jun 28;78(suppl 6B):65-75 **

Carpenter CCJ. The pathophysiology of secretory diarrheas. Med Clin North Am $1982\ \mathrm{May};66\ (3):597-610\ **$

Caspary WF. Diarrhoea associated with carbohydrate malabsorption. Clin

Gastroenterol 1986 Jul;15(3):631-55

Cavalieri SJ, Bohach GA, Snyder IS. <u>Escherichia coli</u> alpha-hemolysin: characteristics and probable role in pathogenicity. Microbiol Rev 1984 Dec;48 (4):326-43

Chadwick VS. Small intestinal secretion in disease. Scand J Gastroenterol 1983;18(suppl 87):91-7 **

Chang EB, Field M. Intestinal electrolyte transport and diarrheal disease. In: Kern F, Jr., Blum AL, eds. The gastroenterology annual, 1/1983. Amsterdam: Elsevier, 1983:148-80

Chang EB, Fedorak RN. Prostaglandins in diarrheal disease [editorial]. J Pediatr Gastroenterol Nutr 1985 Jun;4(3):341-4

Cooke EM. Escherichia coli - an overview. J Hyg (Lond) 1985 Dec;95(3):523-30

Cunha BA. The toxigenic diarrheas. Intern Med 1987 Feb;8(2):92-110 **

Dobbins JW, Binder HJ. Pathophysiology of diarrhoea: alterations in fluid and electrolyte transport. Clin Gastroenterol 1981 Sep;10(3):605-25 **

Evans N. Pathogenic mechanisms in bacterial diarrhoea. Clin Gastroenterol 1979 Sep;8(3):599-623 **

Feldman M, Schiller LR. Disorders of gastrointestinal motility associated with diabetes mellitus. Ann Intern Med 1983 Mar:98(3):378-84 **

Fondacaro JD. Intestinal ion transport and diarrheal disease. Am J Physiol $1986 \, \text{Jan}; 250(1, \, \text{pt} \, 1): 61-8 \, \text{**}$

Fromm H, Malavolti M. Bile acid-induced diarrhoea. Clin Gastroenterol 1986 Jul;15(3):567-82 **

Gardner JD. Plasma VIP in patients with watery diarrhea syndrome. Am J Dig Dis 1978 Apr;23(4):370-6

Gracey M, Burke V. Characteristics of <u>Aeromonas</u> species and their association with human diarrhoeal disease. J Diarrhoeal Dis Res 1986 Jun;4(2):70-3 **

Greenough WB, III. Bacterial diarrhoeal diseases: current concepts on etiology and pathogenesis. Southeast Asian J Trop Med Public Health 1982 Sep;13(3):319-24 **

Greenough WB, III. Protozoal, bacterial and viral diarrhoea: a common mechanism [editorial]. J Diarrhoeal Dis Res $1984 \, Jun; 2(2):68$

Gross RJ. Escherichia coli, J Infect 1983 Nov:7(3):177-92

Gross RJ, Rowe B. <u>Escherichia</u> <u>coli</u> diarrhoea. J Hyg (Lond) 1985 Dec;95(3):531-50 **

Gyr K. Infectious diarrhoea and gastrointestinal hormones: potential therapeutic implications. Scand J Gastroenterol 1983;18(suppl 84):135-40 **

Gyr K. Toxin receptors and their pathogenetic significance. Acta Histochem 1984;(suppl 29):S95-102 **

Harries JT. Mechanisms and mediators of intestinal secretion in the small intestine. J Pediatr Gastroenterol Nutr 1982 Dec;1(4):575-82 **

Hawkey CJ, Rampton DS. Prostaglandins and the gastrointestinal mucosa: are they important in its function, disease, or treatment? Gastroenterology 1985 Nov:89(5):1162-88 **

Holmgren J, Svennerholm A-M. Pathogenic mechanisms and new perspectives in the treatment and prevention of enteric infections. Scand J Gastroenterol 1982;17(suppl 77):47-59 **

Keusch GT, Donowitz M. Pathophysiological mechanisms of diarrhoeal diseases: diverse aetiologies and common mechanisms. Scand J Gastroenterol 1983;18 (suppl 84):33-43 **

Keusch GT, Donohue-Rolfe A, Jacewicz M. Shigella toxin(s); description and role in diarrhea and dysentery. Pharmacol Ther 1982;15(3):403-38 **

Klemm P. Fimbrial adhesions of <u>Escherichia</u> <u>coli</u>. Rev Infect Dis 1985 May-Jun;7(3):321-40

Korman SH, Berant M, Alon U. Review: prostaglandins in diarrheal states. Isr J Med Sci 1981 Dec:17(12):1109-13

Krejs GJ. VIPoma syndrome. Am J Med 1987 May 29:82(suppl 5B):37-48 **

Levine MM. Escherichia coli that cause diarrhea: enterotoxigenic, enteropathogenic, enteroinvasive, enterohemorrhagic, and enteroadherent. J Infect Dis 1987 Mar:155(3):377-89 **

Levine MM, Kaper JB, Black RE, Clements ML. New knowledge on pathogenesis of bacterial enteric infections as applied to vaccine development. Microbiol Rev $1983\ \text{Dec};47(4):510-50$ **

Mathias JR, Clench MH. Review: pathophysiology of diarrhea caused by bacterial overgrowth of the small intestine. Am J Med Sci 1985 Jun;289(6): 243-8 **

Metz SA, McRae JR, Robertson RP. Prostaglandins as mediators of paraneoplastic syndromes: review and up-date. Metabolism 1981 Mar; 30(3): 299-316

Milla PJ. Intestinal motility and its disorders. Clin Gastroenterol 1986 $\mbox{Jan;}15(1):121-36$

Minami H, McCallum RW. The physiology and pathophysiology of gastric emptying in humans. Gastroenterology 1984 Jun;86(6):1592-1610 **

Moon MW. Mechanisms in the pathogenesis of diarrhea: a review. J Am Vet Med Assoc 1978 Feb 15;172(4):443-8

Moriarty KJ, Turnberg LA. Bacterial toxins and diarrhoea. Clin Gastroenterol 1986 Jul;15(3):529-43 **

Neter E. Enteropathogenicity: recent developments. Klin Wochenschr 1982 Jul

15;60(14):699-701 **

Ooms L. Alterations in intestinal fluid movement. Scand J Gastroenterol 1983;18(suppl 84):65-77 **

Ooms L, Degryse A. Pathogenesis and pharmacology of diarrhea. Vet Res. Commun 1986 Sep: 10(5): 355-97

Powell DW. Enterotoxigenic diarrhea: mechanisms and prospects for therapy. Pharmacol Ther 1984;23(3):407-16 **

Rahaman MM, Wahed MA. Direct nutrient loss in diarrhea. <u>In: Chen LC, Scrimshaw NS, eds.</u> Diarrhea and malnutrition: interactions, mechanisms, and interventions. New York: Plenum, 1983:155-60 **

Ramband JC, Hauterfeuille M, Ruskone A, Jacquenod P. Diarrhoea due to circulating agents. Clin Gastroenterol 1986 Jul;15(3):603-29

Rask-Madsen J. Eicosanoids and their role in the pathogenesis of diarrhoeal diseases. Clin Gastroenterol 1986 Jul;15(3):545-66 **

Rask-Madsen J. The role of eicosanoids in the gastrointestinal tract. Scand J Gastroenterol 1987;22(suppl 127):7-19 **

Read NW. Diarrhoee motrice. Clin Gastroenterol 1986 Jul;15(3):657-86 **

Read NW. Speculations on the role of motility in the pathogenesis and treatment of diarrhoea. Scand J Gastroenterol 1983;18(suppl 84):45-63 **

Rennels MB, Levine MM. Classical bacterial diarrhea: perspectives and update - Salmonella, Shigella, Escherichia coli, Aeromonas and Plesiomonas. Pediatr Infect Dis 1986 Jan;5(1):S91-100 **

Rowe B. The role of Escherichia coli in gastroenteritis. Clin Gastroenterol 1979 Sep;8(3):625-44

Rowland HAK. The pathogenesis of diarrhoea. Trans R Soc Trop Med Hyg 1978; 72(3):289-302

Sack R8. Human diarrheal disease caused by enterotoxigenic <u>Escherichia</u> <u>coli</u>. Annu Rev Microbiol 1975;29:333-53

Shiau YF. Clinical and laboratory approaches to evaluate diarrheal disorders. CRC Crit Rev Clin Lab Sci 1987;25(1):43-69 **

Siegenbeek van Heukelom J. Physiological aspects of absorption and secretion in intestine. Vet Res Commun 1986 Sep;10(5):341-54

Simon GL, Gorbach SL. Intestinal microflora. Med Clin North Am 1982 May;66 (3):557-74

Takeda Y, Shimonishi Y, Yamamoto T, Takeda T. [Enterotoxins produced by enterotoxigenic <u>Escherichia coli</u>]. Tanpakushitsu Kakusan Koso 1986 Mar;31 (suppl 4):324-52

Tharavanij S. Pathogenesis of diarrhoea caused by parasites. Southeast, Asian

39

J Trop Med Public Health 1982 Sep;13(3):331-8 **

Turnberg LA. The pathophysiology of diarrhoea. Clin Gastroenterol 1979 Sep;8 (3):551-68

Wagh MG, Ghooi RB, Shetty RK. Lactose intolerance; physiological, clinical and therapeutic considerations. Indian J Pediatr 1984 Nov-Dec;51(413):671-81

Prevention and Control

Ashworth A, Feachem RG. Interventions for the control of diarrhoeal diseases among young children: prevention of low birth weight. Bull WHO 1985;63(1):165-84 **

Blaser MJ. Environmental interventions for the prevention of travelers' diarrhea. Rev Infect Dis 1986 May-Jun;8(suppl 2):S142-50 **

Creese AL. Cost effectiveness of potential immunization interventions against diarrhoeal disease. Soc Sci Med 1986;23(3):231-40 **

Cutting WAM, Hawkins P. The role of water in relation to diarrhoeal disease. J Trop Med Hyg 1982 Feb; 85(1):31-9 **

Cyjetanovic B. Health effects and impact of water supply and sanitation. World Health Stat 0.1986:39(1):105-17

Cvjetanovic B, Chen L, Kronmall R, Rohde C, Suskind R. Measuring and evaluating diarrhea and malabsorption in association with village water supply and sanitation: a review of the Food Wastage/Sanitation Cost Benefit Methodology Project (Guatemala). Arlington, Virginia: Water and Sanitation for Health Project, 1981. 36 p. (WASH technical report, 12)

Cyjetanovic B. Sanitation versus immunization in control of enteric and diarrhoeal diseases. Prog Water Technol 1979;11(1-2):81-7 **

Edelman R. Prevention and treatment of infectious diarrhea: speculations on the next 10 years. Am J Med 1985 Jun 28:78(suppl 68):99-106 **

Esrey SA, Feachem RG, Hughes JM. Interventions for the control of diarrhoeal diseases among young children: improving water supplies and excreta disposal facilities. Bull WHO 1985;63(4):757-72**

Feachem R. Priorities for diarrhoeal disease control: water, excreta, behaviour and diarrhoea. Diarrhoea Dial 1981;(4):4-5

Feachem RG, Hogan RC, Merson MH. Diarrhoeal disease control: reviews of potential interventions. Bull WHO 1983;61(4):637-40 **

Feachem RG, Koblinsky MA. Interventions for the control of diarrhoeal diseases among young children: measles immunization. Bull WHO 1983;61(4): 641-52 **

Feachem RG. Interventions for the control of diarrhoeal diseases among young children: promotion of personal and domestic hygiene. Bull WHO 1984;62(3):467-76 **

Feachem RG. Preventing diarrhoea: what are the policy options? Health Policy Plann 1986;1(2):109-17 **

Feachem RG. The role of water supply and sanitation in reducing mortality in China, Costa Rica, Kerala State (India) and Sri Lanka. <u>In</u>: Halstead SB, Walsh JA, Warren KS, eds. Good health at low cost; proceedings of a conference, held at the Bellagio Conference Center, Italy, 29 Apr-3 May 1985:191-8

Feachem RG. Vitamin A deficiency and diarrhoea: a review of interrelation-ships and their implications for the control of xerophthalmia and diarrhoea. Trop Dis Bull 1987 Mar;84(3):R1-16 **

Holmgren J, Svennerholm A-M. Pathogenic mechanisms and new perspectives in the treatment and prevention of enteric infections. Scand J Gastroenterol 1982;17(suppl 77):47-59 **

Howard P. Diarrhoeal diseases--priorities in research and intervention. Papua New Guinea Med J 1986 Jun; 29(2):125-9

Kawata K. Water and other environmental interventions—the minimum investment concept. Am J Clin Nutr 1978 Nov;31(11):2114-23 **

Khan MU. Interruption of transmission of diarrhoeal agents. <u>In:</u> Programme, papers and abstracts of the Third Asian Conference on Diarrhoeal Diseases, Bangkok, 10-14 Jun 1985:173-80

Kuo C. Measures to control diarrhoeal diseases--environmental sanitation. Regional Meeting on Cholera and Diarrhoeal Diseases, Alexandria, 1-5 Jun 1978. Alexandria: Regional Office for the Eastern Mediterranean, World Health Organization, 1978. 6 p. **

Lee EW. Safe water supply and sanitation in diarrhoeal diseases control. Regional Planning Meeting on Diarrhoeal Diseases Control, Manila, 5-7 Jun 1979. Manila: Regional Office of the Western Pacific, World Health Organization, 1979. 5 p. (WPR/BVD/DDC/79.3) **

Levine MM. Travellers' diarrhoea: prospects for successful immunoprophylaxis. Scand J Gastroenterol 1983;18(suppl 84):121-34 **

Litvinov SK, Merson MH, Oblapenko GP, Herniman R, Lishnevshil MS. [The WHO program for controlling diarrheic diseases: its status and development outlook - the organizational and operative components of the program]. Zh Mikrobiol Epidemiol Immunobiol 1985 Jun; (6):93-8

Porter P, Linggood MA. Development of oral vaccines for preventing diarrhoea caused by enteropathogenic <u>Escherichia coli</u>. J Infect 1983 Mar;6(2):111-21

Rowland MGM. The weanling's dilemma: are we making progress? Acta Paediatr Scand 1986; (suppl 323):33-42 $\,\,$

Sarkar U. Community participation in the control of diarrhoea. J Indian Med Assoc 1987 Jul;85(7):210-2

Walsh JA, Warren KS. Selective primary health care: an interim strategy for disease control in developing countries. Soc Sci Med 1980;14C(2):145-63 **

Therapy

DiJoseph JF, Taylor JA, Mir GN. Alpha+2 receptors in the gastrointestinal system: a new therapeutic approach. Life Sci 1984 Sep 3;35(10):1031-42

Edelman R. Prevention and treatment of infectious diarrhea: speculations on the next 10 years. Am J Med 1985 Jun 28;78(suppl 6B):99-106 **

Gotz VP, Rand KH. Medical management of antimicrobial-associated diarrhea and colitis. Pharmacotherapy 1982 Mar-Apr;2(2):100-9 **

Gyr K. Infectious diarrhoea and gastrointestinal hormones: potential therapeutic implications. Scand J Gastroenterol 1983;18(suppl 84):135-40 **

Holmgren J, Svennerholm A-M. Pathogenic mechanisms and new perspectives in the treatment and prevention of enteric infections. Scand J Gastroenterol 1982;17(suppl 77):47-59 **

Samadi AR, Islam MR, Aziz KMS. ICDDR,B model for treatment of diarrhoeal diseases. Dhaka: International Centre for Diarrhoeal Disease Research, Bangladesh, Jul 1982. 12 p. (ICDDR,B special publication no. 19) **

Ziegler MM. Short bowel syndrome in infancy: etiology and management. Clin Perinatol 1986 Mar;13(1):163-73

Transmission

Khan MU. Interruption of transmission of diarrhoeal agents. <u>In: Programme, papers and abstracts of the Third Asian Conference on Diarrhoeal Diseases, Bangkok, 10-14 Jun 1985:173-80</u>

Veterinary

Acres SD. Enterotoxigenic <u>Escherichia coli</u> infections in newborn calves: a review. J Dairy Sci 1985 Jan;68(1):229-56 **

Blackwell TE. Enteritis and diarrhea. Vet Clin North Am [Large Anim Pract] 1983 Nov;5(3):557-70 **

Bohl EH. Rotaviral diarrhea in pigs: brief review. J Am Vet Med Assoc 1979 Mar 15:174(6):613-5 **

Cilli V, Castrucii G. Viral diarrhea of young animals: a review. Comp Immunol Microbiol Infect Dis 1981;4(3-4):229-42 **

Fettman MJ, Rollin RE. Antimicrobial alternatives for calf diarrhea: iron chelators or competitors. J Am Vet Med Assoc 1985 Oct 1;187(7):746-8

Moon MW. Mechanisms in the pathogenesis of diarrhea: a review. J Am Vet Med Assoc 1978 Feb 15;172(4):443-8

Ooms L, Degryse A. Pathogenesis and pharmacology of diarrhea. Vet Res Commun $1986~{\rm Sep;}10(5):355-97$

Riviere JE, Boosinger TR, Everson RJ. Inorganic arsenic toxicosis in cattle. Mod Vet Pract 1981 Mar;62(3):209-11 **

Saif LJ, Smith KL. Enteric viral infections of calves and passive immunity. J Dairy Sci 1985 Jan;68(1):206-28 **

Saw SP. The causes and economic significance of enteric infections in domestic animals. In: Tzipori S, Barnes G, Bishop R, Holmes I, Robins-Browne R, eds. Infectious diarrhoea in the young: strategies for control in humans and animals; proceedings of an International Seminar on Diarrhoeal Disease in South East Asia and the Western Pacific Region, Geelong, 10-15 Feb 1985. Amsterdam: Elsevier, 1985:160-2 **

Torres-Medina A, Schlafer DH, Mebus CA. Rotaviral and coronaviral diarrhea. Vet Clin North Am (Food Anim Pract) 1985 Nov:1(3):471-93 **

DIARRHEA, ACUTE

Bhattacharya SK. Acute diarrhoeal diseases in newborn. J Indian Med Assoc 1987 Jul;85(7):213, 209

Blaser MJ. Infectious diarrheas: acute, chronic, and iatrogenic [editorial]. Ann Intern Med 1986 Nov;105(5):785-7

Eichenwald HF, McCracken GH, Jr. Acute diarrheal disease. Med Clin North Am 1970 Mar;54(2):443-53

Khin-Maung U. Recent research on acute diarrhoea in Burma. DMR Bull 1987 Jan;1(4):1-19 **

Pickering LK. Evaluation of patients with acute infectious diarrhea. Pediatr Infect Dis 1985 May-Jun;4(suppl 3):S13-9

Rohde JE. Selective primary health care: strategies for control of disease in the developing world. XV. Acute diarrhea. Rev Infect Dis 1984 Nov-Dec;6(6): 840-54 **

Complications

Meeuwisse GW. High sugar worse than high sodium in oral rehydration solutions, Acta Paediatr Scand 1983 Mar;72(2):161-6 **

Diagnosis

Guerrant RL, Shields DS, Thorson SM, Schorling JB, Groschel DHM. Evaluation and diagnosis of acute infectious diarrhea. Am J Med 1985 Jun 28;78(suppl 68):91-8 **

Williams EK, Lohr JA, Guerrant RL. Acute infectious diarrhea. II. Diagnosis, treatment and prevention. Pediatr Infect Dis 1986 Jul-Aug; 5(4):458-65

Diet Therapy

Booth IW, Cutting WAM. Current concepts in the management of acute diarrhoea in children. Postgrad Doc - Middle-East 1984 Jul:418-24 **

Brown KH, MacLean WC, Jr. Nutritional management of acute diarrhea: an appraisal of the alternatives. Pediatrics 1984 Feb;73(2):119-25 **

Hamilton JR. Treatment of acute diarrhea. Pediatr Clin North Am 1985-Apr;32(2):419-27-**

Heim T. Requirements and utilization of macronutrients in enteral and parenteral nutrition in acute and chronic diarrhea. In: Lebenthal E, ed. Chronic diarrhea in children. New York: Raven Press, 1984:541-57 **

Jelliffe EFP, Jelliffe DB, Feldon K, Ngokwey N. Traditional practices concerning feeding during and after diarrhoea (with special reference to acute dehydrating diarrhoea in young children). World Rev Nutr Diet 1987;53:218-95

Kinoti SN, Wasunna A, Turkish J, Gateere R, Desai M, Agwanda R, Juma R. A comparison of the efficacy of maize-based ORS and standard W.H.O. ORS in the treatment of acute childhood diarrhea at Kenyatta National Hospital, Nairobi, Kenya: results of a pilot study. East Afr Med J 1986 Mar;63(3):168-74

Drug Therapy

Booth IW, Cutting WAM. Current concepts in the management of acute diarrhoea in children. Postgrad Doc - Middle-East 1984 Jul:418-24 **

DuPont HL. Nonfluid therapy and selected chemoprophylaxis of acute diarrhea. Am J Med 1985 Jun 28;78(6B):81-90 **

Fedorak RN, Field M. Antidiarrheal therapy: prospects for new agents. Dig Dis Sci 1987 Feb;32(2):195-205 **

Hughes S. Acute secretory diarrhoeas; current concepts in pathogenesis and treatment. Drugs 1983;26:80-90 **

Lambert HP. Antimicrobial agents in diarrhoeal disease. Clin Gastroenterol 1979 Sep;8(3):827-33 **

Netchvolodoff CV, Hargrove MD, Jr. Recent advances in the treatment of diarrhea. Arch Intern Med 1979 Jul;139(7):813-6 **

Etiology

Banatvala JE. The role of viruses in acute diarrhoeal disease. Clin Gastroenterol 1979 Sep;8(3):569-98

Guerrant RL, Lohr JA, Williams EK. Acute infectious diarrhea. I. Epidemiology, etiology and pathogenesis. Pediatr Infect Dis 1986 May-Jun; 5(3):353-9

Mata L, Urrutia JJ, Simhon A. Infectious agents in acute and chronic diarrhea

of childhood. In: Lebenthal E, ed. Chronic diarrhea in children. New York: Raven Press. 1984:237-52 **

Fluid Therapy

Booth IW, Cutting WAM. Current concepts in the management of acute diarrhoea in children. Postgrad Doc - Middle-East 1984 Jul:418-24 **

Netchvolodoff CV, Hargrove MD, Jr. Recent advances in the treatment of diarrhea. Arch Intern Med 1979 Jul;139(7):813-6 **

Metabolism

Auricchio S. Peptide digestion and absorption in the small intestinal mucosa during acute and chronic diarrhea. In: Lebenthal E, ed. Chronic diarrhea in children. New York: Raven Press, $19\overline{84}:179-91$

Molla A, Molla AM, Sarker SA, Khatoon M, Rahaman MM. Effects of acute diarrhea on absorption of macronutrients during disease and after recovery. <u>In:</u> Chen LC, Scrimshaw NS, eds. Diarrhea and malnutrition: interactions, mechanisms, and interventions. New York: Plenum, 1983:143-54

Microbiology

Guerrant RL, Shields DS, Thorson SM, Schorling JB, Groschel DHM. Evaluation and diagnosis of acute infectious diarrhea. Am J Med 1985 Jun 28;78(suppl 6B):91-8 **

Mortality

Snyder JD, Merson MH. The magnitude of the global problem of acute diarrhoeal disease: a review of active surveillance data. Bull WHO 1982;60(4):605-13 **

Occurrence

Guerrant RL, Lohr JA, Williams EK. Acute infectious diarrhea. I. Epidemiology, etiology and pathogenesis. Pediatr Infect Dis 1986 May-Jun;5 (3):353-9

Pickering LK, Bartlett AV, Woodward WE. Acute infectious diarrhea among children in day care: epidemiology and control. Rev Infect Dis 1986 Jul-Aug;8 (4):539-47 **

Snyder JD, Merson MH. The magnitude of the global problem of acute diarrhoeal disease: a review of active surveillance data. Bull WHO 1982;60(4):605-13 **

Oral Therapy

Booth IW, Levine MM, Harries JT. Oral rehydration therapy in acute diarrhoea

Diarrhoeal Diseases 45

in childhood. J Pediatr Gastroenterol Nutr 1984 Sep;3(4):491-9 **

Carpenter CCJ. Oral rehydration: is it as good as parenteral therapy? [editorial]. N Engl J Med 1982 May 6;306(18):1103-4 **

Elliott EJ, Walker-Smith JA, Farthing MJG. The role of bicarbonate and base precursors in treatment of acute gastroenteritis. Arch Dis Child 1987 Jan;62 (1):91-5 **

Hirschhorn N. The treatment of acute diarrhea in children: an historical and physiological perspective. Am J Clin Nutr 1980 Mar;33(3):637-63 **

Kinoti SN, Wasunna A, Turkish J, Gateere R, Desai M, Agwanda R, Juma R. A comparison of the efficacy of maize-based ORS and standard W.H.O. ORS in the treatment of acute childhood diarrhoea at Kenyatta National Hospital, Nairobi, Kenya: results of a pilot study. East Afr Med J 1986 Mar;63(3):168-74

Meeuwisse GW. High sugar worse than high sodium in oral rehydration solutions. Acta Paediatr Scand 1983 Mar;72(2):161-6 **

Netchvolodoff CV, Hargrove MD, Jr. Recent advances in the treatment of diarrhea. Arch Intern Med 1979 Jul;139(7):813-6 **

Sack DA. Treatment of acute diarrhoea with oral rehydration solution. Drugs 1982 Jan-Feb;23(1-2):150-7 **

Physiopathology

Banatvala JE. The role of viruses in acute diarrhoeal disease. Clin Gastroenterol 1979 Sep;8(3):569-98

Guerrant RL, Lohr JA, Williams EK. Acute infectious diarrhea. I. Epidemiology, etiology and pathogenesis. Pediatr Infect Dis 1986 May-Jun; 5(3):353-9

Hughes S. Acute secretory diarrhoeas; current concepts in pathogenesis and treatment. Drugs 1983;26:80-90 **

Sack RB. Escherichia coli and acute diarrheal disease. Ann Intern Med 1981 $Jan;94(1):\overline{129-30}$

Prevention and Control

Merson MH. The ylobal problem of acute diarrhoeal diseases and the WHO Diarrhoeal Diseases Control Programme. <u>In:</u> Takeda Y, Miwatani T, eds. Bacterial diarrhoeal diseases. Tokyo: KTK Scientific Publishers, 1985:1-10

Pickering LK, Bartlett AV, Woodward WE. Acute infectious diarrhea among children in day care: epidemiology and control. Rev Infect Dis 1986 Jul-Aug;8 (4):539-47 **

Williams EK, Lohr JA, Guerrant RL. Acute infectious diarrhea. II. Diagnosis, treatment and prevention. Pediatr Infect Dis 1986 Jul-Aug;5(4):458-65

Therapy

Gertler S, Pressman J, Cartwright C, Dharmsathaphorn K. Management of acute diarrhea. J Clin Gastroenterol $1983\ Dec; 5(6):523-34$

Hamilton Jk. Treatment of acute diarrhea. Pediatr Clin North Am 1985 Apr;32 (2):419-27 **

Hirschhorn N. The treatment of acute diarrhea in children: an historical and physiological perspective. Am J Clin Nutr 1980 Mar;33(3):637-63 **

Hughes S. Acute secretory diarrhoeas; current concepts in pathogenesis and treatment. Drugs 1983;26:80-90 **

Kinoti SN, Wasunna A, Turkish J, Gateere R, Desai M, Agwanda R, Juma R. A comparison of the efficacy of maize-based ORS and standard W.H.O. ORS in the treatment of acute childhood diarrhea at Kenyatta National Hospital, Nairobi, Kenya: results of a pilot study. East Afr Med J 1986 Mar;63(3):168-74

Meeuwisse GW. High sugar worse than high sodium in oral rehydration solutions. Acta Paediatr Scand 1983 Mar;72(2):161-6 **

Williams EK, Lohr JA, Guerrant RL. Acute infectious diarrhea. II. Diagnosis, treatment and prevention. Pediatr Infect Dis 1986 Jul-Aug;5(4):458-65

DIARRHEA, CHRONIC

Arasu TS, Wyllie R, Fitzgerald JF. Chronic diarrhea in infants and children. Am Fam Physician 1979 Apr;19(4):87-94 **

Barness LA. Chronic diarrhea in children. Postgrad Med 1979 Feb;65(2):163-6, 168-9 **

Berrut C. Loizeau E. [Chronic diarrhea: current aspects]. Ther Umsch 1984 Sep;41(9):618-24

Blaser MJ. Infectious diarrheas: acute, chronic, and iatrogenic [editorial]. Ann Intern Med 1986 Nov;105(5):785-7

Booth IW, Candy DCA. Practical problems in protracted diarrhoea. J Trop Pediatr 1987 Apr;33(2):69-74 **

Cichowicz-Emmanuelli E. Chronic non-specific diarrhea of infancy. Bol Asoc Med PR 1982 May-Jun;74(5-6):178-81

Fitzgerald JF, Clark JH. Chronic diarrhea. Pediatr Clin North Am 1982 Feb;29 (1):221-31 **

Gall DG, Hamilton JR. Chronic diarrhea in childhood: a new look at an old problem. Pediatr Clin North Am 1974 Nov;21(4):1001-17

Leung AKC. Chronic nonspecific diarrhea of childhood (irritable colon syndrome). Contem Pediatr 1987 Mar-Apr:10-3

Diarrhoeal Diseases 47

Matseshe JW, Phillips SF. Chronic diarrhea: a practical approach. Med Clin North Am 1978 Jan;62(1):141-54 **

Persistent diarrhoea in children in developing countries: memorandum from a WHO meeting. Bull WHO 1988:66(6):709-17 **

Complications

Auricchio S, Cucchiara S, D'Antonio AM. De Ritis G, De Vizia B, Follo D, laccarino E. Gastrointestinal allergy or intolerance to multiple foods in severe chronic diarrhea in early infancy. In: Lebenthal E, ed. Chronic diarrhea in children. New York: Raven Press. 1984:425-34

Diagnosis

Giannella RA. Chronic diarrhea in travelers: diagnostic and therapeutic considerations. Rev Infect Dis 1986 May-Jun;8(suppl 2):S223-6 **

Mehta S. Investigative approach towards chronic diarrhoea in infants and children. Indian Pediatr 1977 Apr;14(4):303~8

Diet Therapy

Chernoff R, Dean JA. Medical and nutritional aspects of intractable diarrhea. J Am Diet Assoc 1980 Feb;76(2):161-9 **

Cohen S, Lake AM, Mathis RK, Walker WA. Perspectives on chronic nonspecific diarrhea: dietary management. Pediatrics 1978 May:61(5):808-9 **

Gardner FH. Nutritional management of chronic diarrhea in adults. JAMA 1962 Apr 14:180(2):147-52 **

Greene HL. A pathophysiologic approach to dietary management in patients with protracted diarrhea and malnutrition. <u>In:</u> Winter RW, Greene HL, eds. Nutritional support of the seriously ill patient. New York: Academic Press, 1983:181-94

Hamilton JR. Nutritional therapy of chronic diarrhea. <u>In:</u> Lebenthal E, ed. Chronic diarrhea in children. New York: kaven Press, 1984:535-40 **

Heim T. Requirements and utilization of macronutrients in enteral and parenteral nutrition in acute and chronic diarrhea. In: Lebenthal E, ed. Chronic diarrhea in children. New York: Raven Press, 1984:541-57 **

Lebenthal E. Rossi TM. Intractable diarrhea of infancy: an alternative treatment strategy. Postgrad Med 1983 Aug;74(2):153-9 **

Loeb H, Mozin MJ. Prevention of chronic diarrhea: nutritional implications. J Pediatr Gastroenterol Nutr 1983;2(suppl 1):S328-34

Sankaranarayanan YS, Santhanakrishnan BR. Chronic diarrhoea in infancy & childhood: rationalised approach and guidelines for dietary management. Pediatr Bull 1986 Jul:8(1):95-102

Drug Therapy

Gracey M. Antibiotic and antiparasitic therapy in chronic diarrhea. <u>In:</u> Lebenthal E, ed. Chronic diarrhea in children. New York: Raven Press, 1984:469-76

Netchvolodoff CV, Hargrove MD, Jr. Recent advances in the treatment of diarrhea. Arch Intern Med 1979 Jul;139(7):813-6 **

Etiology

Andres JM. Advances in understanding the pathogenesis of persistent diarrhea in young children. Adv Pediatr 1988;35:483-98

Lo CW, Walker WA. Chronic protracted diarrhea of infancy: a nutritional disease. Pediatrics $1983\ Dec;72(6):786-800\ **$

Mata L, Urrutia JJ, Simhon A. Infectious agents in acute and chronic diarrhea of childhood. In: Lebenthal E, ed. Chronic diarrhea in children. New York: Raven Press, 1984:237-52 **

Poley JR. Causes of chronic diarrhea in infants and children. Postgrad Med $1970 \, \mathrm{Dec}; 48(6): 143-7 \, **$

Fluid Therapy

Lebenthal E, Rossi TM. Intractable diarrhea of infancy: an alternative treatment strategy. Postgrad Med 1983 Aug;74(2):153-9 **

Metabolism

Auricchio S. Peptide digestion and absorption in the small intestinal mucosa during acute and chronic diarrhea. In: Lebenthal E, ed. Chronic diarrhea in children. New York: Raven Press. 1984:179-91

Gracey M. Chronic diarrhoea in protein-energy malnutrition. Paediatr Indones 1981 Nov-Dec;21(11-12):235-9 **

Gracey M. The intestinal microflora in malnutrition and protracted diarrhea in infancy. <u>In</u>: Lebenthal E, ed. Chronic diarrhea in children. New York: Raven Press, 1984:223-36

Greene HL. A pathophysiologic approach to dietary management in patients with protracted diarrhea and malnutrition. $\underline{\text{In}}$: Winter RW, Greene HL, eds. Nutritional support of the seriously ill patient. New York: Academic Press, 1983:181-94

Oral Therapy

Netchvolodoff CV, Hargrove MD, Jr. Recent advances in the treatment of diarrhea. Arch Intern Med 1979 Jul;139(7):813-6 **

Physiopathology

Andres JM. Advances in understanding the pathogenesis of persistent diarrhea in young children. Adv Pediatr 1988;35:483-98

Gracey M. Chronic diarrhoea in protein-energy malnutrition. Paediatr Indones 1981 Nov-Dec;21(11-12):235-9 **

Gracey M. The intestinal microflora in malnutrition and protracted diarrhea in infancy. In: Lebenthal E, ed. Chronic diarrhea in children. New York: Raven Press. 1984:223-36

Lo CW, Walker WA. Chronic protracted diarrhea of infancy: a nutritional disease. Pediatrics 1983 Dec;72(6):786-800 **

Protein and fat losses in infants with prolonged diarrhea. Nutr Rev 1982 Nov; 40(11):335-7

Rask-Madsen J, Bukhave K. Prostaglandins and chronic diarrhoea: clinical aspects. Scand J Gastroenterol 1979;14(suppl 53):73-8 **

Rossi TM. Intractable diarrhea of infancy. J Pediatr Gastroenterol Nutr 1983;2(suppl 1):S315-20

Rossi TM, Lebenthal E. Pathogenic mechanisms of protracted diarrhea. Adv Pediatr 1983;30:595-633

Prevention and Control

Loeb H, Mozin MJ. Prevention of chronic diarrhea: nutritional implications. J Pediatr Gastroenterol Nutr 1983;2(suppl 1):S328-34

Therapy

Chernoff R. Dean JA. Medical and nutritional aspects of intractable diarrhea. J Am Diet Assoc 1980 Feb;76(2):161-9 **

Fitzgerald JF. Management of the infant with persistent diarrhea. Pediatr Infect Dis 1985 Jan-Feb; 4(1):6-9

Giannella RA. Chronic diarrhea in travelers: diagnostic and therapeutic considerations. Rev Infect Dis 1986 May-Jun;8(suppl 2):S223-6 **

Heim T. Requirements and utilization of macronutrients in enteral and parenteral nutrition in acute and chronic diarrhea. <u>In</u>: Lebenthal E, ed. Chronic diarrhea in children. New York: Raven Press, 1984:541-57 **

Reisman T, Morton RE, Rogers AI. Gastroenterology: a hypothetical case of chronic diarrhea incorporating a management self-test. Postgrad Med 1976 Feb; 59(2):203-10 **

Rossi TM. Intractable diarrhea of infancy. J Pediatr Gastroenterol Nutr 1983;2(suppl 1):S315-20

Townley RRW. The management of chronic or recurrent diarrhoea in childhood. Postgrad Med J 1969 Feb:45:135-46 **

Veterinary

Burrows CF. Chronic diarrhea in the dog. Vet Clin North Am [Small Anim Pract] 1983 Aug;13(3):521-40 **

DIARRHEA, INFANTILE

Arasu TS, Wyllie R, Fitzgerald JF. Chronic diarrhea in infants and children. Am Fam Physician 1979 Apr;19(4):87-94 **

Auricchio S, Cucchiara S, D'Antonio AM, De Ritis G, De Vizia B, Follo D, Iaccarino E. Gastrointestinal allergy or intolerance to multiple foods in severe chronic diarrhea in early infancy. In: Lebenthal E, ed. Chronic diarrhea in children. New York: Raven Press, 1984:425-34

Barness LA. Chronic diarrhea in children. Postgrad Med 1979 Feb;65(2):163-6, 168-9 **

Bhattacharya SK. Acute diarrhoeal diseases in newborn. J Indian Med Assoc 1987 Jul;85(7):213, 209

Chakraborty B. Diarrhoeal diseases in children [editorial]. J Indian Med Assoc 1987 Jul:85(7):193-5

Cichowicz-Emmanuelli E. Chronic non-specific diarrhea of infancy. Bol Asoc Med PR 1982 May-Jun;74(5-6):178-81

Desai AG. Diarrhoeal disorders in childhood. Q Med Rev 1979 Jul;30(3): 1-33 **

Elzouki AY, Vesikari T. First international conference on infections in children in Arab countries. Pediatr Infect Dis 1985 Sep-Oct;4(5):527-31

Green EC. Traditional healers, mothers and childhood diarrheal disease in Swaziland: the interface of anthropology and health education. Soc Sci Med 1985;20(3):277-85 **

Kinoti SN, Wasunna A, Turkish J, Gateere R, Desai M, Agwanda R, Juma R. A comparison of the efficacy of maize-based ORS and standard W.H.O. ORS in the treatment of acute childhood diarrhea at Kenyatta National Hospital, Nairobi, Kenya: results of a pilot study. East Afr Med J 1986 Mar;63(3):168-74

Leung AKC. Chronic nonspecific diarrhea of childhood (irritable colon syndrome). Contemp Pediatr 1987 Mar-Apr:10-3

Lo CW, Walker WA. Chronic protracted diarrhea of infancy: a nutritional disease. Pediatrics 1983 Dec;72(6):786-800 **

Sen PC, Banerjee H. Diarrhoea - the great killer of infants and children. Your Health 1983 Jan;32(1):9-14 **

Diarrhoeal Diseases 51

Chemically Induced

Gracey M., Burke V. Sugar-induced diarrhoea in children. Arch Dis Child 1973 May:48(261):331-6 **

Complications

Meeuwisse GW. High sugar worse than high sodium in oral rehydration solutions. Acta Paediatr Scand 1983 Mar:72(2):161-6 **

Paneth N. Hypernatremic dehydration of infancy: an epidemiologic review. Am J Dis Child 1980 Aug:134(8):785-92 **

Rowland MGM, Rowland SGJG. Growth faltering in diarrhoea. <u>In:</u> Taylor TG, Jenkins NK, eds. Proceedings of the XIII International Congress of Nutrition, Brighton, 18-23 Aug 1985. London: Libby, 1986:115-9 **

Walsh JA. Selective primary health care: strategies for control of disease in the developing world. IV. Measles. Rev Infect Dis 1983 Mar-Apr;5(2):330-40 **

Diagnosis

Goldschmidt B. Microscopic stool-gazing, a guide to the cause and cure of chronic and recurrent diarrhoea in children. S Afr Med J 1966 Feb 26;40(9):191-5 **

Mehta S. Investigative approach towards chronic diarrhoea in infants and children. Indian Pediatr 1977 Apr;14(4):303-8

Diet Therapy

Behar M. The role of feeding and nutrition in the pathogeny and prevention of diarrheic processes. Bull Pan Am Health Organ 1975;9(1):1-9 **

Booth IW, Cutting WAM. Current concepts in the management of acute diarrhoea in children. Postgrad Doc - Middle-East 1984 Jul:418-24 **

Hamilton JR. Nutritional therapy of chronic diarrhea. <u>In:</u> Lebenthal E, ed. Chronic diarrhea in children. New York: Raven Press. 1984:535-40 **

Jelliffe EFP, Jelliffe DB, Feldon K, Ngokwey N. Traditional practices concerning feeding during and after diarrhoea (with special reference to acute dehydrating diarrhoea in young children). World Rev Nutr Diet 1987;53:218-95

Lebenthal E, Rossi TM. Intractable diarrhea of infancy; an alternative treatment strategy. Postgrad Med 1983 Aug;74(2):153-9 **

Sankaranarayanan YS, Santhanakrishnan BR. Chronic diarrhoea in infancy & childhood: rationalised approach and guidelines for dietary management.

Pediatr Bull 1986 Jul:8(1):95-102

Drug Therapy

Booth IW, Cutting WAM. Current concepts in the management of acute diarrhoea in children. Postgrad Doc - Middle-East 1984 Jul:418-24 **

DuPont HL. Nonfluid therapy and selected chemoprophylaxis of acute diarrhea. Am J Med 1985 Jun 28;78(suppl 6B):81-90 **

Rabbani GH. Drug treatment of infectious diarrhea in children. World Pediatr Child Care 1986;2(3):243-54 **

Etiology

Andres JM. Advances in understanding the pathogenesis of persistent diarrhea in young children. Adv Pediatr 1988;35:483-98

Does malnutrition predispose children to diarrhoea? Nutr Rev 1985 May;43(5): 144-5

Reddy V. Interaction between malnutrition and diarrhoea with particular reference to pediatric practice. Indian J Gastroenterol 1985 Jul;4(3):183-6 **

Rohde J, Northrup RS. Diarrhoea: a nutritional disease. J Indian Med Assoc 1987 Jul:85(7):196-202

Sunoto. Diarrhoeal problems in Southeast Asia. Southeast Asian J Trop Med Public Health 1982 Sep;13(3):306-18 **

Tontisirin K, Valyasevi A. Protein energy malnutrition related to diarrhea in Thai children. J Nutr Sci Vitaminol (Tokyo) 1981;27(6):513-30 **

Fluid Therapy

Booth IW, Cutting WAM. Current concepts in the management of acute diarrhoea in children. Postgrad Doc - Middle-East 1984 Jul:418-24 **

Lebenthal E, Rossi TM. Intractable diarrhea of infancy: an alternative treatment strategy. Postgrad Med 1983 Aug;74(2):153-9 **

Metabolism

Gracey M. The intestinal microflora in malnutrition and protracted diarrhea in infancy. In: Lebenthal E, ed. Chronic diarrhea in children. New York: Raven Press, 1984:223-36

Molla A, Molla AM, Sarker SA. Malabsorption in enteric infection; a nutritional cost in children with diarrhea. <u>In:</u> Chagas C, Keusch GT, eds. The interaction of parasitic diseases and nutrition, 22-26 Oct 1985. Vaticana: Pontificia Academia Scientiarum, 1985:71-80 **

Molla AM, Molla A, Sarker SA, Rahaman MM. Food intake during and after recovery from diarrhoea in children. <u>In</u>: Chen LC, Scrimshaw NS, eds. Diarrhea and malnutrition: interactions, mechanisms, and interventions. New York: Plenum, 1983:113-23 **

Microbiology

Buchino JJ, Suchy FJ, Snyder JW. Bacterial diarrhea in infants and children. Perspect Pediatr Pathol 1984 Summer;8(2):163-80 **

Gracey M. The intestinal microflora in malnutrition and protracted diarrhea in infancy. In: Lebenthal E, ed. Chronic diarrhea in children. New York: Raven Press, 1984:223-36

Thoren A. The role of enteropathogenic \underline{E} , $\underline{\operatorname{coli}}$ in infantile diarrhoea: aspects on bacteriology, epidemiology and therapy. Scand J Infect Dis 1983; (suppl 37):1-51

Mortality

Ashworth A. International differences in child mortality and the impact of malnutrition. Hum Nutr Clin Nutr 1982;36C(4):279-88 **

Ashworth A. International differences in infant mortality and the impact of malnutrition: a review. Hum Nutr Clin Nutr 1982;36C(1):7-23 **

Sunoto. Diarrhoeal problems in Southeast Asia. Southeast Asian J Trop Med Public Health 1982 Sep:13(3):306-18 **

Occurrence

ķ

Chen LC, Huq E, Huffman SL. A prospective study of the risk of diarrhoeal diseases according to the nutritional status of children. Am J Epidemiol 1981 Aug;114(2):284-92 **

Levine MM, Edelman R. Enteropathogenic <u>Escherichia coli</u> of classic serotypes associated with infant diarrhea: epidemiology and pathogenesis. Epidemiol Rev 1984;6:31-51 **

Pickering LK, Bartlett AV, Woodward WE. Acute infectious diarrhea among children in day care: epidemiology and control. Rev Infect Dis 1986 Jul-Aug;8 (4):539-47 **

Pickering LK. Infections in day care. Pediatr Infect Dis 1987 Jun;6(6): 614-7 **

Rohde J, Northrup RS. Diarrhoea: a nutritional disease. J Indian Med Assoc 1987 Jul;85(7):196-202

Sunoto, Diarrhoeal problems in Southeast Asia. Southeast Asian J Trop Med Public Health 1982 Sep;13(3):306-18 **

Thoren A. The role of enteropathogenic \underline{E} , \underline{coli} in infantile diarrhoea: aspects

1

on bacteriology, epidemiology and therapy. Scand J Infect Dis 1983; (suppl 37):1-51

Oral Therapy

Booth IW, Levine MM, Harries JT. Oral rehydration therapy in acute diarrhoea in childhood. J Pediatr Gastroenterol Nutr 1984 Sep;3(4):491-9 **

Hirschhorn N. The treatment of acute diarrhea in children: an historical and physiological perspective. Am J Clin Nutr 1980 Mar; 33(3):637-63 **

Pathology

Giorgi PL, Catassi C, Coppa GV, Valentini V, Sbarbati A. [New protagonists of infectious diarrhea in childhood. Pathogenetic and clinical aspects]. Minerva Pediatr 1985 Jan 31;37(1-2):29-48

Goldschmidt B. Microscopic stool-gazing, a guide to the cause and cure of chronic and recurrent diarrhoea in children. S Afr Med J 1966 Feb 26;40(9): 191-5 **

Physiopathology

Andres JM. Advances in understanding the pathogenesis of persistent diarrhea in young children. Adv Pediatr 1988;35:483-98

Behar M. The role of feeding and nutrition in the pathogeny and prevention of diarrheic processes. Bull Pan Am Health Organ 1975;9(1):1-9 **

Darrow DC, Pratt EL, Flett J, Jr., Gamble AH, Wiess HF. Disturbances of water and electrolytes in infantile diarrhea. Pediatrics 1949 Feb;3(2):129-56 **

Desai AG. Diarrhoeal disorders in childhood. Q Med Rev 1979 Jul;30(3): 1-33 **

Does malnutrition predispose children to diarrhoea? Nutr Rev 1985 May;43(5): 144-5

Giorgi PL, Catassi C, Coppa GV, Valentini V, Sbarbati A. [New protagonists of infectious diarrhea in childhood. Pathogenetic and clinical aspects]. Minerva Pediatr 1985 Jan 31;37(1-2):29-48

Levine MM, Edelman R. Enteropathogenic <u>Escherichia coli</u> of classic serotypes associated with infant diarrhea: epidemiology and pathogenesis. Epidemiol Rev 1984;6:31-51 **

Molla A, Molla AM. Sarker SA. Malabsorption in enteric infection; a nutritional cost in children with diarrhea. <u>In</u>: Chagas C, Keusch GT, eds. The interaction of parasitic diseases and nutrition, 22-26 Oct 1985. Yaticana: Pontificia Academia Scientiarum. 1985;71-80 **

Robins-Browne RM. Traditional enteropathogenic Escherichia coli of infantile

diarrhea. Rev Infect Dis 1987 Jan-Feb;9(1):28-53 **

Prevention and Control

Ashworth A, Feachem RG. Interventions for the control of diarrhoeal diseases among young children; weaning education. Bull WHO 1985;63(6):1115-27 **

Behar M. The role of feeding and nutrition in the pathogeny and prevention of diarrheic processes. Bull Pan Am Health Organ 1975;9(1):1-9 **

Feachem RG, Koblinsky MA. Interventions for the control of diarrhoeal diseases among young children: measles immunization. Bull WHO 1983;61(4): 641-52 **

Feachem RG, Koblinsky MA. Interventions for the control of diarrhoeal diseases among young children: promotion of breast-feeding. Bull WHO 1984;62(2): 271-91 **

Feachem RG. Interventions for the control of diarrhoeal diseases among young children: promotion of personal and domestic hygiene. Bull WHO 1984;62(3): 467-76 **

Levine MM, Losonsky G, Herrington D, Kaper JB, Tacket C, Rennels MB, Morris JG. Pediatric diarrhea: the challenge of prevention. Pediatr Infect Dis 1986 Jan;5(suppl 1):S29-43 **

Pickering LK, Bartlett AV, Woodward WE. Acute infectious diarrhea among children in day care: epidemiology and control. Rev Infect Dis 1986 Jul-Aug;8 (4):539-47 **

Research on improving infant feeding practices to prevent diarrhoea or reduce its severity: Memorandum from a JHU/WHO meeting. Bull WHO 1989;67(1):27-33 **

Therapy

Fitzgerald JF. Management of the infant with persistent diarrhea. Pediatr Infect Dis 1985 Jan-Feb;4(1):6-9

Thoren A. The role of enteropathogenic \underline{E} , \underline{coli} in infantile diarrhoea: aspects on bacteriology, epidemiology and therapy. Scand J Infect Dis 1983; (suppl 37):1-51

DIENTAMOEBA FRAGILIS

Turner JA. Giardiasis and infections with <u>Dientamoeba fragilis</u>. Pediatr Clin North Am 1985 Aug; 32(4):865-80 **

DIETS AND DIET THERAPY

Banwell JG. Treatment of travelers' diarrhea: fluid and dietary management.

I

Rev Infect Dis 1986 May-Jun;8(suppl 2):S182-7 **

Chernoff R, Dean JA. Medical and nutritional aspects of intractable diarrhea. J Am Diet Assoc 1980 Feb;76(2):161-9 **

Cohen S, Lake AM, Mathis RK, Walker WA. Perspectives on chronic nonspecific diarrhea: dietary management. Pediatrics 1978 May;61(5):808-9 **

Diamond LS. Amebiasis: nutritional implications. Rev Infect Dis 1982 Jul-Aug;4(4):843-50 **

Goriup U, Shmerling DH. [Dietary therapy of intestinal diseases in childhood]. Ther Umsch 1978;35(8):673-8 **

Greene HL. A pathophysiologic approach to dietary management in patients with protracted diarrhea and malnutrition. \underline{In} : Winter RW, Greene HL, eds. Nutritional support of the seriously ill patient. New York: Academic Press, 1983:181-94

Kelts D, Jones E. Selected topics in therapeutic nutrition. Curr Probl Pediatr 1983 Mar;13(5):1-62

Loeb H, Mozin MJ. Prevention of chronic diarrhea: nutritional implications. J Pediatr Gastroenterol Nutr 1983;2(suppl 1):S328-34

Morris JA, Jr., Selivanov V, Sheldon GF. Nutritional management of patients with malabsorption syndrome. Clin Gastroenterol 1983 May;12(2):463-74

Person PG, Ahlbom A, Hellers G. Crohn's disease and ulcerative colitis. A review of dietary studies with emphasis on methodologic aspects. Scand J Gastroenterol 1987 May:22(4):385-9

Russell RI. Intestinal adaptation to an elemental diet. Proc Nutr Soc 1985 Feb;44(1):87-93

Sankaranarayanan YS, Santhanakrishnan BR. Chronic diarrhoea in infancy & childhood: rationalised approach and guidelines for dietary management. Pediatr Bull 1986 Jul;8(1):95-102

Thorne GM. Gastrointestinal infections--dietary interactions. J Am Coll Nutr 1986;5(5):487-99 **

Walker-Smith JA. Nutritional management of acute gastroenteritis -- rehydration and realimentation. Hum Nutr Appl Nutr 1986;4DA(suppl 1):39-43

DIGESTION

Auricchio S. Peptide digestion and absorption in the small intestinal mucosa during acute and chronic diarrhea. In: Lebenthal E, ed. Chronic diarrhea in children. New York: Raven Press, $19\overline{84}$:179-91

Freeman HJ, Sleisenger MH, Kim YS. Human protein digestion and absorption: normal mechanisms and protein-energy malnutrition. Clin Gastroenterol 1983 May;12(2):357-78

Nicholl CG, Polak JM, Bloom SR. The hormonal regulation of food intake, digestion, and absorption. Annu Rev Nutr 1985;5:213-39

DIGESTIVE SYSTEM DISEASES

Joiner KA, Gorbach SL. Antimicrobial therapy of digestive diseases. Clin Gastroenterol 1979 Jan;8(1):3-35

DISACCHARIDASES

da Rocha JM. [Disaccharidase disorders]. Bol Inst Puericult 1963 Apr;20(1): 311-22 **

DISEASE.

Feachem RG, Bradley DJ, Garelick H, Mara DD. Sanitation and disease: health aspects of excreta and wastewater management. New York: Wiley, 1983. 501 p. [World Bank Studies in water supply and sanitation, 3]

Jenkins DJA, Jenkins AL, Wolever TMS, Rao AV, Thompson LU. Fiber and starchy foods: gut function and implications in disease. Am J Gastroenterol 1986 Oct;81(10):920-30 **

Walsh JA, Warren KS. Selective primary health care: an interim strategy for disease control in developing countries. Soc Sci Med 1980;14C(2):145-63 **

DISEASE MODELS, ANIMAL

Candy DCA, Leung TSM, Phillips AD, Harries JT, Marshall WC. Models for studying the adhesion of enterobacteria to the mucosa of the human intestinal tract. In: Elliott K, O'Connor M, Whelen J, eds. Adhesion and microorganisms pathogenicity. London: Pitman, 1981:72-93. (Ciba Foundation symposium, 80)

Cornelis G. Laroche Y, Balligand G, Sory M-P, Wauters G. Yersinia enterocolitica, a primary model for bacterial invasiveness. Rev Infect Dis $1987 \ \text{Jan-Feb;} 9(1):64-87 \ **$

Freter R, Jones GW. Models for studying the role of bacterial attachment in virulence and pathogenesis. Rev Infect Dis 1983 Sep-Oct;5(suppl 4):S647-58 **

Morrison DC. Bacterial endotoxins and pathogenesis. Rev Infect Dis 1983 Sep-Oct:5(suppl 4):S733-47 **

Strober W. Animal models for inflammatory bowel disease--an overview. Dig Dis Sci 1985 Dec;30(suppl 12):S3-10

Topalian SL, Ziegler MM. Necrotizing enterocolitis: a review of animal models.

J Surg Res 1984 Oct; 37(4): 320-36

DISEASE OUTBREAKS AND TRANSMISSION

Akin EW, Jakubowski W. Drinking water transmission of giardiasis in the United States. Wat Sci Technol 1986;18(10):219-26 **

Black RH. Invited discussion of Dr R M Glasse's paper. <u>In: Proceedings of the Cholera Research Symposium</u>, Honolulu, 24-29 Jan 1965. Washington, D.C.: U S Government Printing Office, 1965:340 **

Briscoe J. Intervention studies and the definition of dominant transmission routes. Am J Epidemiol 1984 Sep;120(3):449-55 **

Briscoe J. Public health in rural India: the case of excreta disposal. Cambridge: Center for Population Studies, Harvard University, 1976. xix, 414 p. PhD Thesis. (Research paper, 12)

The Child Day Care Infectious Disease Study Group. Considerations of infectious diseases in day care centers. Pediatr Infect Dis 1985 Mar-Apr;4 (2):124-36

Craun GF. A summary of waterborne illness transmitted through contaminated groundwater. J Environ Health 1985 Nov-Dec;48(3):122-7 **

Feachem RG. Environmental aspects of cholera epidemiology, I. A review of selected reports of endemic and epidemic situations during 1961-1980. Trop Dis 8ull 1981 Aug; 78(8):675-98 **

Feachem RG. Environmental aspects of cholera epidemiology. III. Transmission and control. Trop Dis Bull 1982 Jan;79(1):1-47 **

Kawata K. Water and other environmental interventions--the minimum investment concept. Am J Clin Nutr 1978 Nov;31(11):2114-23 **

Khan MU. Interruption of transmission of diarrhoeal agents. <u>In</u>: Programme, papers and abstracts of the Third Asian Conference on Diarrhoeal Diseases, Bangkok, 10-14 Jun 1985:173-80

Morris RJ. Religion and medicine: the cholera pamphlets of 0x ford, 1832, 1849 and 1854. Med Hist 1975 Jul;19(3):256-70 **

Mosley WH, Khan MU. Cholera epidemiology - some environmental aspects. Prog Water Technol 1979;11(1-2):309-16 **

Pitlik S, Berger SA, Huminer D. Nonenteric infections acquired through contact with water. Rev Infect Dis 1987 Jan-Feb;9(1):54-63 **

Ramia S. Transmission of viral infections by the water route: implications for developing countries. Rev Infect Dis 1985 Mar-Apr;7(2):180-8 **

Shuval HI, Yekutiel P, Fattal B. An epidemiological model for the potential health risk associated with various pathogens in wastewater irrigation. Wat

Sci Technol 1986;18(10):191-8 **

DNA, BACTERIAL

Echeverria P, Seriwatana J, Sethabutr O, Taylor DN. DNA hybridization in the diagnosis of bacterial diarrhea. Clim Lab Med 1985 Sep;5(3):447-62 **

Ī

Sternbery N. Evidence that adenine methylation influences DNA-protein interactions in Escherichia coli. J Bacteriol 1985 Oct;164(1):490-3

DOXYCYCLINE

Sack RB. Antimicrobial prophylaxis of travellers' diarrhoea: a summary of studies using doxycycline or trimethoprim and sulphamethoxazole. Scand J Gastroenterol 1983;18(suppl 84):111-7 **

DRUG RESISTANCE, MICROBIAL

Chizhov NP. [Mechanising of the formation of viral resistance to chemotherapeutic preparations]. Vopr Virusol 1985 May-Jun;30(3):266-79

Col NF, O'Connor RW. Estimating worldwide current antibiotic usage: report of Task Force 1. Rev Infect Dis 1987 May-Jun;9(suppl 3):S232-43

Huq I, Aziz KMS. Changing antibiotic sensitivity pattern of the commonly occurring organism causing dysentery. Bangladesh Med J 1977 Apr;5(4): 119-25 **

Huq MI, Ahmed QS, Rahaman MM. Changing pattern of antibiotic resistance in Shigella isolated in Bangladesh. In: Rahaman MM. Greenough WB, III, Novak NR, Rahman S, eds. Shigellosis: a continuing global problem; proceedings of an international conference, Cox's Bazaar, 1981. Dhaka: International Centre for Diarrhoeal Disease Research, Bangladesh, 1983:174-81

Murray BE. Resistance of Shigella, Salmonella, and other selected enteric pathogens to antimicrobial agents. Rev Infect Dis 1986 May-Jun;8(suppl 2):S172-81 **

O'Brien TF. Resistance of bacteria to antibacterial agents: report of Task Force 2. Rev Infect Dis 1987 May-Jun;9(suppl 3):S244-60

Smith SM, Palumbo PE, Edelson PJ. <u>Salmonella</u> strains resistant to multiple antibiotics: therapeutic implications. <u>Pediatr Infect Dis 1984</u> Sep-Oct;3(5): 455-60

DRUG - EVALUATION, INTERACTIONS AND THERAPEUTIC USE

Awouters F, Niemegeers CJE, Janssen PAJ. Pharmacology'of antidiarrheal drugs.

Annu Rev Pharmacol Toxicol 1983;23:279-301 **

Bartlett JG. Antibiotic-associated colitis. DM 1984 Dec;30(15):1-54

Burdon DW. Treatment of pseudomembranous colitis and antibiotic-associated diarrhoea. J Antimicrob Chemother 1984 Dec;14(suppl D):103-9 **

Cline BL. Current drug regimens for the treatment of intestinal helminth infections. Med Clin North Am 1982 May:66 (3):721-42

Col NF, O'Connor RW. Estimating worldwide current antibiotic usage: report of Task Force 1. Rev Infect Dis 1987 May-Jun;9(suppl 3):S232-43

Danhof IE. Pharmacology, toxicology, clinical efficacy, and adverse effects of calcium polycarbophil, and enteral hydrosorptive agent. Pharmacotherapy 1982 Jan-Feb;2(1):18-28 **

de Zoysa I, Feachem RG. Interventions for the control of diarrhoeal diseases among young children: chemoprophylaxis. Bull WHO 1985;63(2):295-315

Dondwitz M, Wicks J, Cusolito S, Sharp GW. Pharmacotherapy of diarrheal diseases: an approach based on physiologic principles. KROC Found Ser 1984;17:329-59

DuPont HL. Nonfluid therapy and selected chemoprophylaxis of acute 'diarrhea. Am J Med 1985 Jun 28;78(68):81-90 **

Fortson WC, Tedesco FJ. Drug-induced colitis: a review. Am J Gastroenterol $1984\ Nov;79(11):878-83\ **$

Gracey M. Antibiotic and antiparasitic therapy in chronic diarrhea. In: Lebenthal E, ed. Chronic diarrhea in children. New York: Rayen Press, 1984:469-76

Greenough WB, Rabbani GH. Antisecretory and antimicrobial drugs for treating diarrhoea. In: Holmgren J, Lindberg A, Mollby R, eds. Development of vaccines and drugs against diarrhea; proceedings of the 11th Nobel Conference, Stockholm, 1985. Lund: Studentilitteratur, 1986:270-7 **

Gross MH. Management of antibiotic-associated pseudomembranous colitis. Clin Pharm 1985 May-Jun;4(3):304-10

Johnson PC, DuPont HL, Ericsson CD. Chemoprophylaxis and chemotherapy of travelers' diarrhea in children. Pediatr Infect Dis 1985 Nov-Dec;4(6):620-1

Joiner KA, Gorbach SL. Antimicrobial therapy of digestive diseases. Clin Gastroenterol 1979 Jan; 8(1):3-35

Lambert HP. Antimicrobial agents in diarrhoeal disease. Clin Gastroenterol 1979 Sep;8(3):827-33 **

Lennard-Jones JE. Medical treatment of ulcerative colitis. Postgrad Med J $1984\ Nov;60(709):797-802$

Levine MM. Antimicrobial therapy for infectious diarrhea. Rev Infect Dis 1986

May-Jun;8(suppl 2):S207-16 **

Lolekha S. Consequences of treatment of gastrointestinal infections. Scand J Infect Dis 1986; (suppl 49):154-9

McHenry MC, Weinstein AJ. Antimicrobial drugs and infections in ambulatory patients. Some problems and perspectives. Med Clin North Am 1983 Jan;67(1): 3-16

Nelson JD, Kusmiesz H, Shelton S. Oral or intravenous trimethoprimsulfamethoxazole therapy for shigellosis. Rev Infect Dis 1982 Mar-Apr;4(2): 546-50 **

Norrby SR. Problems in evaluation of adverse reactions to β -lactam antibiotics. Rev Infect D1s 1986 Jul-Aug:8(suppl 3):S358-70 **

O'Connor TW. Pseudomembranous enterocolitis: a historical and clinical review. Dis Colon Rectum 1981 Sep;24(6):445-8 **

Prichard RK. Interaction of host physiology and efficacy of antiparasitic drugs. Vet Parasitol 1985 Aug;18(2):103-10

Ruiz-Palacious GM. Norfloxacin in the treatment of bacterial enteric infections. Scand J Infect Dis 1986; (suppl 48):55-63 **

Sack RB. Antimicrobial prophylaxis of travellers' diarrhoea: a summary of studies using doxycycline or trimethoprim and sulphamethoxazole. Scand J Gastroenterol 1983;18(suppl 84):111+7 **

Silva J. Jr., Fekety R. <u>Clostridia</u> and antimicrobial enterocolitis. Annu Rev Med 1981;32:327-33

Steffen R, Heusser R, DuPont HL. Prevention of travelers' diarrhea by nonantibiotic drugs. Rev Infect Dis 1986 May-Jun;8(suppl 2):SI51-9 **

Tanowitz HB, Weiss LM, Wittner M. Diagnosis and treatment of protozoan diarrheas. Am J Gastroenterol 1988 Apr;83(4):339-50

Turner AC. Travellers' diarrhoea: prevention by chemoprophylaxis. Scand J Gastroenterol 1983;18(suppl 84):107-10 **

Wolfe MS. The treatment of intestinal protozoan infections. Med Clin North Am 1982 May;66(3):707-20

Wormann B, Hochter W, Othenjiann R. [Drug-induced colitis]. Dtsch Med Wochenschr 1985 Sep 27;110(39):1504-9

DYSENTERY, AMEBIC

Gitler C, Calef E, Rosenberg I. Cytopathogenicity of Entamoeba histolytica. Trans R Soc Lond [Biol] 1984 Nov 13;30(1131):73-85

Martinez-Palomo A. The pathogenesis of amoebiasis. Parasitol Today 1987 Apr; 3(4):111-8

Martinez-Palomo A, Martinez-Baez M. Selective primary health care: strategies for control of disease in the developing world. X. Amebiasis. Rev Infect Dis 1983 Nov-Dec;5(6):1093-1102 **

Mirelman D. Ameba-bacterium relationship in amebiasis. Microbiol Rev 1987 Jun;51(2):272-84 **

Patterson M, Schoppe LE. The presentation of amoebiasis. Med Clin North - Am $1982\ \text{May}; 66(3):689-705$ **

Raydin JI, Guerrant RL. A review of the parasite cellular mechanisms involved in the pathogenesis of amebiasis. Rev Infect Dis 1982 Nov-Dec;4(6): 1185-1207 **

Sepulveda 8. Amebiasis: host-pathoyen biology. Rev Infect Dis 1982 Nov-Dec;4 (6):1247-53 **

Sepulveda B. Progress in amebiasis. Scand J Gastroenterol 1982;17(suppl 77): 153-64

Wright SG. Parasites and travellers' diarrhoea. Scand J Gastroenterol 1983; 18(suppl 84):25-9 **

DYSENTERY, BACILLARY

Anmed ZU, Sack DA, Sarker MR, Haider K. Possible approaches to the development of a vaccine against snigellosis. <u>In</u>: Ahmed ZU, Choudhury N, eds. Proceedings of the International Seminar on Biotechnology and Genetic Engineering, Dhaka, 25-27 Jan 1986. Dhaka: Banyladesh Academy of Sciences, 1987:195-204 **

Cantey JR. Shiya toxin--an expanding role in the pathogenesis of infectious diseases. J Infect Dis 1985 May;151(5):766-71 **

Catterall RD. Clinical aspects of keiter's disease. Br J Rheumatol 1983 Nov:22(4 suppl 2):151-5 **

Development of vaccines against shiyellosis: Memorandum from a WHO meeting. Bull WHO 1987:65(1):17-25 **

Gilman RH. Bacillary dysentery. Compr Ther 1984 Oct;10(10):14-9

Huq I, Aziz KMS. Changing antibiotic sensitivity pattern of the commonly occurring organism causing dysentery. Bangladesh Med J 1977 Apr;5(4): 119-25 **

Huq MI. Ahmed QS, Rahaman MM. Changing pattern of antibiotic resistance in Shigella isolated in Bangladesh. In: Rahaman MM, Greenough WB, III, Novak NR, Rahman S, eds. Shigellosis: a continuing global problem; proceedings of an international conference, Cox's Bazaar, 1981. Dhaka: International Centre for Diarrhoeal Disease Research, Bangladesh, 1983:174-81 **

Keusch GT. Shigella infections. Clin Gastroenterol 1979 Sep;8(3):645-62

Keusch GT, Donohue-Rolfe A, Jacewicz M. <u>Shigella</u> toxin and the pathogenesis of shigellosis. Ciba Found Symp 1985;112:193-214 **

Keusch GT. Donohue-Rolfe A, Jacewicz M. <u>Shigella</u> toxin(s); description and role in diarrhea and dysentery. Pharmacol Ther 1982;15(3):403-38 **

Kopecko DJ, Baron LS, Buysse J. Genetic determinants of virulence in <u>Shigella</u> and dysenteric strains of <u>Escherichia coli</u>: their involvement in the pathogenesis of dysentery. Curr Top Microbiol Immunol 1985;118:71-95

Levine MM. Bacillary dysentery: mechanisms and treatment. Med Clin North Am 1982 May:66(3):623-38 **

Levine MM, Kaper JB, Black RE, Clements ML. New knowledge on pathogenesis of bacterial enteric infections as applied to vaccine development. Microbiol Rev $1983\ Dec; 47(4):510-50$ **

Nelson JD, Kusmiesz H, Shelton S. Oral or intravenous trimethoprimsulfamethoxazole therapy for shigellosis. Rev Infect Dis 1982 Mar-Apr;4(2): 546-50 **

O'Brien AD, Holmes RK. Shiga and Shiga-like toxins. Microbiol Rev 1987 Jun;51(2):206-20 **

Rahaman MM. Shigellosis: an old disease with new faces [editorial]. J Diarrhoeal Dis Res 1984 Dec;2(4):208

Sansonetti PJ, d'Hauteville H, Formal SB, Toucas M. Plasmid-mediated invasiveness of <Shigella-like> Escherichia coli. Ann Microbiol (Paris) 1982 May-Jun:133(3):351-5 **

Zhalko-Titarenko VP, Bondarenko VM, Grigoryev AV, Kupchinsky LG, Rybalko SL. Dynamics of the interaction of <u>Shigella</u> with the epithelium in the process of infection. Zh Mikrobiol Epidemiol Immunobiol 1986 Apr; (4):21-4 **

EICOSANOIC ACIDS

Rask-Madsen J. Eicosanoids and their role in the pathogenesis of diarrhoeal diseases. Clin Gastroenterol 1986 Jul;15(3):545-66 **

Rask-Madsen J. The role of eicosanoids in the gastrointestinal tract. Scand J Gastroenterol 1987;22(suppl 127):7-19 **

ELECTROLYTES

Chang EB, Field M. Intestinal electrolyte transport and diarrheal disease. In: Kern F, Jr., Blum AL, eds. The gastroenterology annual, 1/1983. Amsterdam: Elsevier, 1983:148-80

Dobbins JW, Binder HJ. Pathophysiology of diarrhoea: alterations in fluid and electrolyte transport. Clin Gastroenterol 1981 Sep;10(3):605-25 **

Fondacaro JD. Intestinal ion transport and diarrheal disease. Am J Physiol 1986 Jan; $250(1, pt\ 1)$: 61-8

Milla PJ. Disorders of electrolyte absorption. Clin Gastroenterol 1982 Jan;11(1):31-46

ENDOTOXINS

Morrison DC. Bacterial endotoxins and pathogenesis. Rev Infect Dis 1983 Sep-Oct;5(suppl 4):S733-47 **

ENTAMOEBA HISTOLYTICA

Gitler C, Calef E, Rosenberg I. Cytopathogenicity of Entamoeba histolytica. Trans R Soc Lond [Biol] 1984 Nov 13;30(1131):73-85

Martinez-Palomo A. The pathogenesis of amoebiasis. Parasitol Today 1987 Apr; 3(4):111-8

Mirelman D. Ameba-bacterium relationship in amebiasis. Microbiol Rev 1987 Jun;51(2):272-84 **

Ravdin JI, Guerrant RL. A review of the parasite cellular mechanisms involved in the pathogenesis of amebiasis. Rev Infect Dis 1982 Nov-Dec;4(6): 1185-1207 **

Sepulveda B. Amebiasis: host-pathogen biology. Rev Infect Dis 1982 Nov-Dec;4 (6):1247-53 **

Sepulveda B. Progress in amebiasis. Scand J Gastroenterol 1982;17(suppl 77): 153-64′

Triss) D. Immunology of Entamoeba histolytica in human and animal hosts. Rev Infect Dis 1982 Nov-Dec; 4(6):1154-84

ENTERAL FEEDING

Heim T. Requirements and utilization of macronutrients in enteral and parenteral nutrition in acute and chronic diarrhea. In: Lebenthal E, ed. Chronic diarrhea in children. New York: Raven Press, 1984:541-57**

Love AHG. Metabolic response to malnutrition: its relevance to enteral feeding. Gut 1986 Nov;27(suppl 1):9-13 **

ENTERITIS

Alexandrescu M, Coman G, Ene L, Manuca M. [Human infection with Campylobacter

jejuni/coli]. Rev Ig [Bacteriol] 1984 Jul-Sep;29(3):245-57

Blackwell TE. Enteritis and diarrhea. Vet Clin North Am [Large Anim Pract] 1983 Nov:5(3):557-70 **

Blaser MJ, Reller LB. <u>Campylobacter</u> enteritis. N Engl J Med 1981 Dec 10:305(24):1444-52 **

Blaser MJ, Taylor DN, Feldman RA. Epidemiology of <u>Campylobacter</u> <u>jejuni</u> infections. Epidemiol Rev 1983;5:157-76 **

Butzler JP, Skirrow MB. <u>Campylobacter</u> enteritis. Clin Gastroenterol 1979 Sep;8(3):737-65 **

Chowdhury MNH. <u>Campylobacter</u> <u>jejuni</u> enteritis: a review. Trop Geogr Med 1984 Sep;36(3):215-22 **

Chretien JH, Garagusi VF. Current management of fungal enteritis. Med Clin North Am 1982 May:66(3):675-87 **

Fox JG. Campylobacteriosis - a "new" disease in laboratory animals. Lab Anim Sci 1982 Dec:32(6):625-37 **

Mabilangan LM, Tiangco-Torres N. Focus on Salmonella enteritis. Asean J Clin Sci 1983 Dec;4(4):392-4 **

Pal SC, Nair GB. Epidemiology of campylobacteriosis in developing countries. ICMR Bull 1984 Jul;14(7):1-4 **

Pollock RV, Carmichael L. Canine viral enteritis. Recent developments. Mod Vet Pract 1979 May;60(5):375-80

Sakai S, Ito T. <u>[Campylobacter</u> infections]. Nippon Saikingaku Zasshi 1985 May;40(3):563-80

Shane SM, Montrose MS. The occurrence and significance of <u>Campylobacter</u> <u>jejuni</u> in man and animals. Vet Res Commun 1985 Jul;9(3):167-98 **

Vantrappen G, Geboes K, Ponette E. <u>Yersinia</u> enteritis. Med Clin North Am 1982 May;66(3):639-53 **

Walker RI, Caldwell MB, Lee EC, Guerry P, Trust TJ, Ruiz-Palacios GM. Pathophysiology of <u>Campylobacter</u> enteritis. Microbiol Rev 1986 Mar;50(1): 81-94 **

Weber A. [Occurrence of Campylobacter jejuni in animals and its significance for the human]. Tierarztl Prax 1985;13(2):151-7

ENTEROBACTERIACEAE

Candy DCA, Leung TSM, Phillips AD, Harries JT, Marshall WC. Models for studying the adhesion of enterobacteria to the mucosa of the human intestinal tract. In: Elliott K, O'Connor M, Whelen J, eds. Adhesion and microorganisms

pathogenicity. London: Pitman, 1981:72-93. (Ciba Foundation symposium, 80)

ENTEROTOXINS

Carpenter CCJ. The pathophysiology of secretory diarrheas. Med Clin North Am 1982 May;66 (3):597-610 **

Finkelstein RA, Dorner F. Cholera enterotoxin (choleragen). Pharmacol Ther 1985:27(1):37-47

Gyr K. Toxin receptors and their pathogenetic significance. Acta Histochem 1984;(suppl 29):S95-102 **

Ljungh A, Eneroth P, Wadstrom T. Cytotonic enterotoxin from $\frac{\text{Aeromonas}}{\text{hydrophila}}$. Toxicon 1982;20(4):787-94 **

Neter E. Enteropathogenicity: recent developments. Klin Wochenschr 1982 Jul 15;60(14):699-701 **

ENTEROVIRUSES

Hyypia T, Pettersson U. Spot hybridization for the detection of adenoviruses and enteroviruses. Clin Lab Med 1985 Sep;5(3):491-501

ENVIRONMENTAL POLLUTION

Feachem R, Miller C, Drasar B. Environmental aspects of cholera epidemiology. II. Occurrence and survival of <u>Vibrio cholerae</u> in the environment. Trop Dis Bull 1981 Oct;78(10):865-80 **

Feachem RG. Environmental aspects of cholera epidemiology, I. A review of selected reports of endemic and epidemic situations during 1961-1980. Trop Dis Bull 1981 Aug;78(8):675-98 **

Feachem RG. Environmental aspects of cholera epidemiology. III. Transmission and control. Trop Dis Bull 1982 Jan;79(1):1-47 **

EOSINOPHILS

Blackshaw AJ, Levison DA. Eosinophilic infiltrates of the gastrointestinal tract. J Clin Pathol 1986 Jan;39(1):1-7

EPIDEMIOLOGY AND EPIDEMIOLOGIC METHODS

Anderson RM. The population dynamics and epidemiology of intestinal nematode

÷

infections. Trans R Soc Trop Med Hyg 1986;80(5):686-96 **

Blaser MJ, Taylor DN, Feldman RA. Epidemiology of <u>Campylobacter</u> <u>jejuni</u> infections. Epidemiol Rev 1983;5:157-76 **

Bundy DAP. Epidemiological aspects of $\frac{Trichuris}{1986;80(5):706-18}$ **

Burchard GD. [Clinical importance, epidemiology and laboratory diagnosis of intestinal cryptosporidia infection]. Immun Infekt 1986 Apr;14(2):51-7

Calkins BM, Mendeloff AI. Epidemiology of inflammatory bowel disease. Epidemiol Rev 1986:8:60-91 **

Cvjetanovic B. Epidemiological models of diarrhoeal diseases [editorial]. J Diarrhoeal Dis Res 1985 Jun;3(2):63-4

D'Aoust J-Y. Recent developments in <u>Salmonella</u> epidemiology and methodology. Food Lab Newslett 1987 Apr;(9):32-6

Drossman DA, Lowman BC. Irritable bowel syndrome: epidemiology, diagnosis and treatment. Clin Gastroenterol 1985 Jul;14(3):559-73 **

Feachem R, Miller C, Drasar B. Environmental aspects of cholera epidemiology. II. Occurrence and survival of <u>Vibrio cholerae</u> in the environment. Trop Dis Bull 1981 Oct;78(10):865-80 **

Feachem RG. Environmental aspects of cholera epidemiology. I. A review of selected reports of endemic and epidemic situations during 1961-1980. Trop Dis Bull 1981 Aug;78(8):675-98 **

Feachem RG. Environmental aspects of cholera epidemiology. III. Transmission and control. Trop Dis Bull 1982 Jan;79(1):1-47**

Guerrant RL, Lohr JA, Williams EK. Acute infectious diarrhea. I. Epidemiology, etiology and pathogenesis. Pediatr Infect Dis 1986 May-Jun; 5(3):353-9

Hrdy DB. Epidemiology of rotaviral infection in adults. Rev Infect Dis 1987 May-Jun; 9(3):461-9

Levine MM, Edelman R. Enteropathogenic <u>Escherichia coli</u> of classic serotypes associated with infant diarrhea: epidemiology and pathogenesis. Epidemiol Rev 1984;6:31-51 **

MacDonald KL, Cohen ML. Epidemiology of travelers' diarrhea: current perspectives. Rev Infect Dis 1986 May-Jun;8(suppl 2):S117-21 **

Mayberry JF. Some aspects of the epidemiology of ulcerative colitis. Gut 1985 Sep;26(9):968-74

Minette HP. Epidemiologic aspects of salmonellosis in reptiles, amphibians, mollusks and crustaceans - a review. Int J Zoonoses 1984 Jun;11(1):95-104

Monsur KA. Epidemiology of Escherichia coli - an important but neglected field

1

[editorial]. J Diarrhoeal Dis Res 1985 Sep;3(3):128-30

Morger H, Steffen R, Schar M. Epidemiology of cholera in travellers, and conclusions for vaccination recommendations. Br Med J 1983 Jan 15;286(6360):184-6 **

Mosley WH, Khan MU. Cholera epidemiology - some environmental aspects. Prog Water Technol 1979:11(1-2):309-16 **

Navin TR, Juranek DD. Cryptosporidiosis: clinical, epidemiologic, and parasitologic review. Rev Infect Dis 1984 May-Jun;6(3):313-27 **

Navin TR. Cryptosporidiosis in humans: review of recent epidemiologic studies. Eur J Epidemiol 1985 Jun;1(2):77-83 **

Nelson JD. Etiology and epidemiology of diarrheal diseases in the United States. Am J Med 1985 Jun 28;78(suppl 6B):76-80 **

Numbur T. Epidemiological aspect of viral diarrhea in tropical area. $Jpn \ J$ Trop Med Hyg 1985 Mar;13(1):51 **

Pal SC, Nair GB. Epidemiology of campylobacteriosis in developing countries. ICMR Bull 1984 Jul;14(7):1-4 **

Pickering LK, Bartlett AV, Woodward WE. Acute infectious diarrhea among children in day care: epidemiology and control. Rev Infect Dis 1986 Jul-Aug;8 (4):539-47 **

Saran M, Dabral M, Srivastava RN, Sharma VK. Epidemiology of human rotavirus diarrhoea -- a review. J Indian Assoc Commun Dis 1982 Sep-Dec;5(3-4):50-7 **

Shuval HI, Yekutiel P, Fattal B. An epidemiological model for the potential nealth risk associated with various pathogens in wastewater irrigation. Wat Sci Technol 1986;18(10):191-8 **

Snuval HI, Fattal B, Yekutiel P. State of the art review: an epidemiological approach to the health effects of wastewater reuse. Wat Sci Technol 1986:18 (9):147-62 **

Snyder JD, Merson MH. The magnitude of the global problem of acute diarrhoeal disease: a review of active surveillance data. Bull WHO 1982;60(4):605-13 **

Steffen R. Epidemiologic studies of travelers' diarrhea, severe gastrointestinal infections, and cholera. Rev Infect Dis 1986 May-Jun;8(suppl 2):S122-30 **

Steffen R. Epidemiology of travellers' diarrhoea. Scand J Gastroenterol 1983;18(suppl 84):5-17 **

Sunoto. Diarrhoeal problems in Southeast Asia. Southeast Asian J Trop Med Public Health 1982 Sep;13(3):306-18 **

Taylor DN, Echeverria P. Etiology and epidemiology of travelers' diarrhea in Asia. Rev Infect Dis 1986 May-Jun;8(suppl 2):S136-41 **

Thoren A. The role of enteropathogenic E. coli in infantile diarrhoea: aspects

on bacteriology, epidemiology and therapy. Scand J Infect Dis 1983; (suppl 37):1-51

Turnbull PCB. Food poisoning with special reference to <u>Salmonella</u> - its epidemiology, pathogenesis and control. Clin Gastroenterol 1979 Sep;8(3): 665-714

ESCHERICHIA COLI AND ESCHERICHIA COLI INFECTIONS

Acres SD. Enterotoxigenic <u>Escherichia coli</u> infections in newborn calves: a review. J Dairy Sci 1985 Jan;68(1):229-56 **

Bachmann BJ. Linkage map of Escherichia coli K-12, edition 7. Microbiol Rev 1983 Jun;47(2):180-230

Cavalieri SJ, Bohach GA, Snyder IS. <u>Escherichia coli</u> alpha-hemolysin: characteristics and probable role in pathogenicity. Microbiol Rev 1984 Dec:48(4):326-43

Cooke EM. <u>Escherichia coli</u> - an overview. J Hyg (Lond) 1985 Dec;95(3): 523-30

Evans DJ, Jr., Evans DG. Classification of pathogenic <u>Escherichia coli</u> according to serotype and the production of virulence factors, with special reference to colonization-factor antigens. Rev Infect Dis 1983 Sep-Oct;5 (suppl 4):S692-701 **

Gross RJ. Escherichia coli. J Infect 1983 Nov;7(3):177-92

Gross RJ, Rowe B. <u>Escherichia</u> <u>coli</u> diarrhoea. J Hyg (Lond) 1985⁻ Dec: 95(3):531-50 **

Hinton M. The sub-specific differentiation of <u>Escherichia coli</u> with particular reference to ecological studies in young animals including man. J Hyg (Lond) $1985 \, \text{Dec}:95(3):595-609 \, **$

Karmali MA. Infection by verocytotoxin-producing Escherichia coli. Clin Microbiol Rev 1989 Jan;2(1):15-38 **

Klemm P. Fimbrial adhesions of <u>Escherichia</u> <u>coli</u>. Rev Infect Dis 1985 May-Jun:7(3):321-40

Kopecko DJ, Baron LS, Buysse J. Genetic determinants of virulence in <u>Shigella</u> and dysenteric strains of <u>Escherichia coli</u>: their involvement in the pathogenesis of dysentery. Curr Top Microbiol Immunol 1985;118:71-95

Levine MM, Edelman R. Enteropathogenic <u>Escherichia coli</u> of classic serotypes associated with infant diarrhea: epidemiology and pathogenesis. Epidemiol Rev 1984;6:31-51 **

Levine MM. <u>Escherichia coli</u> that cause diarrhea: enterotoxigenic, enteropathogenic, enteroinvasive, enterohemorrhagic, and enteroadherent. J Infect Dis 1987 Mar;155(3):377-89 **

Levine MM, Black RE, Brinton CC, Jr., Clements ML, Fusco P, Hughes TP, O'Donnell S, Robins-Browne R, Wood S, Young CR. Reactogenicity, immunogenicity, and efficacy studies of <u>Escherichia coli</u> type 1 somatic pili parenteral vaccine in man. Scand J Infect Dis 1982; (suppl 33):83-95

Monsur KA. Epidemiology of Escherichia coli - an important but neylected field [editorial]. J Diarrhoeal Dis Res 1985 Sep;3(3):128-30

Neilands JB, Bindereif A, Montyomerie JZ. Genetic basis of iron assimilation in pathogenic Escherichia coli. Curr Top Microbiol Immunol 1985;118:179-95

Neter E. Enteropathogenicity: recent developments. Klin Wochenschr 1982 Jul 15;60(14):699-701 **

Rowe B. The role of <u>Escherichia coli</u> in gastroenteritis. Clin Gastroenterol 1979 Sep;8(3):625-44

Sack RB. Escherichia coli and acute diarrheal disease. Ann Intern Med 1981 $Jan;94(1):\overline{129-30}$

Sansonetti PJ, d'Hauteville H, Formal SB, Toucas M. Plasmid-mediated invasiveness of <Shiyella-like> Escherichia coli. Ann Microbiol (Paris) 1982 May-Jun;133(3):351-5 **

Sternberg N. Evidence that adenine methylation influences DNA-protein interactions in Escherichia coli. J Bacteriol 1985 Oct;164(1):490-3

ESCHERICHIA COLI, ENTEROPATHOGENIC

Evans DJ, Jr., Evans DG. Classification of pathogenic <u>Escherichia coli</u> according to serotype and the production of virulence factors, with special reference to colonization-factor antigens. Rev Infect Dis 1983 Sep-Oct;5 (suppl 4):S692-701 **

Levine MM, Edelman R. Enteropathogenic <u>Escherichia coli</u> of classic serotypes associated with infant diarrhea: epidemiology and pathogenesis. Epidemiol Rev 1984;6:31-51 **

Porter P, Linggood MA. Development of oral vaccines for preventing diarrhoea caused by enteropathogenic <u>Escherichia</u> <u>coli</u>. J Infect 1983 Mar;6(2):111-21

Robins-Browne RM. Traditional enteropathogenic Escherichia coli of infantile diarrhea. Rev Infect Dis 1987 Jan-Feb; 9(1):28-53**

Thoren A. The role of enteropathogenic \underline{E} , \underline{coli} in infantile diarrhoea: aspects on bacteriology, epidemiology and therapy. Scand J Infect Dis 1983; (suppl 37):1-51

Wadstrom T, Baloda SB, Yuk YR. Cytotoxic and cytolytic proteins of enteropathogenic Escherichia coli and Salmonella: new concepts on possible role in intestinal colonization. Zentralbl Bakteriol Mikrobiol Hyg [A] 1986;(suppl 15):153-60

ESCHERICHIA COLI, ENTEROTOXIGENIC

Acres SD. Enterotoxigenic <u>Escherichia coli</u> infections in newborn calves: a review. J Dairy Sci 1985 Jan;68(1):229-56 **

Georgescu MB. [Colonization factors in enterotoxigenic <u>Escherichia coli</u>]. Rev Ig [Bacteriol] 1985 Apr-Jun;30(2):115-30

Isaacson RE. Pili of enterotoxigenic <u>Escherichia</u> <u>coli</u> from pigs and calves. Adv Exp Med Biol 1985;185:83-99

Mooi FR, de Graaf FK. Molecular biology of fimbriae of enterotoxigenic Escherichia coli. Curr Top Microbiol Immunol 1985;118:119-38

Powell DW. Enterotoxigenic diarrhea: mechanisms and prospects for therapy. Pharmacol Ther 1984:23(3):407-16 **

Sack RB. Human diarrheal disease caused by enterotoxigenic <u>Escherichia</u> <u>coli</u>. Annu Rev Microbiol 1975;29:333-53

Takeda Y, Shimonishi Y, Yamamoto T, Takeda T. [Enterotoxins produced by enterotoxigenic <u>Escherichia coli</u>]. Tanpakushitsu Kakusan Koso 1986 Mar;31 (suppl 4):324-52

Wadstrom T, Baloda SB. Molecular aspects on small bowel colonization by enterotoxigenic <u>Escherichia coli</u>. Microecol Ther 1986;16:243-55

FEEDING BEHAVIOR

Feeding during diarrhea. Nutr Rev 1986 Mar;44(3):102

Jelliffe EFP, Jelliffe DB, Feldon K, Ngokwey N. Traditional practices concerning feeding during and after diarrhoea (with special reference to acute dehydrating diarrhoea in young children). World Rev Nutr Diet 1987;53:218-95

Research on improving infant feeding practices to prevent diarrhoea or reduce its severity: Memorandum from a JHU/WHO meeting. Bull WHO 1989;67(1):27-33 **

FIBER

Jenkins DJA, Jenkins AL, Wolever TMS, Rao AV, Thompson LU. Fiber and starchy foods: gut function and implications in disease. Am J Gastroenterol 1986 0ct:81(10):920-30 **

FLUID THERAPY

Feld LG, Kaskel FJ, Schoeneman MJ. The approach to fluid and electrolyte

therapy in pediatrics. Adv Pediatr 1988;35:497-536

FOOD HYPERSENSITIVITY

Auricchio S, Cucchiara S, D'Antonio AM. De Ritis G. De Vizia B. Follo D. Iaccarino E. Gastrointestinal allergy or intolerance to multiple foods in severe chronic diarrhea in early infancy. In: Lebenthal E, ed. Chronic diarrhea in children. New York: Raven Press, 1984:425-34

Auricchio S. Gluten-sensitive enteropathy and infant nutrition. J Pediatr Gastroenterol Nutr 1983;2(supp) 1):S304-9

Pearson DJ, McKee A. Food allergy. Adv Nutr Res 1985;7:1-37

FUNGI

Chretien JH, Garagusi VF. Current management of fungal enteritis. Med Clin North Am 1982 May;66(3):675-87 **

GASTRIC EMPTYING

Minami H, McCallum RW. The physiology and pathophysiology of gastric emptying in humans. Gastroenterology 1984 Jun;86(6):1592-1610 **

GASTRITIS AND PEPTIC ULCER

Blaser MJ. Gastric <u>Campylobacter</u>-like organisms, gastritis, and peptic ulcer disease. Gastroenterology 1987 Aug;93(2):371-83

Marshall BJ. <u>Campylobacter pyloridis</u> and gastritis. J Infect Dis 1986 Apr; 153(4):650-7

GASTROENTERITIS

Baqai R. Rotavirus gastroenteritis [editorial]. J Pak Med Assoc 1983 Oct; 33(10):240-2 **

Barnett B. Viral gastroenteritis. Med Clin North Am 1983 Sep;67(5):1031-58 **

Blacklow NR, Cukor G. Viral gastroenteritis. N Engl J Med 1981 Feb 12;304 (7):397-406 **

Blackshaw AJ, Levison DA. Eosinophilic infiltrates of the gastrointestinal tract. J Clin Pathol 1986 Jan; 39(1):1-7

Broor S, Singh V. Viral gastroenteritis. Indian J Gastroenterol 1984 Oct;3 (4):225-9 **

Choudhuri PK. Acute gastroenteritis in children. J Indian Med Assoc 1987 Jul;85(7):214-5

Christensen ML. Human viral gastroenteritis. Clin Microbiol Rev 1989 Jan; 2(1):51-89 **

Craun GF. A summary of waterborne illness transmitted through contaminated groundwater. J Environ Health 1985 Nov-Dec;48(3):122-7 **

Drachman RH. Acute infectious gastroenteritis. Pediatr Clin North Am 1974 Aug;21(3):711-37

Elliott EJ, Walker-Smith JA, Farthing MJG. The role of bicarbonate and base precursors in treatment of acute gastroenteritis. Arch Dis Child 1987 Jan;62 (1):91-5 **

Konno T. [Viral gastroenteritis: serologiccal characteristics and molecular epidemiology of human rotavirus]. Rinsho Byori 1985 Feb;33(2):129-35

Nazer H. Astrovirus gastroenteritis. J Trop Pediatr 1985 Apr;31(2):67-71 **

Rodrick GE, Hood MA, Blake NJ. Human <u>Vibrio</u> gastroenteritis. Med Clin North Am 1982 May:66 (3):665-73

Rowe 8. The role of Escherichia coli in gastroenteritis. Clin Gastroenterol 1979 Sep;8(3):625-44

Walker-Smith JA. Nutritional management of acute gastroenteritis -- rehydration and realimentation. Hum Nutr Appl Nutr 1986;40A(suppl 1):39-43

Wolf JL, Schreiber DS. Viral gastroenteritis. Med Clin North Am 1982 May;66 (3):575-95

Wood DJ. Adenovirus gastroenteritis. Br Med J 1988 Jan 23;296(6617): 229-30

GASTROINTESTINAL AND COLONIC MOTILITY

7

Feldman M, Schiller LR. Disorders of gastrointestinal motility associated with diabetes mellitus. Ann Intern Med 1983 Mar; 98(3):378-84**

Milla PJ. Intestinal motility and its disorders. Clin Gastroenterol 1986 Jan;15(1):121-36

Misiewicz JJ. Human colonic motility. Scand J Gastroenterol 1984;19(suppl 93):43-51 **

Ooms L. Alterations in intestinal fluid movement. Scand J Gastroenterol 1983;18(suppl 84):65-77 **

Read NW. The relationships between colonic motility and transport. Scand J Gastroenterol 1984;19(suppl 93):35-42 **

Read NW. Speculations on the role of motility in the pathogenesis and treatment of diarrhoea. Scand J Gastroenterol 1983;18(suppl 84):45-63 **

Tucker H, Schuster MM. Irritable bowel syndrome: newer pathophysiologic concepts. Adv Intern Med 1982;27:183-204

Weber J, Ducrotte P. Colon motility in health and disease. Dig Dis 1987;5(1):1-12

GASTROINTESTINAL DISEASES

Allason-Jones E, Mindel A. Sex and the bowel. Int J Color Dis 1987 Feb;2(1): 32-7

Boyd WP, Jr., Bachman BA. Gastrointestinal infections in the compromised host. Med Clin North Am 1982 May:66(3):743-53

Chandra RK. Nutritional regulation of immunity and infection in the gastrointestinal tract. J Pediatr Gastroenterol Nutr 1983;2(suppl 1):S181-7

DiJoseph JF, Taylor JA, Mir GN. Alpha-2 receptors in the gastrointestinal system; a new therapeutic approach. Life Sci 1984 Sep 3;35(10):1031-42

Doe WF. Immunodeficiency and the gastrointestinal tract. Clin Gastroenterol 1983 Sep;12(3):839-53

Kubba R. Gastrointestinal manifestations of skin diseases: a review. Trop Gastroenterol 1983 Apr-Jun;4(2):67-78

Lake AM, Kleinman RE, Walker WA. Enteric alimentation in specialized gastrointestinal problems: an alternative to total parenteral nutrition. Adv Pediatr 1981;28:319-39

Lolekha S. Consequences of treatment of gastrointestinal infections. Scand J Infect Dis 1986; (suppl 49):154-9 **

Marks J. The relationship of gastrointestinal disease and the skin. Clin Gastroenterol 1983 Sep;12(3):693-712

Pitlik S, Berger SA, Huminer D. Nonenteric infections acquired through contact with water. Rev Infect Dis 1987 Jan-Feb;9(1):54-63 **

Prasad AS. The role of zinc in gastrointestinal and liver disease. Clin Gastroenterol 1983 Sep;12(3):713-41

Russell RI. Intestinal adaptation to an elemental diet. Proc Nutr Soc 1985 Feb;44(1):87-93

Shiner M. Autoimmunity in gastrointestinal diseases. Arq Gastroenterol 1986 Apr-Jun;23(2):99-103 **

Thorne GM. Gastrointestinal infections--dietary interactions. J Am Coll Nutr 1986;5(5):487-99 **

GENETICS. MICROBIAL

Guidolin A, Manning PA. Genetics of <u>Vibrio</u> <u>cholerae</u> and its bacteriophages. Microbiol Rev 1987 Jun;51(2):285-98 **

Hale TL, Formal SB. Genetics of virulence in Shigella. Microbial Pathogen 1986 Dec;1(6):511-8 **

Kopecko DJ, Baron LS, Buysse J. Genetic determinants of virulence in <u>Shigella</u> and dysenteric strains of <u>Escherichia</u> coli: their involvement in the pathogenesis of dysentery. Curr Top Microbiol Immunol 1985;118:71-95

Mekalonos JJ. Cholera toxin: genetic analysis, regulation, and role in pathogenesis. Curr Top Microbiol Immunol 1985;118:97-118 **

Mool FR, de Graaf FK. Molecular biology of fimbriae of enterotoxigenic Escherichia coli. Curr Top Microbiol Immunol 1985;118:119-38

Neilands JB, Bindereif A, Montyomerie JZ. Genetic basis of iron assimilation in pathogenic Escherichia coli. Curr Top Microbiol Immunol 1985;118:179-95

Piggot PJ, Hoch JA. Revised genetic linkage map of <u>Bacillus</u> <u>subtilis</u>. Microbiol Rev 1985 Jun;49(2):158-79

Wakelin D. Genetic and other constraints on resistance to infection with gastrointestinal nematodes. Trans R Soc Trop Med Hyg 1986:80(5):742-7 **

GIARDIA AND GIARDIASIS

Akin EW, Jakubowski W. Drinking water transmission of giardiasis in the United States. Wat Sci Technol 1986;18(10):219-26 **

Datta T. Intestinal amoebiasis and giardiasis in children. Indian J Pediatr 1985 Mar-Apr;52(415):184-5 **

Pickering LK. Problems in diagnosing and managing giardiasis. Pediatr Infect Dis 1985 May-Jun;4(suppl 3):S6-10

Steffen R. Epidemiologic studies of travelers' diarrhea, severe gastrointestinal infections, and cholera. Rev Infect Dis 1986 May-Jun;8(suppl 2):S122-30 **

Stevens DP. Selective primary health care: strategies for control of disease in the developing world. XIX. Giardiasis. Rev Infect Dis 1985 Jul-Aug;7(4): 530-5 **

Turner JA. Giardiasis and infections with <u>Dientamoeba</u> <u>fragilis</u>. Pediatr Clin North Am 1985 Aug; 32(4):865-80 **

Wolfe MS. The treatment of intestinal protozoan infections. Med Clin North Am 1982 May; 66(3):707-20

Wright SG. Parasites and travellers' diarrhoea. Scand J Gastroenterol 1983; 18(suppl 84):25-9 **

GROWTH AND GROWTH DISORDERS

Rohde J, Northrup RS. Diarrhoea: a nutritional disease. J Indian Med Assoc 1987 Jul;85(7):196-202

Rowland MGM, Rowland SGJG. Growth faltering in diarrhoea. <u>In:</u> Taylor TG, Jenkins NK, eds. Proceedings of the XIII International Congress of Nutrition, Brighton, 18-23 Aug 1985. London: Libby, 1986:115-9

Soutter VL, Kristidis P, Gruca MA, Gaskin KJ. Chronic undernutrition/growth retardation in cystic fibrosis. Clin Gastroenterol 1986 Jan;15(1):137-55

HEALTH, HEALTH EDUCATION, HANDWASHING AND HYGIENE

Abed FH. Household teaching of ORT in rural Bangladesh. Assign Child 1983; (61/62):249-65 **

Abed FH. Household teaching of oral rehydration therapy in rural Bangladesh. J Indian Med Assoc 1987 Jul;85(7):205-9

Blaser MJ. Environmental interventions for the prevention of travelers' diarrhea. Rev Infect Dis 1986 May-Jun;8(suppl 2):S142-50 **

Briscoe J. Public health in rural India: the case of excreta disposal. Cambridge: Center for Population Studies, Harvard University, 1976. xix, 414 p. PhD Thesis. (Research paper, 12)

Derryberry M. Health education aspects of sanitation programmes in rural areas and small communities. Bull WHO 1954;10(2):145-54 **

Feachem R, McGarry M, Mara D, eds. Water, wastes and health in hot climates. London: Wiley, 1977. xvi, 399 p.

Feachem RG. Interventions for the control of diarrhoeal diseases among young children: promotion of personal and domestic hygiene. Bull WHO 1984:62(3):467-76 **

Feachem RG, Burns E, Cairncross AM, Cronin A, Cross R, Curtis D, Khan MK, Lamb D, Southal H. Water, health and development: an interdisciplinary evaluation. London: Tri-Med Books, 1978. 267 p.

Green EC. Traditional healers, mothers and childhood diarrheal disease in Swaziland: the interface of anthropology and health education. Soc Sci Med 1985;20(3):277-85 **

HELMINTHS AND HELMINTHIASIS

Arfaa F. Selective primary health care: strategies for control of disease in the developing world. XII. Ascariasis and trichuriasis. Rev Infect Dis 1984 May-Jun;6(3):364-73 **

Befus D. Immunity in intestinal helminth infections: present concepts, future directions. Trans R Soc Trop Med Hyg 1986;80(5):735-41 **

Butterworth AE. Cell-mediated damage to helminths. Adv Parasitol 1984;23:143-235

Cline BL. Current drug regimens for the treatment of intestinal helminth infections. Med Clin North Am 1982 May:66 (3):721-42

Cook GC. The clinical significance of gastrointestinal helminths - a review. Trans R Soc Trop Med Hyg 1986;80(5):675-85 **

Crompton DWT. Nutritional aspects of infection. Trans R Soc Trop Med Hyg 1986;80(5):697-705 **

Intestinal protozoan and helminthic infections. WHO Tech Rep Ser 1981;(666): 1-150

Janssen PAJ, van Den Bossche H. Treatment of helminthasis. Scand J Infect Dis 1982;(suppl 36):52-7

Shuval HI, Yekutiel P. Fattal B. An epidemiological model for the potential health risk associated with various pathogens in wastewater irrigation. Wat Sci Technol 1986;18(10):191-8 **

Shuval HI, Fattal B, Yekutiel P. State of the art review: an epidemiological approach to the health effects of wastewater reuse. Wat Sci Technol 1986;18 (9):147-62 **

HEMOLYSINS

Cavalieri SJ, Bohach GA, Snyder IS. <u>Escherichia coli</u> alpha-hemolysin: characteristics and probable role in pathogenicity. Microbiol Rev 1984 Dec:48(4):326-43

Ljungh A, Wadstrom T. Aeromonas toxins. Pharmacol Ther 1982;15(3):339-54 **

Ljungh A, Eneroth P, Wadstrom T. Cytotonic enterotoxin from <u>Aeromonas</u> hydrophila. Toxicon 1982;20(4):787-94 **

HEPATITIS, VIRAL

Judson FN. Sexually transmitted viral hepatitis and enteric pathogens. Urol

Clin North Am 1984 Feb:11(1):177-85

HORMONES

Gyr K. Infectious diarrhoea and gastrointestinal hormones: potential therapeutic implications. Scand J Gastroenterol 1983;18(suppl 84):135-40 **

Nicholl CG, Polak JM, Bloom SR. The hormonal regulation of food intake, digestion, and absorption. Annu Rev Nutr 1985;5:213-39

HOST-PARASITE RELATIONS

Befus D. Immunity in intestinal helminth infections: present concepts, future directions. Trans R Soc Trop Med Hyg 1986;80(5):735-41 **

Mitchell GF. Injection versus infection: the cellular immunology of parasitism. Parasitol Today 1987 Apr;3(4):106-11

Prichard RK. Interaction of host physiology and efficacy of antiparasitic drugs. Vet Parasitol 1985 Aug;18(2):103-10

Pritchard DI. Antigens of gastrointestinal nematodes. Trans R Soc Trop Med Hyg 1986;80(5):728-34 **

HYBRIDIZATION

Echeverria P, Seriwatana J, Sethabutr O, Taylor DN. DNA hybridization in the diagnosis of bacterial diarrhea. Clin Lab Med 1985 Sep;5(3):447-62 **

Hyypia T, Pettersson U. Spot hybridization for the detection of adenoviruses and enteroviruses. Clin Lab Med 1985 Sep;5(3):491-501

HYPERNATREMIA

Meeuwisse GW. High sugar worse than high sodium in oral rehydration solutions. Acta Paediatr Scand 1983 Mar;72(2):161-6 **

Paneth N. Hypernatremic dehydration of infancy: an epidemiologic review. Am J Dis Child 1980 Aug:134(8):785-92 **

ILEOSTOMY

Metcalf AM, Phillips SF. Ileostomy diarrhoea. Clin Gastroenterol 1986 Jul;15 (3):705-22

IMMUNE RESPONSE AND TOLERANCE

Rowley D, La Brooy J. Intestinal immune responses in relation to diarrhoeal diseases. J Diarrhoeal Dis Res 1986 Mar;4(1):1-9 **

Shiner M. Autoimmunity in gastrointestinal diseases. Arq Gastroenterol 1986 Apr-Jun:23(2):99-103 **

IMMUNITY

Befus D. Immunity in intestinal helminth infections: present concepts, future directions. Trans R Soc Trop Med Hyg 1986;80(5):735-41 **

Boyd WP, Jr., Bachman BA. Gastrointestinal infections in the compromised host. Med Clin North Am 1982 May:66(3):743-53

Chandra RK. Nutritional regulation of immunity and infection in the gastrointestinal tract. J Pediatr Gastroenterol Nutr 1983;2(suppl 1): S181-7

Doe WF. Immunodeficiency and the gastrointestinal tract. Clin Gastroenterol 1983 Sep;12(3):839-53

Doe WF, Hapel AJ. Intestinal immunity and malabsorption. Clin Gastroenterol 1983 May:12(2):415~35

Elson CO, Kagnoff MF, Fiocchi C, Befus AD, Targan S. Intestinal immunity and inflammation: recent progress. Gastroenterology 1986 Sep;91(3):746-68

Finkelstein RA, Sciortino CV, McIntosh MA. Role of iron in microbe-host interactions. Rev Infect Dis 1983 Sep-Oct;5(suppl 4):S759-77

Freier S, Kuperman O. Immune regulation in the gastrointestinal tract. C Pediatr Gastroenterol Nutr 1983;2(suppl 1):S310-4

May JT. Antimicrobial properties and microbial contaminants of breast milk - an update. Aust Paediatr J 1984 Nov;20(4):265-9 **

Pritchard DI. Antigens of gastrointestinal mematodes. Trans R Soc Trop Med Hyg 1986;80(5):728-34 **

Sepulveda B. Amebiasis: host-pathogen biology. Rev Infect Dis 1982 Nov-Dec;4 (6):1247-53 **

Svennerholm A-M, Jertborn M, Gothefors L, Karim A, Sack D, Holmgren J. Secretory immunity to <u>Vibrio cholerae</u> bacteria and cholera toxin: prospects for an improved cholera vaccine. <u>In</u>: Takeda Y, Miwatani T, eds. Bacterial diarrheal diseases. Tokyo: KTK Scientific Publishers, 1985:169-74 **

Trissl D. Immunology of Entamoeba histolytica in human and animal hosts. Rev Infect Dis 1982 Nov-Dec;4(6):1154-84 **

IMMUNOASSAY

Schmidt NJ. Rapid viral diagnosis. Med Clin North Am 1983 Sep;67(5): 953-72 **

Yolken RH, Leggiadro RJ. Immunoassays for the diagnosis of viral enteric pathogens. Diagn Microbiol Infect Dis 1986 Mar;4(suppl 3):S61-9

IMMUNOLOGIC DEFICIENCY SYNDROMES

Arbo A, Santos Jl. Diarrheal diseases in the immunocompromised host. Pediatr Infect Dis $1987 \ \text{Oct}; 6(10):894-906$

Hitzig WH. Protean appearances of immunodeficiencies: syndromes and inborn erros involving other systems which express associated primary immunodeficiency. Birth Defects 1983;19(3):307-12 **

IMPACT AND EVALUATION STUDIES

Briscoe J, Feachem RG, Rahaman MM. Measuring the impact of water supply and sanitation facilities on diarrhoea morbidity: prospects for case-control methods. Geneva: World Health Organization, 1985. 71 p. (WHO/CWS/85.3; CDD/OPR/85.1) **

Briscoe J. Water supply and health in developing countries: selective primary health care revisited. Am J Public Health 1984 Sep:74(9):1009-13 **

Chowdhury AMR, D'Souza S. A design and field methods for monitoring impact on mortality of an oral therapy programme. Dhaka: International Centre for Diarrhoeal Disease Research, Bangladesh, 1982. 23 p. (ICDDR,B working paper 27) **

Cvjetanovic B. Health effects and impact of water supply and sanitation. World Health Stat Q 1986;39(1):105-17 **

Cvjetanovic B, Chen L, Kronmall R, Rohde C, Suskind R. Measuring and evaluating diarrhea and malabsorption in association with village water supply and sanitation: a review of the Food Wastage/Sanitation Cost Benefit Methodology Project (Guatemala). Arlington, Virginia: Water and Sanitation for Health Project, 1981. 36 p. (WASH technical report, 12)

Cvjetanovic B. Sanitation versus immunization in control of enteric and diarrhoeal diseases. Prog Water Technol 1979;11(1-2):81-7 **

Esrey SA, Habicht J-P. Epidemiologic evidence for health benefits from improved water and sanitation in developing countries. Epidemiol Rev 1986;8:117-28 **

INFANT, LOW BIRTH WEIGHT

Ashworth A, Feachem RG. Interventions for the control of diarrhoeal diseases among young children: prevention of low birth weight. Bull WHO 1985;63(1): 165-84 **

INFANT MORTALITY

Ashworth A. International differences in child mortality and the impact of malnutrition. Hum Nutr Clin Nutr 1982;36C(4):279-88 **

Ashworth A. International differences in infant mortality and the impact of malnutrition: a review. Hum Nutr Clin Nutr 1982;36C(1):7-23 **

Ashworth A, Feachem RG. Interventions for the control of diarrhoeal diseases among young children: prevention of low birth weight. Bull WHO 1985;63(1): 165-84

Ashworth A, Feachem RG. Interventions for the control of diarrhoeal diseases among young children: weaning education. Bull WHO 1985;63(6):1115-27 **

INFANT NUTRITION AND FOOD

١

Auricchio S. Cucchiara S. D'Antonio AM. De Ritis G. De Vizia B. Follo D. Iaccarino E. Gastrointestinal allergy or intolerance to multiple foods in severe chronic diarrhea in early infancy. In: Lebenthal E. ed. Chronic diarrhea in children. New York: Raven Press. 1984:425-34

Auricchio S. Gluten-sensitive enteropathy and infant nutrition. J Pediatr Gastroenterol Nutr 1983;2(suppl 1):S304-9

Behar M. The role of feeding and nutrition in the pathogeny and prevention of diarrheic processes. Bull Pan Am Health Organ 1975;9(1):1-9 **

Brown KH, MacLean WC, Jr. Nutritional management of acute diarrhea: an appraisal of the alternatives. Pediatrics 1984 Feb;73(2):119-25 **

Hamilton JR. Nutritional therapy for chronic diarrhea. <u>In: Lebenthal E, ed.</u> Chronic diarrhea in children. New York: Raven Press, 1984:535-40 **

Jelliffe EFP, Jelliffe DB, Feldon K, Ngokwey N. Traditional practices concerning feeding during and after diarrhoea (with special reference to acute dehydrating diarrhoea in young children). World Rev Nutr Diet 1987;53:218-95

Lake AM, Kleinman RE, Walker WA. Enteric alimentation in specialized gastrointestinal problems: an alternative to total parenteral nutrition. Adv Pediatr 1981;28:319-39

Lebenthal E, Rossi TM. Intractable diarrhea of infancy: an alternative treatment strategy. Postgrad Med 1983 Aug;74(2):153-9 **

1

Molla A, Molla AM, Sarker SA. Malabsorption in enteric infection; a nutritional cost in children with diarrhea. <u>In</u>: Chagas C, Keusch GT, eds. The interaction of parasitic diseases and nutrition, 22-26 Oct 1985. Vaticana: Pontificia Academia Scientiarum, 1985:71-80 **

Motil KJ, Grand RJ. Nutritional management of inflammatory bowel disease. Pediatr Clin North Am 1985 Apr;32(2):447-69 **

Reddy V. Interaction between malnutrition and diarrhoea with particular reference to pediatric practice. Indian J Gastroenterol 1985 Jul;4(3):183-6 **

Research on improving infant feeding practices to prevent diarrhoea or reduce its severity: Memorandum from a JHU/WHO meeting. Bull WHO 1989;67(1):27-33 **

Rohde J, Northrup RS. Diarrhoea: a nutritional disease. J Indian Med Assoc 1987 Jul;85(7):196-202

Wharton BA. Food for weanling: the next priority in infant nutrition. Acta Paediatr Scand 1986;(suppl 323):96-102

Yip WC, Tay JS, Ho TF, Wong HB. Total parenteral nutrition in paediatric practice. J Singapore Paediatr Soc 1984;26(1-2):59-72

INFANT NUTRITION DISORDERS

Ashworth A. International differences in child mortality and the impact of malnutrition. Hum Nutr Clin Nutr 1982;36C(4):279-88 **

Ashworth A. International differences in infant mortality and the impact of malnutrition: a review. Hum Nutr Clin Nutr 1982;36C(1):7-23 **

Auricchio S. Gluten-sensitive enteropathy and infant nutrition. J Pediatr Gastroenterol Nutr 1983;2(suppl 1):S304-9

Chen LC, Huq E, Huffman SL. A prospective study of the risk of diarrhoeal diseases according to the nutritional status of children. Am J Epidemiol 1981 Aug;114(2):284-92 **

Does malnutrition predispose children to diarrhoea? Nutr Rev 1985 May;43(5): 144-5

Gracey M. The intestinal microflora in malnutrition and protracted diarrhea in infancy. In: Lebenthal E, ed. Chronic diarrhea in children. New York: Raven Press, 1984:223-36

Keusch GT, Scrimshaw NS. Selective primary health care: strategies for control of disease in the developing world. XXIII. Control of infection to reduce the prevalence of infantile and childhood malnutrition. Rev Infect Dis 1986 Mar-Apr;8(2):273-87 **

Motil KJ, Grand RJ. Nutritional management of inflammatory bowel disease. Pediatr Clin North Am 1985 Apr;32(2):447-69 **

Reddy V. Interaction between malnutrition and diarrhoea with particular reference to pediatric practice. Indian J Gastroenterol 1985 Jul;4(3): 183-6 **

Rohde J. Northrup RS. Diarrhoea: a nutritional disease. J Indian Med Assoc 1987 Jul:85(7):196-202

Soutter VL, Kristidis P, Gruca MA, Gaskin KJ. Chronic undernutrition/growth retardation in cystic fibrosis. Clin Gastroenterol 1986 Jan:15(1):137-55

Tontisirin K, Valyasevi A. Protein energy malnutrition related to diarrhea in Thai children. J Nutr Sci Vitaminol (Tokyo) 1981:27(6):513-30 **

INFECTIONS

Crompton DWT. Nutritional aspects of infection. Trans R Soc Trop Med Hyg 1986;80(5):697-705 **

Finkelstein RA, Sciortino CV, McIntosh MA. Role of iron in microbe-host interactions. Rev Infect Dis 1983 Sep-Oct:5(suppl 4):S759-77

Gracey MS. Nutrition, bacteria and the gut. Br Med Bull 1981 Jan;37(1):

Ho DD, Pomerantz RJ, Kaplan JC. Pathogenesis of infection with human immunodeficiency virus. N Engl J Med 1987 Jul 30;317(5):278-86

Hoerr RA, Young VR. Alterations in nutrient intake and utilization caused by disease. Ann NY Acad Sci 1987;499:124-31

Keusch GT, Scrimshaw NS. Selective primary health care: strategies for control of disease in the developing world. XXIII. Control of infection to reduce the prevalence of infantile and childhood malnutrition. Rev Infect Dis 1986 Mar-Apr:8(2):273-87 **

McHenry MC, Weinstein AJ. Antimicrobial drugs and infections in ambulatory patients. Some problems and perspectives. Med Clin North Am 1983 Jan;67(1): 3-16

Pickering LK. Infections in day care. Pediatr Infect Dis J 1987 Jun;6(6):614-7 **

Sharp JCM. Infections associated with milk and dairy products in Europe and North America, 1980-85. Bull WHO 1987;65(3):397-405 **

INFLAMMATION

Elson CO, Kagnoff MF, Fiocchi C, Befus AD, Targan S. Intestinal immunity and inflammation: recent progress. Gastroenterology 1986 Sep;91(3):746-68

INFLAMMATORY BOWEL DISEASE

Booth IW, Harries JT. Inflammatory bowel disease in childhood. Gut 1984 Feb;25(2):188-202 **

Calkins BM, Mendeloff AI. Epidemiology of inflammatory bowel disease. Epidemiol Rev 1986;8:60-91 **

Cello JP. Inflammatory and malignant diseases of the small bowel causing malabsorption. Clin Gastroenterol 1983 May;12(2):511-32

Kodner IJ, Fry RD. Inflammatory bowel disease. Clin Symp 1982;34(1):3-32

Motil KJ, Grand RJ. Nutritional management of inflammatory bowel disease. Pediatr Clin North Am 1985 Apr;32(2):447-69 **

Pillai DK, Matts SG. Chronic inflammatory bowel disease--a review. Br J Clin Pract 1983 May: 37(5):165-72

Pringot J, Bodart P. Inflammatory diseases of the small bowel. Verh K Acad Geneeskd Belg 1984;46(5):309-49 **

Rosenberg IH, Bengoa JM, Sitrin MD. Nutritional aspects of inflammatory bowel disease. Annu Rev Nutr 1985;5:463-84

Strober W. Animal models for inflammatory bowel disease--an overview. Dig Dis Sci 1985 Dec;30(suppl 12):S3-10

INTERVENTIONS

Ashworth A, Feachem RG. Interventions for the control of diarrhoeal diseases among young children: prevention of low birth weight. Bull WHO 1985;63(1): 165-84 **

Ashworth A, Feachem RG. Interventions for the control of diarrhoeal diseases among young children: weaning education. Bull WHO 1985;63(6):1115-27 **

Black RH. Invited discussion of Dr R M Glasse's paper. In: Proceedings of the Cholera Research Symposium, Honolulu, Hawaii, 24-29 Jan 1965. Washington, D.C.: U S Government Printing Office, 1965:340

Blaser MJ. Environmental interventions for the prevention of travelers diarrhea. Rev Infect Dis 1986 May-Jun;8(suppl 2):S142-50 **

Briscoe J. Intervention studies and the definition of dominant transmission routes. Am J Epidemiol 1984 Sep;120(3):449-55 **

Creese AL. Cost effectiveness of potential immunization interventions against diarrhoeal disease. Soc Sci Med 1986;23(3):231-40 **

de Zoysa I, Feachem RG. Interventions for the control of diarrhoeal diseases among young children: chemoprophylaxis. Bull WHO 1985;63(2):295-315

Esrey SA, Feachem RG, Hughes JM. Interventions for the control of diarrhoeal diseases among young children: improving water supplies and excreta disposal facilities. Bull WHO 1985;63(4):757-72 **

Feachem RG, Hoyan RC, Merson MH. Diarrhoeal disease control: reviews of potential interventions. Bull WHO 1983;61(4):637-40 **

Feachem RG, Koblinsky MA. Interventions for the control of diarrhoeal diseases among young children: measles immunization. Bull WHO 1983;61(4): 641-52 **

Feachem RG, Koblinsky MA. Interventions for the control of diarrhoeal diseases among young children: promotion of breast-feeding. Bull WHO 1984;62(2): 271-91 **

Feachem RG. Interventions for the control of diarrhoeal diseases among young children: promotion of personal and domestic hygiene. Bull WHO 1984;62(3):467-76 **

Feachem RG. Preventing diarrhoea: what are the policy options? Health Policy Plann 1986;1(2):109-17 **

Howard P. Diarrhoeal diseases--priorities in research and intervention. Papua New Guinea Med J 1986 Jun;29(2):125-9

Kawata K. Water and other environmental interventions—the minimum investment concept. Am J Clin Nutr 1978 Nov;31(11):2114-23 **

Keusch GT, Scrimshaw NS. Selective primary health care: strategies for control of disease in the developing world. XXIII. Control of infection to reduce the prevalence of infantile and childhood malnutrition. Rev Infect Dis 1986 Mar-Apr;8(2):273-87 **

Khan MU. Interruption of transmission of diarrhoeal agents. <u>In</u>: Programme, papers and abstracts of the Third Asian Conference on Diarrhoeal Diseases, Bangkok, 10-14 Jun 1985:173-80

Kuo C. Measures to control diarrhoeal diseases--environmental sanitation. Regional Meeting on Cholera and Diarrhoeal Diseases, Alexandria, 1-5 Jun 1978. Alexandria: Regional Office for the Eastern Mediterranean, World Health Organization, 1978. 6 p. **

Martinez-Palomo A, Martinez-Baez M. Selective primary health care: strategies for control of disease in the developing world. X. Amebiasis. Rev Infect Dis 1983 Nov-Dec;5(6):1093-1102 **

Reddy V. Interaction between malnutrition and diarrhoea with particular reference to pediatric practice. Indian J Gastroenterol 1985 Jul;4(3):183-6 **

Research on improving infant feeding practices to prevent diarrhoea or reduce its severity: Memorandum from a JHU/WHO meeting. Bull WHO 1989:67(1):27-33 **

Walsh JA, Warren KS. Selective primary health care: an interim strategy for disease control in developing countries. Soc Sci Med 1980;14C(2):145-63 **

Warren KS. Selective primary health care: strategies for control of disease in

the developing world. I. Schistosomiasis. Rev Infect Dis 1982 May-Jun;4 (3):715-26 **

INTESTINAL ABSORPTION

Auricchio S. Peptide digestion and absorption in the small intestinal mucosa during acute and chronic diarrhea. In: Lebenthal E, ed. Chronic diarrhea in children. New York: Raven Press, 1984:179-91

Freeman HJ, Sleisenger MH, Kim YS. Human protein digestion and absorption: normal mechanisms and protein-energy malnutrition. Clin Gastroenterol 1983 May;12(2):357-78

Lake AM, Kleinman RE, Walker WA. Enteric alimentation in specialized gastrointestinal problems: an alternative to total parenteral nutrition. Adv Pediatr 1981;28:319-39

Mahalanabis D, Patra FC. In search of a super oral rehydration solution: can optimum use of organic solute-mediated sodium absorption lead to the development of an absorption promoting drug? J Diarrhoeal Dis Res 1983 Jun;1 (2):76-81 .**

Milla PJ. Disorders of electrolyte absorption. Clin Gastroenterol 1982 Jan;11(1):31-46

Molla A, Molla AM, Sarker A, Knatoon M, Rahaman MM. Effects of acute diarrhea on absorption of macronutrients during disease and after recovery. In: Chen LC, Scrimshaw NS, eds. Diarrhea and malnutrition: interactions, mechanisms, and interventions. New York: Plenum, 1983:143-54

Molla AM, Molla A, Sarker SA, Rahaman MM. Food intake during and after recovery from diarrhoea in children. <u>In: Chen LC, Scrimshaw NS, eds. Diarrhea and malnutrition: interactions, mechanisms, and interventions. New York: Plenum, 1983:113-23 **</u>

Nicholl CG, Polak JM, Bloom SR. The hormonal regulation of food intake, digestion, and absorption. Annu Rev Nutr 1985;5:213-39

Perman JA, Modler S. Role of the intestinal microflora in disposition of nutrient in the gastrointestinal tract. J Pediatr Gastroenterol Nutr 1983;2(suppl 1):S193-6

Ravich WJ, Bayless TM. Carbohydrate absorption and malabsorption. Clin Gastroenterol 1983 May;12(2):335-56

Siegenbeek van Heukelom J. Physiological aspects of absorption and secretion in intestine. Vet Res Commun 1986 Sep;10(5):341- 4

INTESTINAL DISEASES

Chadwick VS. Small intestinal secretion in disease. Scand J Gastroenterol 1983:18(suppl 87):91-7 **

1

Chiapella AM. Treatment of intestinal disease. Vet Clin North Am [Small Anim Pract] 1983 Aug;13(3):567-84

Creamer B. The small intestine: a review. Pahlavi Med J 1978 Jan;9(1): 50-103

Goriup U, Shmerling DH. [Dietary therapy of intestinal diseases in childhood]. Ther Umsch 1978:35(8):673-8 **

lsaacs PET, Kim YS. Blind loop syndrome and small bowel bacterial contamination. Clin Gastroenterol 1983 May:12(2):395-414

MacIntyre PB. The short bowel. Br J Surg 1985 Sep; (suppl 72):S92-3

Mathan VI. Small intestine failure. <u>In:</u> Taylor TG, Jenkins NK, eds. Proceedings of the XIII International Congress of Nutrition. London: Libbey, 1986:671-4 **

Mathias JR, Clench MH. Review: pathophysiology of diarrhea caused by bacterial overgrowth of the small intestine. Am J Med Sci 1985 Jun;289(6): 243-8 **

Milla PJ. Intestinal motility and its disorders. Clin Gastroenterol 1986 Jan:15(1):121-36

Perman JA, Modler S. Role of the intestinal microflora in disposition of nutrient in the gastrointestinal tract. J Pediatr Gastroenterol Nutr 1983;2 (suppl 1):S193-6

Polin RA. Monoclonal antibodies against microorganisms. Eur J Clin Microbiol 1984 Oct;3(5):387-98

Rambaud J-C. Small intestinal lymphomas and alpha-chain disease. Clin Gastroenterol 1983 Sep:12(3):743-66

Read NW. The relationships between colonic motility and transport. Scand J Gastroenterol 1984;19(suppl 93):35-42 **

Read NW. Speculations on the role of motility in the pathogenesis and treatment of diarrhoea. Scand J Gastroenterol 1983;18(suppl 84):45-63 **

Simon GL. Gorbach SL. Intestinal microflora. Med Clin North Am 1982 May;66 (3):557-74

Simon GL, Gorbach SL. Intestinal flora in health and disease. Gastroenterology 1984 Jan;86(1):174-93 **

Taryan SR, Kagnoff MF, Brogan MD, Shanahan F. Immunologic mechanisms in intestinal diseases. Ann Intern Med 1987 Jun;106(6):853-70 **

Thompson JS, Rikkers LF. Surgical alternatives for the short bowel syndrome. Am J Gastroenterol 1987 Feb;82(2):97-106 **

Ziegler MM. Short bowel syndrome in infancy: etiology and management. Clin Perinatol 1986 Mar;13(1):163-73

INTESTINAL DISEASES, PARASITIC

Anderson RM. The population dynamics and epidemiology of intestinal nematode infections. Trans R Soc Trop Med Hyg 1986;80(5):686-96 **

Befus D. Immunity in intestinal helminth infections: present concepts, future directions. Trans R Soc Trop Med Hyg 1986:80(5):735-41 **

Brasitus TA. Parasites and malabsorption. Clin Gastroenterol 1983 May;12(2): 495-510 **

Cline BL. Current drug regimens for the treatment of intestinal helminth infections. Med Clin North Am 1982 May;66 (3):721-42

Cook GC. The clinical significance of gastrointestinal helminths - a review. Trans R Soc Trop Med Hyg 1986;80(5):675-85 **

Crompton DWT. Nutritional aspects of infection. Trans R Soc Trop Med Hyg 1986:80(5):697-705 **

Feachem RG, Guy MW, Harrison S, Iwugo KO, Marshall T, Mbere N, Muller R, Wright AM. Excreta disposal facilities and intestinal parasitism in urban Africa: preliminary studies in Botswana, Ghana and Zambia. Trans R Soc Trop Med Hyg 1983;77(4):515-21 **

Gilles HM. Progress in the pathogenesis and therapy of parasitic diseases. Scand J Infect Dis 1982;(suppl 36):12-4

Ozeretskovskaya NN. Intestinal parasitic infections. Scand J Infect Dis 1982;(suppi 36):46-51

Tanowitz HB, Weiss LM, Wittner M. Diagnosis and treatment of protozoan diarrheas. Am J Gastroenterol 1988 Apr:83(4):339-50

Wakelin D. Genetic and other constraints on resistance to infection with gastrointestinal nematodes. Trans R Soc Trop Med Hyg 1986;80(5):742-7 **

INTESTINAL SECRETIONS

Carpenter CCJ. The pathophysiology of secretory diarrheas. Med Clin North Am 1982 May;66 (3):597-610 **

Chadwick VS. Small intestinal secretion in disease. Scand J Gastroenterol 1983;18(suppl 87):91-7 **

Harries JT. Mechanisms and mediators of intestinal secretion in the small intestine. J Pediatr Gastroenterol Nutr 1982;1(4):575-82 **

Krejs GJ. VIPoma syndrome. Am J Med 1987 May 29;82(suppl 5B):37-48 **

Siegenbeek van Heukelom J. Physiological aspects of absorption and secretion in intestine. Vet Res Commun 1986 Sep;10(5):341-54

Turnberg LA. Antisecretory activity of opiates in vitro and in vivo in man. Scand J Gastroenterol 1983;18(suppl 84):79-83 **

IONS

Fondacaro JD. Intestinal ion transport and diarrheal disease. Am J Physiol 1986 Jan: 250(1. pt 1):G1-8 **

IRON

Burman D. Iron deficiency in infancy and childhood. Clin Haematol 1982 Jun; 11(2):339-51

Fettman MJ, Rollin RE. Antimicrobial alternatives for calf diarrhea: iron chelators or competitors. J Am Vet Med Assoc 1985 Oct 1:187(7):746-8

Finkelstein RA, Sciortino CV, McIntosh MA. Role of iron in microbe-host interactions. Rev Infect Dis 1983 Sep-Oct;5(suppl 4):S759-77

Neilands JB, Bindereif A, Montgomerie JZ. Genetic basis of iron assimilation in pathogenic Escherichia coli. Curr Top Microbiol Immunol 1985;118: 179-95

ISLET CELL TUMOR

Zallinger RM. Islet cell tumors of the pancreas and the alimentary tract. Am J Surg 1975 Feb;129(2):102-10 **

ISOSPORA BELLI

Ma P, Kaufman D. <u>Isospora belli</u> diarrheal infection in homosexual men. AIDS Res 1984;1(5):327-38 **

KNOWLEDGE, ATTITUDES AND PRACTICE

Green EC. Traditional healers, mothers and childhood diarrheal disease in Swaziland: the interface of anthropology and health education. Soc Sci Med 1985;20(3):277-85 **

Jelliffe EFP, Jelliffe DB, Feldon K, Ngokwey N. Traditional practices concerning feeding during and after diarrhoea (with special reference to acute dehydrating diarrhoea in young children). World Rev Nutr Diet 1987;53: 218-95

٠

LIVER ABSCESS, AMEBIC

Knight R. Hepatic amebiasis. Semin Liver Dis 1984 Nov;4(4):277-92

MALABSORPTION SYNDROMES

Balistreri WF, Heubi JE, Suchy FJ. Bile acid metabolism: relationship of bile acid malabsorption and diarrhea. J Pediatr Gastroenterol Nutr 1983:2(1):105-21

Booth CC. Diarrhoea due to intestinal malabsorption. Proc R Soc Med 1963 Dec;56(12):1058-70 **

Brasitus TA. Parasites and malabsorption. Clin Gastroenterol 1983 May;12(2): 495-510 **

Caspary WF. Diarrhoea associated with carbohydrate malabsorption. Clin Gastroenterol 1986 Jul;15(3):631-55

Cello JP. Inflammatory and malignant diseases of the small bowel causing malabsorption. Clin Gastroenterol 1983 May;12(2):511-32

Cvjetanovic B, Chen L, Kronmall R, Rohde C, Suskind R. Measuring and evaluating diarrhea and malabsorption in association with village water supply and sanitation: a review of the Food Wastage/Sanitation Cost Benefit Methodology Project (Guatemala). Arlington, Virginia: Water and Sanitation for Health Project, 1981. 36 p. (WASH technical report, 12)

da Rocha JM. [Disaccharidase disorders]. Bol Inst Puericult 1963 Apr;20(1): 311-22 **

Diarrhea and malabsorption associated with the acquired immunodeficiency syndrome (AIDS). Nutr Rev 1985 Aug;43(8):235-7

Doe WF, Hapel AJ. Intestinal immunity and malabsorption. Clin Gastroenterol 1983 May;12(2):415-35

Freeman HJ, Sleisenger MH, Kim YS. Human protein digestion and absorption: normal mechanisms and protein-energy malnutrition. Clin Gastroenterol 1983 May;12(2):357-78

Gracey M, Burke V. Sugar-induced diarrhoea in children. Arch Dis Child 1973 May;48(261):331-6 **

Greene HL. A pathophysiologic approach to dietary management in patients with protracted diarrhea and malnutrition. \underline{In} : Winter RW, Greene HL, eds. Nutritional support of the seriously ill patient. New York: Academic Press, 1983:181-94

Isaacs PET, Kim YS. Blind loop syndrome and small bowel bacterial contamination. Clin Gastroenterol 1983 May;12(2):395-414

Keusch GT, Solomons NW. Microorganisms, malabsorption, diarrhea and

dysnutrition. J Environ Pathol Toxicol Oncol 1985 Jul;5(6):165-209

McClain CJ. Zinc metabolism in malabsorption syndromes. J Am Coll Nutr 1985; 4(1):49-64 **

Mathan VI. Small intestine failure. <u>In</u>: Taylor TG, Jenkins NK, eds. Proceedings of the XIII International Congress of Nutrition. London: Libbey, 1986:671-4 **

Molla A, Molla AM, Sarker SA. Malabsorption in enteric infection; a nutritional cost in children with diarrhea. In: Chagas C, Keusch GT, eds. The interaction of parasitic diseases and nutrition, 22-26 Oct 1985. Vaticana: Pontificia Academia Scientiarum. 1985:71-80 **

Morris JA, Jr., Selivanov V, Sheldon GF. Nutritional management of patients with malabsorption syndrome. Clin Gastroenterol 1983 May:12(2):463-74

Owen RL, Brandborg LL. Mucosal histopathology of malabsorption. Clin Gastroenterol 1983 May:12(2):575-90

Perman JA, Modler S. Role of the intestinal microflora in disposition of nutrient in the gastrointestinal tract. J Pediatr Gastroenterol Nutr 1983;2 (suppl 1):S193-6

Ravich WJ, Bayless TM. Carbohydrate absorption and malabsorption. Clin Gastroenterol 1983 May:12(2):335-56

Rosenberg IH, Solomons NW, Schneider RE. Malabsorption associated with diarrhea and intestinal infections. Am J Clin Nutr 1977 Aug;30(8):1248-53 **

Ryan ME, Olsen WA. A diagnostic approach to malabsorption syndromes: a pathophysiological approach. Clin Gastroenterol 1983 May:12(2):533-50

Tasman-Jones C, Kay RG, Lee SP. Zinc and copper deficiency, with particular reference to parenteral nutrition. Surg Annu 1978;10:23-52 **

Wagh MG, Ghooi RB, Shetty RK. Lactose intolerance; physiological, clinical and therapeutic considerations. Indian J Pediatr 1984 Nov-Dec;51(413):671-81

MALIGNANT CARCINOID SYNDROME

Cello JP. Inflammatory and malignant diseases of the small bowel causing malabsorption. Clin Gastroenterol 1983 May;12(2):511-32

MEASLES

Axton JH. Measles and the state of nutrition. \$ Afr Med J 1979 Jan 27;55(4): 125-6 **

Feachem RG, Koblinsky MA. Interventions for the control of diarrhoeal diseases among young children: measles immunization. Bull WHO 1983;61(4): 641-52 **

Guillozet N. Measles in Africa: a deadly disease. Some personal comments. Clin Pediatr (Phila) 1979 Feb;18(2):95-100 **

Walsh JA. Selective primary health care: strategies for control of disease in the developing world. IV. Measles. Rev Infect Dis 1983 Mar-Apr;5(2):330-40 **

MEMBRANES

Eidels L, Proia RL, Hart DA. Membrane receptors for bacterial toxins. Microbiol Rev 1983 Dec;47(4):596-20

METABOLISM, INBORN ERRORS

Hitzig WH. Protean appearances of immunodeficiencies: syndromes and inborn errors involving other systems which express associated primary immunodeficiency. Birth Defects 1983;19(3):307-12 **

MICROSCOPY

Goldschmidt B. Microscopic stool-gazing, a guide to the cause and cure of chronic and recurrent diarrhoea in children. S Afr Med J 1966 Feb 26;40(9): 191-5 **

MILK

Behar M. The role of feeding and nutrition in the pathogeny and prevention of diarrheic processes. Bull Pan Am Health Organ 1975;9(1):1-9 **

May JT. Antimicrobial properties and microbial contaminants of breast milk - an update. Aust Paediatr J 1984;20:265-9 **

Sharp JCM. Infections associated with milk and dairy products in Europe and North America, 1980-85. Bull WHO 1987;65(3):397-406 **

MORBIDITY AND MORTALITY

Ashworth A. International differences in child mortality and the impact of malnutrition. Hum Nutr Clin Nutr 1982;36C(4):279-88 **

Ashworth A, Feachem RG. Interventions for the control of diarrhoeal diseases among young children: prevention of low birth weight. Bull WHO 1985;63(1): 165-84 **

Briscoe J, Feachem RG, Rahaman MM. Measuring the impact of water supply and sanitation facilities on diarrhoea morbidity: prospects for case-control

methods. Geneva: World Health Organization, 1985. 71 p. (WHO/CWS/85.3; CDD/OPR/85.1) **

Esrey SA, Feachem RG, Hughes JM. Interventions for the control of diarrhoeal diseases among young children: improving water supplies and excreta disposal facilities. Bull WHO 1985;63(4):757-72**

Feachem RG, Koblinsky MA. Interventions for the control of diarrhoeal diseases among young children: measles immunization. Bull WHO 1983;61(4): 641-52 **

Feachem RG, Koblinsky MA. Interventions for the control of diarrhoeal diseases among young children: promotion of breast-feeding. Bull WHO 1984;62(2): 271-91 **

Research on improving infant feeding practices to prevent diarrhoea or reduce its severity: Memorandum from a JHU/WHO meeting. Bull WHO 1989;67(1): 27-33 **

Ruzicka LT, Hansluwka H. Mortality transition in South and East Asia: technology confronts poverty. Pop Dev Rev 1982 Sep;8(3):567-88 **

Snyder JD, Merson MH. The magnitude of the global problem of acute diarrhoeal disease: a review of active surveillance data. Bull WHO 1982;60(4): 605-13 **

Sunoto. Diarrhoeal problems in Southeast Asia. Southeast Asian J Trop Med Public Health 1982 Sep;13(3):306-18 **

MYCOBACTERIUM INFECTIONS

Chiodini RJ. Crohn's disease and the mycobacterioses: a review and comparison of two disease entities. Clin Microbiol Rev 1989 Jan;2(1):90-117 **

NARCOTICS

Turnberg LA. Antisecretory activity of opiates in vitro and in vivo in man. Scand J Gastroenterol 1983;18(suppl 84):79-83 **

NEMATODE AND NEMATODE INFECTIONS

Anderson RM. The population dynamics and epidemiology of intestinal nematode infections. Trans R Soc Trop Med Hyg 1986;80(5):686-96 **

Pritchard DI. Antigens of gastrointestinal nematodes. Trans κ Soc Trop Med Hyg 1986;80(5):728-34 **

Wakelin D. Genetic and other constraints on resistance to infection with gastrointestinal nematodes. Trans R Soc Trop Med Hyg 1986;80(5):742-7 **

٠

NEOPLASMS

Randeria JD. Malnutrition and cancer. J Environ Pathol Toxicol Oncol 1985 Jul:5(6):103-14

NORMALK AGENT

Barnett B. Viral gastroenteritis. Med Clin North Am 1983 Sep;67(5):1031-58 **

Blacklow NR, Cukor G. Viral gastroenteritis. N Engl J Med 1981 Feb 12:304(7):397-406 **

Broor S, Singh V. Viral gastroenteritis. Indian J Gastroenterol 1984 Oct;3 (4):225-9 **

Butler TC. Viral diarrhoeas [editorial perspective]. J Diarrhoeal Dis Kes 1984 Sep:2(3):137-41

Steinhoff MC. Viruses and diarrhea--a review. Am J Dis Child 1978 Mar;132 (3):302-7 **

Wolf JL, Schreiber DS. Viral gastroenteritis. Med Clin North Am 1982 May;66 (3):575-95

MUTRITION. NUTRITION DISORDERS AND NUTRITIONAL REQUIREMENTS

Ashworth A. International differences in child mortality and the impact of malnutrition. Hum Nutr Clin Nutr 1982;36C(4):279-88 **

Axton JH. Measles and the state of nutrition. S Afr Med J 1979 Jan 27:55(4):125-6 **

Booth CC. Diarrhoea due to intestinal malabsorption. Proc R Soc Med 1963 Dec;56(12):1068-70 **

Calloway DH. Nutritional requirements in parasitic diseases. Rev Infect Dis 1982 Jul-Aug;4(4):891-5 **

Chandra RK, Greenough WB, III, Guerrant RL, Martorell R, Mata LJ, Warren KS, Wu C-C. Diarrhea and malnutrition: research priorities. In: Chen LC, Scrimshaw NS, eds. Diarrhea and malnutrition: interactions, mechanisms, and interventions. New York: Plenum, 1983:305-8

Chandra RK. • Nutritional regulation of immunity and infection in the gastrointestinal tract. J Pediatr Gastroenterol Nutr 1983;2(suppl 1):S181-7

Chen LC, Huq E, Huffman SL. A prospective study of the risk of diarrhoeal diseases according to the nutritional status of children. Am J Epidemiol 1981 Aug;114(2):284-92 **

Chernoff R. Dean JA. Medical and nutritional aspects of intractable diarrhea.

J Am Diet Assoc 1980 Feb;76(2):161-9 **

Crompton DWT. Nutritional aspects of infection. Trans R Soc Trop Med Hyg 1986;80(5):697-705 **

Diamond LS. Amebiasis: nutritional implications. Rev Infect Dis 1982 Jul-Aug;4(4):843-50 **

Dionigi R, Cremaschi RE, Jemos V, Dominioni L, Monico R. Nutritional assessment and severity of illness classification systems: a critical review on their clinical relevance. World J Surg 1986 Feb; 10(1):2-11**

Gardner FH. Nutritional management of chronic diarrhea in adults. JAMA 1962 Apr 14;180(2):147-52 **

Goriup U, Shmerling DH. [Dietary therapy of intestinal diseases in childhood]. Ther Umsch 1978;35(8):673-8 **

Gracey MS. Nutrition, bacteria and the gut. Br Med Bull 1981 Jan;37(1): 71-5 **

Greene HL. A pathophysiologic approach to dietary management in patients with protracted diarrhea and malnutrition. <u>In:</u> Winter RW, Greene HL, eds. Nutritional support of the seriously ill patient. New York: Academic Press, 1983:181-94

Hamilton JR. Treatment of acute diarrhea. Pediatr Clin North Am 1985 Apr; 32(2):419-27 **

Heim T. Requirements and utilization of macronutrients in enteral and parenteral nutrition in acute and chronic diarrhea. In: Lebenthal E, ed. Chronic diarrhea in children. New York: Raven Press, 1984:541-57 **

Kelts D, Jones E. Selected topics in therapeutic nutrition. Curr Probl Pediatr 1983 Mar;13(5):1-62

Keusch GT, Solomons NW. Microoryanisms, malabsorption, diarrhea and dysnutrition. J Environ Pathol Toxicol Oncol 1985 Jul;5(6):165-209

Loeb H, Mozin MJ. Prevention of chronic diarrhea: nutritional implications. J Pediatr Gastroenterol Nutr 1983;2(suppl 1):S328-34

Love AHG. Metabolic response to malnutrition: its relevance to enteral feeding. Gut 1986 Nov;27(suppl 1):9-13 **

Mathan VI. Small intestine failure. <u>In</u>: Taylor TG, Jenkins NK, eds. Proceedings of the XIII International Congress of Nutrition. London: Libbey, 1986:671-4 **

Molla A, Molla AM, Sarker SA, Khatoon M, Rahaman MM. Effects of acute diarrhea on absorption of macronutrients during disease and after recovery. \underline{In} : Chen LC, Scrimshaw NS, eds. Diarrhea and malnutrition: interactions, mechanisms, and interventions. New York: Plenum, 1983:143-54

Morris JA, Jr., Selivanov V, Sheldon GF. Nutritional management of patients

١

with malabsorption syndrome. Clin Gastroenterol 1983 May;12(2):463-74

The nutritional origin of cataracts. Nutr Rev 1984 Nov;42(11):377-9

Randeria JD. Malnutrition and cancer. J Environ Pathol Toxicol Uncol 1985 Jul;5(6):103-14

Reddy V. Interaction between malnutrition and diarrhoea with particular reference to pediatric practice. Indian J Gastroenterol 1985 Jul;4(3):183-6 **

Rosenberg IH, Bengoa JM, Sitrin MD. Nutritional aspects of inflammatory bowel disease. Annu Rev Nutr 1985;5:463-84

Russell RI. Intestinal adaptation to an elemental diet. Proc Nutr Soc 1985 Feb:44(1):87-93

Soutter VL, Kristidis P, Gruca MA, Gaskin KJ. Chronic undernutrition/growth retardation in cystic fibrosis. Clin Gastroenterol 1986 Jan;15(1):137-55

Walker-Smith JA. Nutritional management of acute gastroenteritis -- rehydration and realimentation. Hum Nutr Appl Nutr 1986:40A(suppl 1):39-43

ORAL REHYDRATION

Abed FH. Household teaching of ORT in rural Bangladesh. Assign Child 1983; (61/62):249-65 **

Abed FH. Household teaching of oral rehydration therapy in rural Bangladesh. J Indian Med Assoc 1987 Jul:85(7):205-9

Ahmad K, Jahan K, Huq I. Decontamination of drinking water by alum for the preparation of oral rehydration solution. Food Nutr Bull 1984 Jun;6(2):54-7 **

Ahmed HS, Molla AM. Rice-based oral rehydration. J Diarrhoeal Dis Res. 1987 Mar;5(1):1-6 **

Black RE. The prophylaxis and therapy of secretory diarrhea. Med Clin North Am 1982 May; 66(3):611-21

Booth IW, Levine MM, Harries JT. Oral rehydration therapy in acute diarrhoea in childhood. J Pediatr Gastroenterol Nutr 1984 Sep;3(4):491-9 **

Carpenter CCJ. Oral rehydration: is it as good as parenteral therapy? [editorial]. N Engl J Med 1982 May 6;306(18):1103-4 **

Chatterjee HN. Control of vomiting in cholera and oral replacement of fluid. Lancet 1953 Nov 21;2(6795):1063 **

Edelman R. Prevention and treatment of infectious diarrhoea: speculations on the next 10 years. Am J Med 1985 Jun 28;78(suppl 6B):99-106 **

Elliott EJ, Walker-Smith JA, Farthing MJG. The role of bicarbonate and base precursors in treatment of acute gastroenteritis. Arch Dis Child 1987 Jan;62 (1):91-5 **

Feachem RG. Preventing diarrhoea: what are the policy options? Health Policy Plann 1986;1(2):109-17 **

Ghatikar KN. Oral rehydration therapy--an overview. Q Mec Rev 1980 Oct;31 (4):1-12

Gopalan C. Oral rehydration therapy - the need for a proper perspective. Food Nutr Bull 1986 Sep;8(3):69-70 **

Greenough WB, III. Principles and prospects in the treatment of cholera and related dehydrating diarrheas. <u>In:</u> Ouchterlony O, Holmgren J, eds. Cholera and related diarrheas: molecular aspects of a global health problem. Basel: Karger, 1980:211-8 (43rd Nobel Symposium) **

Greenough WB, III. "Super ORT" [editorial]. J Diarrhoeal Dis Res 1983 Jun;1 (2):74-5 **

Hirschhorn N. The treatment of acute diarrhea in children: an historical and physiological perspective. Am J Clin Nutr 1980 Mar;33(3):637-63 **

Kinoti SN, Wasunna A, Turkish J, Gateere R, Desai M, Agwanda R, Juma R. A comparison of the efficacy of maize-based ORS and standard W.H.O. ORS in the treatment of acute childhood diarrhoea at Kenyatta National Hospital, Nairobi, Kenya: results of a pilot study. East Afr Med J 1986 Mar:63(3):168-74

Leung AKC, Darling P, Auclair C. Oral rehydration therapy: a review. J R Soc Health 1987:107(2):64-7 **

Levine MM, Clements ML, Black RE, Hughes TP, Tome FC. Oral rehydration with simple sugar/salt solutions as an alternative in rural areas when glucose/electrolyte solutions are unavailable. In: Holme T, Holmgren J, Merson MH, Mollby R, eds. Acute enteric infections in children: new prospects for treatment and prevention. Amsterdam: Elsevier, 1981:325-31 **

Mahalanabis D, Patra FC. In search of a super oral rehydration solution: can optimum use of organic solute-mediated sodium absorption lead to the development of an absorption promoting drug? J Diarrhoeal Dis Res 1983 Jun;1 (2):76-81 **

Mahalanabis D, Choudhuri AB, Bagchi NG, Bhattacharya AK, Simpson TW. Oral fluid therapy of cholera among Bangladesh refugees. Johns Hopkins Med J 1973 Apr; 132(4):197-205 **

Mahalanabis D, Merson MH, Barua D. Oral rehydration therapy - recent advances. World Health Forum 1981;2(2):245-9 **

Meeuwisse GW. High sugar worse than high sodium in oral rehydration solutions. Acta Paediatr Scand 1983 Mar;72(2):161-6 **

Molla AM, Sarker SA, Hossain M, Molla A, Greenough WB, III. Rice-powder electrolyte solution as oral therapy in diarrhoea due to <u>Vibrio cholerae</u> and <u>Escherichia coli</u>. Lancet 1982 Jun 12;1(8285):1317-9 **

Nalin DR. Oral replacement of water and electrolyte losses due to travellers' diarrhoea. Scand J Gastroenterol 1983;18(suppl 84):95-8 **

Rohde JE, Cash RA, Guerrant RL, Mahalanabis D, Molla AM, Valyasevi A. Therapeutic interventions in diarrhea. <u>In</u>: Chen LC, Scrimshaw NS, eds. Diarrhea and malnutrition: interactions, mechanisms, and interventions. New York: Plenum, 1983:287-95 **

Sack DA. Treatment of acute diarrhoea with oral rehydration solution. Drugs 1982 Jan-Feb;23(1-2):150-7 **

Sunoto. Oral rehydration salts: a simple and appropriate tool against dehydration due to diarrhoea. Paediatr Indones 1981 Mar-Apr;21(3-4):90-100 **

Tulloch J, Burton P. Global access to oral rehydration salts and use of oral rehydration therapy. World Health Stat Q 1987;40(2):110-5

PARASITES AND PARASITIC DISEASES

Baker RW, Peppercorn MA. Enteric diseases of homosexual men. Pharmacotherapy 1982 Jan-Feb;2(1):32-42 **

Brasitus TA. Parasites and malabsorption. Clin Gastroenterol 1983 May;12(2):495-510 **

Calloway DH. Nutritional requirements in parasitic diseases. Rev Infect Dis 1982 Jul-Aug;4(4):891-5 **

Feachem RG, Guy MW, Harrison S, Iwugo KO, Marshall T, Mbere N, Muller R, Wright AM. Excreta disposal facilities and intestinal parasitism in urban Africa: preliminary studies in Botswana, Ghana and Zambia. Trans R Soc Trop Med Hyg 1983;77(4):515-21 **

Gilles HM. Progress in the pathogenesis and therapy of parasitic diseases. Scand J Infect Dis 1982;(suppl 36):12-4

Gracey M. Antibiotic and antiparasitic therapy in chronic diarrhea. <u>In:</u> Lebenthal E, ed. Chronic diarrhea in children. New York: Raven Press, 1984:469-76

Judson FN. Sexually transmitted viral hepatitis and enteric pathogens. Urol Clin North Am 1984 Feb;11(1):177-85

Mahmoud AA. A physician's guide to the diagnosis of common parasitic infections. Med Clin North Am 1983 Jan;67(1):253-8

Mansour TE. Serotonin receptors in parasitic worms. Adv Parasitol 1984;23: 1-36

Mauel J. Mechanisms of survival of protozoan parasites in mononuclear phagocytes. Parasitology 1984 Aug;88(pt 4):579-92

Mitchell GF. Injection versus infection: the cellular immunology of parasitism. Parasitol Today 1987 Apr;3(4):106-11

Ozeretskovskaya NN. Intestinal parasitic infections. Scand J Infect Dis

99

1982;(suppl 36):46-51

Prichard RK. Interaction of host physiology and efficacy of antiparasitic drugs. Yet Parasitol 1985 Aug;18(2):103-10

Tanowitz HB, Weiss LM, Wittner M. Diagnosis and treatment of protozoan diarrheas. Am J Gastroenterol 1988 Apr;83(4):339-50

Tharavanij S. Pathogenesis of diarrhoea caused by parasites. Southeast Asian J Trop Med Public Health 1982 Sep;13(3):331-8 **

Wright SG. Parasites and travellers' diarrhoea. Scand J Gastroenterol 1983; 18(suppl 84):25-9 **

PARENTERAL HYPERALIMENTATION

Heim T. Requirements and utilization of macronutrients in enteral and parenteral nutrition in acute and chronic diarrhea. In: Lebenthal E, ed. Chronic diarrhea in children. New York: Raven Press, 1984:541-57 **

Lake AM, Kleinman RE, Walker WA. Enteric alimentation in specialized gastrointestinal problems: an alternative to total parenteral nutrition. Adv Pediatr 1981;28:319-39

McClain CJ. Trace metal abnormalities in adults during hyperalimentation. JPEN 1981 Sep-Oct;5(5):424-9 **

Muller JM, Keller HW, Brenner U, Walter M, Holzmuller W. Indications and effects of preoperative parenteral nutrition. World J Surg 1986 Feb;10(1): 53-63

Yip WC, Tay JS, Ho TF, Wong HB. Total parenteral nutrition in paediatric practice. J Singapore Paediatr Soc 1984;26(1-2):59-72

PEPTIDES

Auricchio S. Peptide digestion and absorption in the small intestinal mucosa during acute and chronic diarrhea. In: Lebenthal E, ed. Chronic diarrhea in children. New York: Raven Press, $19\overline{84}$:179-91

PLAGUE

Butler T. Plague and other <u>Yersinia</u> infections. New York: Plenum, 1983. 220 p.

PLASMIDS

Portnoy DA, Martinez RJ. Role of a plasmid in the pathogenicity of Yersinia

species. Curr Top Microbiol Immunol 1985;118:29-51

Sansonetti PJ, d'Hauteville H, Formal SB, Toucas M. Plasmid-mediated invasiveness of <Shigella-like> Escherichia coli. Ann Microbiol (Paris) 1982 May-Jun; 133(3):351-5 **

PLESIOMONAS

Holmberg SD, Farmer JJ, 3d. Aeromonas hydrophila and Plesiomonas shigelloides as causes of intestinal infections. Rev Infect Dis 1984 Sep-Oct;6(5):633-9 **

Ljungh A, Wadstrom T. Aeromonas and Plesiomonas as possible causes of diarrhoea. Infection $1985 \frac{\text{Aut-Aug}}{\text{Jul-Aug}}; 13(4): 169-73$ **

von Graevenitz A. <u>Aeromonas</u> and <u>Plesiomonas</u> as agents for diarrhea. <u>In:</u> Ellner PD, ed. Infectious diarrheal diseases; current concepts and laboratory procedures. New York: Marcel, 1984:59-75

von Graevenitz A. <u>Aeromonas</u> and <u>Plesiomonas</u>. <u>In: Lennette EH. Balows A, Hausler WJ, Jr., Truant JP, eds. <u>Manual of clinical microbiology</u>. 3d ed. Washington, D.C.: American Society for Microbiology, 1980:220-5</u>

von Graevenitz A, Mensch AH. The genus Aeromonas in human bacteriology: report of 30 cases and review of the literature. N Engl J Med 1968 Feb; 278(5): 245-9 **

Wadstrom T. Aeromonas and Plesiomonas as possible causes of diarrhoea. Third Meeting of the Scientific Working Group on Bacterial Enteric Infections, Geneva, 12-14 Sep 1984. Geneva: World Health Organization, 1984. 10 p. (Unpublished document)

PRIMARY HEALTH CARE

Arfaa F. Selective primary health care: strategies for control of disease in the developing world. XII. Ascariasis and trichuriasis. Rev Infect Dis 1984 May-Jun; 6(3):364-73 **

Briscoe J. Water supply and health in developing countries: selective primary health care revisited. Am J Public Health 1984 Sep:74(9):1009-13 **

Hornick RB. Selective primary health care: strategies for control of disease in the developing world. XX. Typhoid fever. Rev Infect Dis 1985 Jul-Aug;7(4):536-46 **

Keusch GT, Scrimshaw NS. Selective primary health care: strategies for control of disease in the developing world. XXIII. Control of infection to reduce the prevalence of infantile and childhood malnutrition. Rev Infect Dis 1986 Mar-Apr:8(2):273-87 **

Martinez-Palomo A, Martinez-Baez M. Selective primary health care: strategies for control of disease in the developing world. X. Amebiasis. Rev Infect Dis

1983 Nov-Dec;5(6):1093-1102 **

Rohde JE. Selective primary health care: strategies for control of disease in the developing world. XV. Acute diarrhea. Rev Infect Dis 1984 Nov-Dec;6(6): 840-54 **

Stevens DP. Selective primary health care: strategies for control of disease in the developing world. XIX. Giardiasis. Rev Infect Dis 1985 Jul-Aug;7(4): 530-5 **

Walsh JA, Warren KS. Selective primary health care: an interim strategy for disease control in developing countries. Soc Sci Med 1980;14C(2):145-63 **

Watsh JA. Selective primary health care: strategies for control of disease in the developing world. IV. Measles. Rev Infect Dis 1983 Mar-Apr;5(2):330-40 **

Warren KS. Selective primary health care: strategies for control of disease in the developing world. I. Schistosomiasis. Rev Infect Dis 1982 May-Jun;4 (3):715-26 **

PROSTAGLANDINS

Chang EB, Fedorak RN. Prostaglandins in diarrheal disease [editorial]. J Pediatr Gastroenterol Nutr 1985 Jun;4(3):341-4 **

Hawkey CJ, Rampton DS. Prostaglandins and the gastrointestinal mucosa: are they important in its function, disease, or treatment? Gastroenterology 1985 Nov:89(5):1162-88 **

Korman SH, Berant M, Alon U. Review: prostaglanding in diarrheal states. Isr J Med Sci $1981 \, \mathrm{Dec}; 17(12): 1109-13$

Metz SA, McRae JR, Robertson RP. Prostaglandins as mediators of parameoplastic syndromes: review and up-date. Metabolism 1981 Mar;30(3): 299-316

Rampton DS, Hawkey CJ. Prostaglandins and ulcerative colitis. Gut 1984 Dec; 25(12):1399-413

Rask-Madsen J, Bukhave K. Prostaglandins and chronic diarrhoea: clinical aspects. Scand J Gastroenteroi 1979;14(suppl 53):73-8 **

PROTEIN-CALORIE MALNUTRITION

Freeman HJ, Sleisenger MH, Kim YS. Human protein digestion and absorption: normal mechanisms and protein-energy malnutrition. Clin Gastroenterol 1983 May:12(2):357-78

Gracey M. Chronic diarrhoea in protein-energy malnutrition. Paediatr Indones 1981 Nov-Dec;21(11-12):235-9 **

Tontisirin K, Valyasevi A. Protein energy malnutrition related to diarrhea in

Thai children. J Nutr Sci Vitaminol (Tokyo) 1981:27(6):513-30 **

PROTEIN-LOSING ENTEROPATHY

Freeman HJ, Sleisenger MH, Kim YS. Human protein digestion and absorption: normal mechanisms and protein-energy malnutrition. Clin Gastroenterol 1983 May:12(2):357-78

Molla A, Molla AM, Sarker SA. Malabsorption in enteric infection; a nutritional cost in children with diarrhea. <u>In:</u> Chagas C, Keusch GT, eds. The interaction of parasitic diseases and nutrition, 22-26 Oct 1985. Vaticana: Pontificia Academia Scientiarum, 1985:71-80 **

Protein and fat losses in infants with prolonged diarrhea. Nutr Rev 1982 Nov; 40(11):335-7

Rahaman MM, Wahed MA. Direct nutrient loss in diarrhea. <u>In:</u> Chen LC, Scrimshaw NS, eds. Diarrhea and malnutrition: interactions, mechanisms, and interventions. New York: Plenum. 1983:155-60 **

PROTEINS

Freeman HJ, Sleisenger MH, Kim YS. Human protein digestion and absorption: normal mechanisms and protein-energy malnutrition. Clin Gastroenterol 1983 May;12(2):357-78

PROTOZOA AND PROTOZOAN INFECTIONS

DeGirolami PC, Dunn JC, Federman M. Infections caused by intestinal protozoa. Pathol Annu 1985:20(pt 2):463-505 **

Greenough WB, III. Protozoal, bacterial and viral diarrhoea: a common mechanism [editorial]. J Diarrhoeal Dis Res 1984 Jun;2(2):68

Intestinal protozoan and helminthic infections. WHO Tech Rep Ser 1981;(666):1-150

Sudo R, Aiba S. Role and function of protozoa in the biological treatment of polluted waters. Adv Biochem Eng Biotechnol 1984;29:117-41

Wolfe MS. The treatment of intestinal protozoan infections. Med €lin North Am 1982 May;56(3):707-20

PSEUDOMONAS

Kalina GP. [The genus Pseudomonas: new aspects of an old problem]. Zh

Mikrobiol Epidemiol Immunobiol 1985 May: (5):91-8

PUBLIC HEALTH

Briscoe J. Public health in rural India: the case of excreta disposal. Cambridge: Center for Population Studies, Harvard University, 1976. xix, 414 p. PhD Thesis. (Research paper, 12)

Briscoe J. The role of water supply in improving health in poor countries (with special reference to Bangladesh). Am J Clin Nutr 1978 Nov;31(11): 2100-13 **

Briscoe J. Water supply and health in developing countries: selective primary health care revisited. Am J Public Health 1984 Sep:74(9):1009-13 **

Cvjetanovic B. Health effects and impact of water supply and sanitation. World Health Stat 0.1986:39(1):105-17

Esrey SA, Habicht J-P. Epidemiologic evidence for health benefits from improved water and sanitation in developing countries. Epidemiol Rev 1986;8:117-28

Feachem RG. Infections related to water and excreta: the health dimension of the decade. In: Water supply and sanitation in developing countries. London: Institute of Water Engineers and Scientists, 1983:25-46

RECEPTORS

Eidels L. Proia RL, Hart DA. Membrane receptors for bacterial toxins. Microbiol Rev 1983 Dec;47(4):596-20

Mansour TE. Serotonin receptors in parasitic worms. Adv Parasitol 1984;23: 1-36

REITER'S DISEASE

Catterall RD. Clinical aspects of Reiter's disease. Br J Rheumatol 1983 Nov;22(4 suppl 2):151-5 **

Keat A. Reiter's syndrome and reactive arthritis in perspective. N Engl J Med 1983 Dec 29;309(26):1606-15

RISK FACTORS

Cantey JR. Infectious diarrhea. Pathogenesis and risk factors. Am J Med 1985 Jun 28;78(suppl 68):65-75 **

ROTAVIRUSES AND ROTAVIRUS INFECTIONS

Baqai R. Rotavirus gastroenteritis [editorial]. J Pak Med Assoc 1983 Oct; 33(10):240-2 **

Bohl EH. Rotaviral diarrhea in pigs: brief review. J Am Vet Med Assoc 1979 Mar 15:174(6):613-5 **

Flewett TH, Woode GN. The rotaviruses: brief review. Arch Virol 1978;57(1): 1-23 **

Hrdy DB. Epidemiology of rotaviral infection in adults. Rev Infect Dis 1987 May-Jun; 9(3):461-9

Kapikian AZ, Flores J, Hoshino Y, Glass RI, Midthun K, Gorziglia M, Chanock RM. Rotavirus: the major etiologic agent of severe infantile diarrhea may be controllable by a "Jennerian" approach to vaccination. J Infect Dis 1986 May:153(5):815-22

Konno T. [Viral gastroenteritis: serologiccal characteristics and molecular epidemiology of human rotavirus]. Rinsho Byori 1985 Feb;33(2):129-35

Kurstak E, Kurstak C, van Den Hurk J, Morisset R. Animal rotaviruses. <u>In:</u> Comparative diagnosis of viral diseases, v. 4. New York: Academic Press, 1981:105-48

Pickering LK. Rotaviruses infection. Pediatr Infect Dis 1985 May-Jun;4(suppl 3):S2-6 **

Saran M, Dabral M, Srivastava RN, Sharma VK. Epidemiology of human rotavirus diarrhoea -- a review. J Indian Assoc Commun Dis 1982 Sep-Dec;5(3-4):50-7 **

Steinhoff MC. Rotavirus: the first five years. J Pediatr 1980 Apr;96(4): 611-22 **

Torres-Medina A, Schlafer DH, Mebus CA. Rotaviral and coronaviral diarrhea. Vet Clin North Am (Food Anim Pract) 1985 Nov;1(3):471-93 **

Vesikari T. Progress in rotavirus vaccination. Pediatr Infect Dis 1985 Nov-Dec;4(6):612-4

SALMONELLA AND SALMONELLA INFECTIONS

D'Aoust J-Y. Recent developments in Salmonella epidemiology and methodology. Food Lab Newslett 1987 Apr; (9):32-6

Formal SB, Hale TL, Sansonetti PJ. Invasive enteric pathogens. Rev Infect Dis 1983 Sep-Oct;5(suppl 4):S702-7 **

Hornick RB. Selective primary health care: strategies for control of disease in the developing world. XX. Typnoid fever. Rev Infect Dis 1985 Jul-Aug;7(4):536-46 **

Mabilangan LM, Tiangco-Torres N. Focus on Salmonella enteritis. Asean J Clin Sci 1983 Dec; 4(4):392-4 **

Minette HP. Epidemiologic aspects of salmonellosis in reptiles, amphibians, mollusks and crustaceans - a review. Int J Zoonoses 1984 Jun;11(1):95-104

Moudgil KD, Narang BS. Pathogenesis of typhoid fever. Indian J Pediatr 1985 Jul-Aug:52(417):371-8 **

Murray BE. Resistance of <u>Shigelia</u>, <u>Salmonella</u>, and other selected enteric pathogens to antimicrobial agents. Rev Infect Dis 1986 May-Jun;8(suppl 2):S172-81 **

Sharp JCM. Infections associated with milk and dairy products in Europe and North America, 1980-85. Bull WHO 1987;65(3):397-406 **

Smith SM, Palumbo PE, Edelson PJ. <u>Salmonella</u> strains resistant to multiple antibiotics: therapeutic implications. <u>Pediatr Infect Dis 1984 Sep-Oct;3(5): 455-60</u>

Turnbull PCB. Food poisoning with special reference to <u>Salmonella</u> - its epidemiology, pathogenesis and control. Clin Gastroenterol 1979 Sep;8(3): 665-714

Wadstrom T, Baloda SB, Yuk YR. Cytotoxic and cytolytic proteins of enteropathogenic Escherichia coli and Salmonella: new concepts on possible role in intestinal colonization. Zentralol Bakteriol Mikrobiol Hyg [A] 1986;(suppl 15):153-60

SANITATION AND SEWAGE

Briscoe J, Feachem RG, kahaman MM. Measuring the impact of water supply and sanitation facilities on diarrhoea morbidity: prospects for case-control methods. Geneva: World Health Organization, 1985. 71 p. (WHO/CWS/85.3; CDD/OPR/85.1) **

Briscoe J. Public health in rural India: the case of excreta disposal. Cambridge: Center for Population Studies, Harvard University, 1976. xix, 414 p. PhD Thesis. (Research paper, 12)

Cvjetanovic B. Health effects and impact of water supply and sanitation. World Health Stat Q 1986; 39(1):105-17

Cvjetanovic B, Chen L, Kronmall R, Rohde C, Suskind R. Measuring and evaluating diarrhea and malabsorption in association with village water supply and sanitation: a review of the Food Wastage/Sanitation Cost Benefit Methodology Project (Guatemala). Arlington, Virginia: Water and Sanitation for Health Project, 1981. 36 p. (WASH technical report, 12)

Cyjetanovic 8. Sanitation versus immunization in control of enteric and diarrhoeal diseases. Prog Water Technol 1979;11(1-2):81-7 **

Derryberry M. Health education aspects of sanitation programmes in rural areas

and small communities. Buil WHO 1954;10(2):145-54 **

Esrey SA, Feachem RG, Hughes JM. Interventions for the control of diarrhoeal diseases among young children: improving water supplies and excreta disposal facilities. Bull WHO 1985:63(4):757-72**

Esrey SA, Habicht J-P. Epidemiologic evidence for health benefits from improved water and sanitation in developing countries. Epidemiol Rev 1986;8:117-28 **

Feachem R. Priorities for diarrhoeal disease control: water, excreta, behaviour and diarrhoea. Diarrhoea Dialogue 1981;(4):4-5

Feachem R, McGarry M, Mara D, eds. Water, wastes and health in hot climates. London: Wiley, 1977. xvi, 399 p.

Feachem RG, Guy MW, Harrison S, Iwugo KO, Marshall T, Mbere N, Muller R, Wright AM. Excreta disposal facilities and intestinal parasitism in urban Africa: preliminary studies in Botswana, Ghana and Zambia. Trans R Soc Trop Med Hyg 1983;77(4):515-21 **

Feachem RG. Infections related to water and excreta: the health dimension of the decade. In: Water supply and sanitation in developing countries. London: Institute of Water Engineers and Scientists, 1983:25-46

Feachem RG. Infectious disease related to water supply and excreta disposal facilities. AMBIO 1977;6(1):55-8

Feachem RG. The role of water supply and sanitation in reducing mortality in China, Costa Rica, Kerala State (India) and Sri Lanka. In: Halstead SB, Walsh JA, Warren KS, eds. Good health at low cost; proceedings of a conference, held at the Bellagio Conference Center, Italy, 29 Apr-3 May 1985:191-8

Feachem RG. Rural water and sanitation; community participation in appropriate water supply and sanitation technologies: the mythology for the decade. Proc R Soc Lond (B) 1980 Jul;209(1174):15-29 **

Feachem RG, Bradley DJ, Garelick H, Mara DD. Sanitation and disease: health aspects of excreta and wastewater management. New York: Wiley, 1983. 501 p. [World Bank studies in water supply and sanitation, 3]

The International Drinking Water Supply and Sanitation Decade. Review of regional and global data (as at 31 December 1983). WHO Offset Publ 1986; (92):1-30

Kawata K. Water and other environmental interventions--the minimum investment concept. Am J Clin Nutr 1978 Nov;31(11):2114-23 **

Kuo C. Measures to control diarrhoeal diseases--environmental sanitation. Regional Meeting on Cholera and Diarrhoeal Diseases, Alexandria, 1-5 Jun 1978. Alexandria: Regional Office for the Eastern Mediterranean, World Health Organization, 1978. 6 p. **

Lee EW. Safe water supply and sanitation in diarrhoeal diseases control. Regional Planning Meeting on Diarrhoeal Diseases Control, Manila, 5-7 Jun 1979.

Manila: Regional Office of the Western Pacific, World Health Organization, 1979. 5 p. (WPR/BVD/DDC/79.3) **

van Zijl WJ. Studies on diarrhoeal diseases in seven countries by the WHO Diarrhoeal Diseases Advisory Team. Bull WHO 1966;35(2):249-61 **

SCHISTOSOMIASIS

Warren KS. Selective primary health care: strategies for control of disease in the developing world. I. Schistosomiasis. Rev Infect Dis 1982 May-Jun;4 (3):715-26 **

SEROTYPING

Evans DJ, Jr., Evans DG. Classification of pathogenic <u>Escherichia coli</u> according to serotypes and the production of virulence factors, with special reference to colonization-factor antigens. Rev Infect Dis 1983 Sep-Oct;5 (suppl 4):S692-701 **

SEXUALLY TRANSMITTED DISEASES

Allason-Jones E, Mindel A. Sex and the bowel. Int J Color Dis 1987 Feb;2(1): 32-7

Baker RW, Peppercorn MA. Enteric diseases of homosexual men. Pharmacotherapy 1982 Jan-Feb; 2(1): 32-42 **

Judson FN. Sexually transmitted viral hepatitis and enteric pathogens. Urol Clin North Am 1984 Feb;11(1):177-85

Ma P, Kaufman D. <u>Isospora belli</u> diarrheal infection in homosexual men. AIDS Res 1984;1(5):327-38 **

SHIGELLA

Cantey JR. Shiga toxin--an expanding role in the pathogenesis of infectious diseases. J Infect Dis 1985 May;151(5):766-71 **

Formal SB, Hale TL, Sansonetti PJ. Invasive enteric pathogens. Rev Infect Dis 1983 Sep-Oct;5(suppl 4):S702-7 **

Hale TL, Formal SB. Genetics of virulence in Shigella. Microbial Pathogen 1986 Dec; 1(6):511-8 **

Hug MI, Ahmed QS, Rahaman MM. Changing pattern of antibiotic resistance in Shigella isolated in Bangladesh. $\underline{\text{In}}$: Rahaman MM, Greenough WB, III, Novak NR,

Rahman S, eds. Shigellosis: a continuing global problem; proceedings of an international conference, Cox's Bazaar, 1981. Dhaka: International Centre for Diarrhoeal Disease Research, Bangladesh, 1983:174-81 **

Keusch GT. Shigella infections. Clin Gastroenterol 1979 Sep;8(3):645-62

Keusch GT, Donohue-Rolfe A, Jacewicz M. <u>Shigella</u> toxin and the pathogenesis of shigellosis. Ciba Found Symp 1985;112:193-214 **

Keusch GT. Donohue-Rolfe A, Jacewicz M. Shigella toxin(s); description and role in diarrhea and dysentery. Pharmacol Ther 1982;15(3):403-38 **

Kopecko DJ, Baron LS, Buysse J. Genetic determinants of virulence in Shigella and dysenteric strains of Escherichia coli: their involvement in the pathogenesis of dysentery. Curr Top Microbiol Immunol 1985;118:71-95

Linde K. Stable, highly immunogenic mutants of <u>Salmoneila</u> with two independent, attenuating markers as potential live vaccine and their validity for Shigella and other bacteria. Dev Biol Stand 1983;53:15-28 **

Murray BE. Resistance of <u>Shigella</u>, <u>Salmonella</u>, and other selected enteric pathogens to antimicrobial agents. Rev Infect Dis 1986 May-Jun;8(suppl 2):S172-81 **

Nelson JD, Kusmiesz H, Shelton S. Oral or intravenous trimethoprimsulfamethoxazole therapy for shigellosis. Rev Infect Dis 1982 Mar-Apr;4(2): 546-50 **

O'Brien AD, Holmes RK. Shiga and Shiga-like toxins. Microbiol Rev 1987 Jun;51(2):206-20 **

Ranaman MM. Shigellosis: an old disease with new faces [editorial]. J Diarrhoeal Dis Res 1984 Dec;2(4):208

Zhalko-Titarenko VP, Bondarenko VM, Grigoryev AV, Kupchinsky LG, Rybalko SL. Dynamics of the interaction of <u>Shigellae</u> with the epithelium in the process of infection. Zh Mikrobiol Epidemiol Immunobiol 1986 Apr; (4):21-4

SHIGHLIA DYSENTERIAE

 0° Brien AD, Holmes RK. Shiga and Shiga-like toxins. Microbiol Rev 1987 Jun; 51(2):206-20 **

SHIGELLA FLEXNERI

Sansonetti PJ, d'Hauteville H, Formal SB, Toucas M. Plasmid-mediated invasiveness of <Shigella-like> Escherichia coli. Ann Microbiol (Paris) 1982 May-Jun;133(3):351-5 **

Simmons DAR, Romanowska E. Structure and biology of Shigella flexneri 0

antigens. J Med Microbiol 1987 Jun;23(4):289-302 **

SKIN DISEASES

Kupba R. Gastrointestinal manifestations of skin diseases: a review. Trop Gastroenterol 1983 Apr-Jun;4(2):67-78

Marks J. The relationship of gastrointestinal disease and the skin. Clin Gastroenterol 1983 Sep;12(3):693-712

SOCIOECONOMIC AND SOCIOCULTURAL FACTORS

Ellencweig AY, Slater PE. Demographic and socio-economic patterns of hospitalization for infectious diseases in Israel. Eur J Epidemiol 1986 Jun; 2(2):83-9 **

Green EC. Traditional healers, mothers and childhood diarrheal disease in Swaziland: the interface of anthropology and health education. Soc Sci Med 1985;20(3):277-85 **

Ruzicka LT, Hansluwka H. Mortality transition in South and East Asia: technology confronts poverty. Pop Dev Rev 1982 Sep;8(3):567-88 **

SPIROCHAETALES

Holt SC. Anatomy and chemistry of spirochetes. Microbiol Rev 1978 Mar;42(1): 114-60

SPLENIC DISEASES

Corazza GR, Gasbarrini G. Defective splenic function and its relation to bowel disease. Clin Gastroenterol 1983 Sep;12(3):651-69

STARCH

Jenkins DJA, Jenkins AL, Wolever TMS, Rao AV, Thompson LU. Fiber and starchy foods: gut function and implications in disease. Am J Gastroenterol 1986 0ct;81(10):920-30 **

SURGERY

Thompson JS, Rikkers LF. Surgical alternatives for the short bowel syndrome. Am J Gastroenterol 1987 Feb;82(2):97-106 **

THIOSULEATES

Barrett EL, Clark MA. Tetrathionate reduction and production of hydrogen sulfide from thiosulfate. Microbiol Rev 1987 Jun:51(2):192-205 **

TOROVIRIDAE

Horzinek MC, Flewett TH, Saif LJ, Spaan WJ, Weiss M, Woode GN. A new family of vertebrate viruses: Toroviridae. Intervirology 1987;27(1):17-24 **

TOXINS

Gyr K. Toxin receptors and their pathogenetic significance. Acta Histochem 1984 (suppl 29):S95-102 **

TRACE ELEMENTS

McClain CJ. Trace metal abnormalities in adults during hyperalimentation. JPEN 1981 Sep-Oct:5(5):424-9 **

TRICHOSTRONGYLOIDIASIS

Holmes PH. Pathogenesis of trichostrongylosis. Vet Parasitol 1985;18: 89-101 **

TRICHURIS AND TRICHURIASIS

Arfaa F. Selective primary health care: strategies for control of disease in the developing world. XII. Ascariasis and trichuriasis. Rev Infect Dis 1984 May-Jun;6(3):364-73 **

Bundy DAP. Epidemiological aspects of <u>Trichuris</u> and trichuriasis in Caribbean communities. Trans R Soc Trop Med Hyg <u>1986;80(5)</u>:706-18 **

TRIMETHOPRIM

Nelson JD, Kusmiesz H, Shelton S. Oral or intravenous trimethoprimsulfamethoxazole therapy for snigellosis. Rev Infect Dis 1982 Mar-Apr;4(2): 546-50 **

Sack RB. Antimicrobial prophylaxis of travellers' diarrhoea: a summary of studies using doxycycline or trimethoprim and sulphamethoxazole. Scand J

Gastroenterol 1983;18(suppl 84):111-7 **

TROPICAL CLIMATE AND DISEASES

Brown KR, Phillips SM. Tropical diseases of importance to the traveler. Adv Intern Med 1984:29:59-84

Feachem R, McGarry M, Mara D, eds. Water, wastes and health in hot climates. London: Wiley, 1977. xvi, 399 p.

Number T. Epidemiological aspect of viral diarrhea in tropical area. Jpn J Trop Med Hyg 1985 Mar: 13(1): 51

Pearson RD, Hewlett EL, Guerrant RL. Tropical diseases in North America. DM 1984 Mar;30(6):1-68

TYPHOID

ı

 \P^{-1}

Hornick RB. Selective primary health care: strategies for control of disease in the developing world. XX. Typhoid fever. Rev Infect Dis 1985 Jul-Aug;7(4):536-46 **

Levine MM, Kaper JB, Black RE, Ciements ML. New knowledge on pathogenesis of bacterial enteric infections as applied to vaccine development. Microbiol Rev $1983\ Dec; 47(4): 510-50$ **

Moudgil KD, Narang BS. Pathogenesis of typhoid fever. Indian J Pediatr 1985 Jul-Aug;52(417):371-8 **

Steffen R. Epidemiologic studies of travelers' diarrhea, severe gastrointestinal infections, and cholera. Rev Infect Dis 1986 May-Jun;8(suppl 2):S122-30 **

TYPHOID-PARATYPHOID VACCINES

Dougan G, Hormaeche CE, Maskell DJ. Live oral <u>Salmonella</u> vaccines: potential use of attenuated strains as carriers of heterologous antigens to the immune system. Parasite Immunol 1987 Mar;9(2):151-60 **

Germanier R. Oral vaccination against enteric bacterial infections: an overview. Infection 1984 Mar-Apr;12(2):138-42 **

Levine MM, Kaper JB, Black RE, Clements ML. New knowledge on pathogenesis of bacterial enteric infections as applied to vaccine development. Microbiol Rev $1983\ Dec; 47(4):510-50$ **

Linde K. Stable, highly immunogenic mutants of <u>Salmonella</u> with two independent, attenuating markers as potential live vaccine and their validity for <u>Shigella</u> and other bacteria. Dev Biol Stand 1983;53:15-28 **

VACCINES, VACCINATION, IMMUNIZATION AND VACCINE DEVELOPMENT

Ahmed ZU, Sack DA, Sarker MR, Haider K. Possible approaches to the development of a vaccine against shigellosis. <u>In</u>: Ahmed ZU, Choudhury N, eds. Proceedings of the International Seminar on Biotechnology and Genetic Engineering, Dhaka, 25-27 Jan 1986. Dhaka: Bangladesh Academy of Sciences, 1987:195-204 **

Alouf JE. [Anti-toxin vaccines]. Ann Inst Pasteur Microbiol 1985 Nov-Dec; 1368(3):309-21

Creese AL. Cost effectiveness of potential immunization interventions against diarrhoeal disease. Soc Sci Med 1986;23(3):231-40 **

Cvjetanovic B. Sanitation versus immunization in control of enteric and diarrhoeal diseases. Prog Water Technol 1979;11(1-2):81-7 **

Development of vaccines against shigellosis: Memorandum from a WHO meeting. Bull WHO 1987;65(1):17-25 **

Dougan G, Hormaeche CE, Maskell DJ. Live oral <u>Salmonella</u> vaccines: potential use of attenuated strains as carriers of heterologous antigens to the immune system. Parasite Immunol 1987 Mar;9(2):151-60 **

Feachem RG, Koblinsky MA. Interventions for the control of diarrhoeal diseases among young children: measles immunization. Bull WHO 1983;61(4): 641-52 **

Germanier R. Oral vaccination against enteric bacterial infections: an overview. Infection 1984 Mar-Apr;12(2):138-42 **

Hinman AR, Bart KJ, Orenstein WA. New vaccines. Int J Epidemiol 1985 Dec;14 (4):502-4

Kaper JB, Lockman HA, Baldini MM, Levine MM. Development of live oral cholera vaccine candidates through recombinant DNA techniques. <u>In: Kuwahara S, Pierce NF, eds. Advances in research on cholera and related diarrheas.</u> Tokyo: KTK Scientific Publishers. 1986:181-91

Kapikian AZ, Flores J, Hoshino Y, Glass RI, Midthun K, Gorziglia M, Chanock RM. Rotavirus: the major etiologic agent of severe infantile diarrhea may be controllable by a "Jennerian" approach to vaccination. J Infect Dis 1986 May;153(5):815-22 **

La Brooy J., Rowley D. Cholera vaccine - recent progress. <u>In:</u> Easmon CSF, Jeljasewicz, eds. Medical Microbiology. v. 2. London: Academic Press, 1983:157-76

Levine MM, Kaper JB, Black RE, Clements ML. New knowledge on pathogenesis of bacterial enteric infections as applied to vaccine development. Microbiol Rev 1983 Dec;47(4):510-50 **

Levine MM, Losonsky G, Herrington D, Kaper JB, Tacket C, Rennels MB, Morris JG. Pediatric diarrhea: the challenge of prevention. Pediatr Infect Dis 1986 Jan;5(suppl 1):S29-43 **

Levine MM, Black RE, Brinton CC, Jr., Clements ML, Fusco P, Hughes TP,

O'Donnell S, Robins-Browne R, Wood S, Young CR. Reactogenicity, immunogenicity, and efficacy studies of <u>Escherichia coli</u> type 1 somatic pili parenteral vaccine in man. Scand J Infect Dis 1982; (suppl 33):83-95

Levine MM. Travellers' diarrhoea: prospects for successful immunoprophylaxis. Scand J Gastroenterol 1983;18(suppl 84):121-34 **

Linde K. Stable, highly immunogenic mutants of <u>Salmonella</u> with two independent, attenuating markers as potential live vaccine and their validity for Shigella and other bacteria. Dev Biol Stand 1983;53:15-28 **

Manning PA. Involvement of cell envelope components in the pathogenesis of Vibrio cholerae: targets for cholera vaccine development. Vaccine 1987 Jun:5(2):83-7

Mitchell GF. Injection versus infection: the cellular immunology of parasitism. Parasitol Today 1987 Apr;3(4):106-11

Morger H, Steffen R, Schar M. Epidemiology of cholera in travellers, and conclusions for vaccination recommendations. Br Med J 1983 Jan 15;286(6360): 184-6 **

Porter P, Linggood MA. Development of oral vaccines for preventing diarrhoea caused by enteropathogenic Escherichia coli. J Infect 1983 Mar;6(2):111-21

Saif LJ, Smith KL. Enteric viral infections of calves and passive immunity. J Dairy Sci 1985 Jan;68(1):206-28 **

Sever JL. Infectious diseases and immunizations. Rev Infect Dis 1982 Jan-Feb;4(1):136-46

Summary of recommendations and conclusions: vaccines. <u>In: Priorities in biotechnology research for international development; proceedings of a workshop, Washington, D.C. and Berkeley Springs, 20-26 Jul 1982. Washington, D.C.: Board on Science and Technology for International Development Affairs, National Research Council, 1982:1-7</u>

Svennerholm A-M, Jertborn M, Gothefors L, Karim A, Sack D, Holmgren J. Secretory immunity to $\frac{\text{Vibrio}}{\text{vaccine.}}$ cholerae bacteria and cholera toxin: prospects for an improved cholera $\frac{\text{Vibrio}}{\text{Vaccine.}}$ Takeda Y, Miwatani T, eds. Bacterial diarrheal diseases. Tokyo: KTK Scientific Publishers, 1985:169-74

Vesikari T. Progress in rotavirus vaccination. Pediatr Infect Dis 1985 Nov-Dec:4(6):612-4

Walsh JA. Selective primary health care: strategies for control of disease in the developing world. IV. Measles. Rev Infect Dis 1983 Mar-Apr;5(2):330-40 **

Warren KS. New scientific opportunities and old obstacles in vaccine development. Proc Natl Acad Sci USA 1986 Dec;83(24):9275-7 **

VASOACTIVE INTESTINAL PEPTIDE

Gardner JD. Plasma VIP in patients with watery diarrhea syndrome. Am J Dig

ľ

1

Ļ

Dis 1978 Apr;23(4):370-6

Krejs GJ. VIPoma syndrome. Am J Med 1987 May 29;82(suppl 5B):37-48 **

VIBRIO AND VIBRIO INFECTIONS

Blake PA, Weaver RE, Hollis DG. Diseases of humans (other than cholera) caused by vibrios. Annu Rev Microbiol 1980:34:341-67 **

Butzler JP, Dekeyser P, Detrain M, Dehaen F. Related Vibrio in stools. J Pediatr 1973 Mar:82(3):493-5 **

Janda JM, Powers C, Bryant RG, Abbott SL. Current perspectives on the epidemiology and pathogenesis of clinically significant <u>Vibrio</u> spp. Clin Microbiol Rev 1988 Jul;1(3):245-67

Janda JM, Bryant RG. Pathogenic <u>Vibrio</u> spp: an organism group of increasing medical significance. Clin Microbiol Newslett 1987 Apr 1;9(7):49-56

Joseph SW, Colwell RR, Kaper JB. <u>Vibrio parahaemolyticus</u> and related halophilic vibrios. CRC Crit Rev Microbiol 1982;10(1):77-124

Rodrick GE, Hood MA, Blake NJ. Human <u>Vibrio</u> gastroenteritis. Med Clin North Am 1982 May;66 (3):665-73

Sanyal SC. NAG Vibrio toxin. Pharmacol Ther 1983;20(2):183-201

Tison DL, Kelly MT. Vibrio species of medical importance. Diagn Microbiol Infect Dis 1984 Sep; 2(4):263-76 **

VIBRIO CHOLERAE

Black RH. Invited discussion of Dr R M Glasse's paper. In: Proceedings of the Cholera Research Symposium, Honolulu, 24-29 Jan 1965. Washington, D.C.: U S Government Printing Office, 1965:340 **

Feachem R, Miller C, Drasar B. Environmental aspects of cholera epidemiology. II. Occurrence and survival of $\frac{\text{Vibrio cholerae}}{\text{cholerae}}$ in the environment. Trop Dis Bull 1981 Oct;78(10):865-80 **

Guidolin A, Manning PA. Genetics of Vibrio cholerae and its bacteriophages. Microbiol Rev 1987 Jun;51(2):285-98

Holmgren J. Pathogenesis and prevention of cholera. Scand J Infect Dis 1982; (suppl 36):58-64

Manning PA. Involvement of cell envelope components in the pathogenesis of Vibrio cholerae: targets for cholera vaccine development. Vaccine 1987 Jun;5(2):83-7

Monsur KA. How this happened? [editorial]. J Diarrhoeal Dis Res 1983 Mar;1 (1):3-4

Neter E. Enteropathogenicity: recent developments. Klin Wochenschr 1982 Jul 15:60(14):699-701 **

Seal SC. Centenary of discovery of cholera <u>Vibrio</u> [editorial]. Indian S Public Health 1983 Jan-Mar:27(1):1-4 **

Svennerholm A-M, Jertborn M, Gothefors L, Karim A, Sack D, Holmgren J. Secretory immunity to <u>Vibrio cholerae</u> bacteria and cholera toxin: prospects for an improved cholera vaccine. <u>In:</u> Takeda Y, Miwatani T, eds. Bacterial diarrheal diseases. Tokyo: KTK Scientific Publishers, 1985:169-74 **

Takeda Y. "Second century of <u>Vibrio</u> chôlerae" [editorial]. J Diarrhoeal Dis Res 1984 Mar;2(1):1-2

van Heyningen S. Cholera toxin: review. Biosci Rep 1982:2:135-46 **

VIBRIO PARAHAEMOLYTICUS

Joseph SW, Colwell RR, Kaper JB. <u>Vibrio parahaemolyticus</u> and related halophilic vibrios. CRC Crit Rev Microbiol 1982;10(1):77-124

VIRULENCE

Cornelis G, Laroche Y, Balligand G, Sory M-P, Wauters G. <u>Yersinia</u> enterocolitica, a primary model for bacterial invasiveness. Rev Infect Dis 1987 Jan-Feb; 9(1):64-87 **

Evans DJ, Jr., Evans DG. Classification of pathogenic <u>Escherichia coli</u> according to serotype and the production of virulence factors, with special reference to colonization-factor antigens. Rev Infect Dis 1983 Sep-Oct;5 (suppl 4):S692-701 **

Formal SB, Hale TL, Sansonetti PJ. Invasive enteric pathogens. Rev Infect Dis 1983 Sep-Oct;5(suppl 4):S702-7 **

Freter R. Jones GW. Models for studying the role of bacterial attachment in virulence and pathogenesis. Rev Infect Dis 1983 Sep-Oct;5(suppl 4):S647-58 **

Georgescu MB. [Colonization factors in enterotoxigenic <u>Escherichia coli</u>]. Rev Iq [Bacteriol] 1985 Apr-Jun:30(2):115-30

Hale TL, Formal SB. Genetics of virulence in Shigella. Microbial Pathogen 1986 Dec;1(6):511-8 **

Kopecko DJ, Baron LS. Buysse J. Genetic determinants of virulence in <u>Shigella</u> and dysenteric strains of <u>Escherichia coli</u>: their involvement in the pathogenesis of dysentery. Curr Top Microbiol Immunol 1985;118:71-95

0'Brien AD, Holmes RK. Shiga and Shiga-like toxins. Microbiol Rev 1987 Jun; 51(2):206-20 **

ď.

ì.

Sansonetti PJ, d'Hauteville H, Formal SB, Toucas M. Plasmid-mediated invasiveness of <Shigella-like> Escherichia coli. Ann Microbiol (Paris) 1982 May-Jun:133(3):351-5 **

Sparling PF. Bacterial virulence and pathogenesis: an overview. Rev Infect Dis 1983 Sep-Oct:5(suppl 4):S637-46 **

Wadstrom T, Baloda SB, Yuk YR. Cytotoxic and cytolytic proteins of enteropathogenic Escherichia coli and Salmonella: new concepts on possible role in intestinal colonization. Zentralbi Bakteriol Mikrobiol Hyg [A] 1986;(suppl 15):153-60

Wadstrom T, Baloda SB. Molecular aspects on small bowel colonization by enterotoxigenic Escherichia coli. Microecol Ther 1986;16:243-55

VIRUS AND VIRAL DISEASES

Banatvala JE. The role of viruses in acute diarrhoeal disease. Clin Gastroenterol 1979 Sep;8(3):569-98

Barnett B. Viral gastroenteritis. Med Clin North Am 1983 Sep;67(5):1031-58 **

Black RE. Viral diarrheas. <u>In</u>: Strickland GT, ed. Hunter's Tropical medicine. 6th ed. Philadelphia: Saunders, 1984:124-31

Blacklow NR, Cukor G. Viral gastroenteritis. N Engl J Med 1981 Feb 12;304(7):397-406 **

Broor S, Singh V. Viral gastroenteritis. Indian J Gastroenterol 1984 Oct;3 (4):225-9 **

Butler TC. Viral diarrhoeas [editorial perspective]. J Diarrhoeal Dis Res 1984 Sep;2(3):137-41

Chizhov NP. [Mechanising of the formation of viral resistance to chemotherapeutic preparations]. Vopr Virusol 1985 May-Jun;30(3):266-79

Christensen ML. Human viral gastroenteritis. Clin Microbiol Rev 1989 Jan;2(1):51-89 **

Cilli V, Castrucii G. Viral diarrhea of young animals: a review. Comp Immunol Microbiol Infect Dis 1981;4(3-4):229-42 **

Davidson GP. Viral diarrhoea. Clin Gastroenterol 1986 Jan:15(1):39-53 **

Greenough WB, III. Protozoal, bacterial and viral diarrhoea: a common mechanism [editorial]. J Diarrhoeal Dis Res 1984 Jun;2(2):68

Horzinek MC, Flewett TH, Saif LJ, Spaan WJ, Weiss M, Woode GN. A new family of vertebrate viruses: Toroviridae. Intervirology 1987;27(1):17-24 **

Konno T. [Viral gastroenteritis: serologiccal characteristics and molecular epidemiology of human rotavirus]. Rinsho Byori 1985 Feb;33(2):129-35

Lang W. Progress in the pathogenesis and therapy of viral and bacterial diseases. Scand J Infect Dis 1982; (suppl 36):7-11

Pollock RV, Carmichael L. Canine viral enteritis. Recent developments. Mod Vet Pract 1979 May;60(5):375-80

Ramia S. Transmission of viral infections by the water route: implications for developing countries. Rev Infect Dis 1985 Mar-Apr;7(2):180-8 **

Rapid laboratory techniques for the diagnosis of viral infections. WHO Tech Rep Ser 1981:(661):1-60

Saif LJ, Smith KL. Enteric viral infections of calves and passive immunity. J Dairy Sci 1985 Jan:68(1):206-28 **

Schmidt NJ. Rapid viral diagnosis. Med Clin North Am 1983 Sep;67(5):953-72 **

Shuval HI, Yekutiel P, Fattal B. An epidemiological model for the potential health risk associated with various pathogens in wastewater irrigation. Wat Sci Technol 1986;18(10):191-8 **

Shuval HI, Fattal B, Yekutiel P. State of the art review: an epidemiological approach to the health effects of wastewater reuse. Wat Sci Technol 1986;18 (9):147-62 **

Steinhoff MC. Viruses and diarrhea--a review. Am J Dis Child 1978 Mar;132 (3):302-7 **

Wolf JL, Schreiber DS. Viral gastroenteritis. Med Clin North Am 1982 May;66 (3):575-95

Yolken RH, Leggiadro RJ. Immunoassays for the diagnosis of viral enteric pathogens. Diagn Microbiol Infect Dis 1986 Mar;4(suppl 3):S61-9

VITAMIN A DEFICIENCY AND BLINDNESS/XEROPHTHALMIA

Feachem RG. Vitamin A deficiency and diarrhoea: a review of interrelationships and their implications for the control of xerophthalmia and diarrhoea. Trop Dis Bull 1987;84(3):R1-16 **

Pirie A. Vitamin A deficiency and child blindness in the developing world. Proc Nutr Soc 1983 Jan;42(1):53-64

WATER-ELECTROLYTE BALANCE AND IMBALANCE

Darrow DC, Pratt EL, Flett J, Jr., Gamble AH, Wiess HF. Disturbances of water and electrolytes in infantile diarrhea. Pediatrics 1949 Feb;3(2):129-56 **

Darrow DC, Pratt EL. Fluid therapy: relation to tissue composition and the expenditure of water and electrolyte. JAMA 1950 May 27;143(4):365-73

Darrow DC, Pratt EL. Fluid therapy: relation to tissue composition and the expenditure of water and electrolyte. JAMA 1950 Jun 3;143(5):432-9

Feld LG, Kaskel FJ, Schoeneman MJ. The approach to fluid and electrolyte therapy in pediatrics. Adv Pediatr 1988;35:497-536

Leung AKC, Darling P, Auclair C. Oral rehydration therapy: a review. J R Soc Health 1987;107(2):64-7 **

Milla PJ. Disorders of electrolyte absorption. Clin Gastroenterol 1982 Jan;11(1):31-46

Nalin DR. Oral replacement of water and electrolyte losses due to travellers' diarrhoea. Scand J Gastroenterol 1983;18(suppl 84):95-8 **

WATER POLLUTION

Craun GF. A summary of waterborne illness transmitted through contaminated groundwater. J Environ Health 1985 Nov-Dec:48(3):122-7 **

Feachem R, McGarry M, Mara D, eds. Water, wastes and health in hot climates. London: Wiley, 1977. xvi. 399 p.

Feachem RG. Infections related to water and excreta: the health dimension of the decade. In: Water supply and sanitation in developing countries. London: Institute of Water Engineers and Scientists, 1983:25-46

Feachem RG. Infectious disease related to water supply and excreta disposal facilities. AMBIO 1977;6(1):55-8

Feachem RG, Bradley DJ, Garelick H, Mara DD. Sanitation and disease: health aspects of excreta and wastewater management. New York: Wiley, 1983. 501 p. [World Bank studies in water supply and sanitation, 3]

Feachem RG, Burns E, Cairncross AM, Cronin A, Cross R, Curtis D, Khan MK, Lamb D, Southal H. Water, health and development: an interdisciplinary evaluation. London: Tri-Med Books, 1978. 267 p.

Pitlik S, Berger SA, Huminer D. Nonenteric infections acquired through contact with water. Rev Infect Dis 1987 Jan-Feb;9(1):54-63 **

Shuval HI, Yekutiel P, Fattal B. An epidemiological model for the potential health risk associated with various pathogens in wastewater irrigation. Wat Sci Technol 1986;18(10):191-8 **

Shuval HI, Fattal B, Yekutiel P. State of the art review: an epidemiological approach to the health effects of wastewater reuse. Wat Sci Technol 1986;18 (9):147-62 **

Sudo R, Aiba S. Role and function of protozoa in the biological treatment of polluted waters. Adv Biochem Eng Biotechnol 1984;29:117-41

WATER SUPPLY

Briscoe J. Intervention studies and the definition of dominant transmission routes. Am J Epidemiol 1984 Sep;120(3):449-55 **

Briscoe J, Feachem RG, Rahaman MM. Measuring the impact of water supply and sanitation facilities on diarrhoea morbidity: prospects for case-control methods. Geneva: World Health Organization, 1985. 71 p. (WHO/CWS/85.3; CDD/OPR/85.1) **

Briscoe J. The role of water supply in improving health in poor countries (with special reference to Bangladesh). Am J Clin Nutr 1978 Nov;31(11): 2100-13 **

Briscoe J. Water supply and health in developing countries: selective primary health care revisited. Am J Public Health 1984 Sep:74(9):1009-13 **

Cutting WAM, Hawkins P. The role of water in relation to diarrhoeal disease. J Trop Med Hyg 1982 Feb;85(1):31-9 **

Cvjetanovic B. Health effects and impact of water supply and sanitation. World Health Stat Q 1986; 39(1):105-17 **

Cvjetanovic B, Chen L, Kronmall R, Rohde C, Suskind R. Measuring and evaluating diarrhea and malabsorption in association with village water supply and sanitation: a review of the Food Wastage/Sanitation Cost Benefit Methodology Project (Guatemala). Arlington, Virginia: Water and Sanitation for Health Project, 1981. 36 p. (WASH technical report, 12)

Esrey SA, Habicht J-P. Epidemiologic evidence for health benefits from improved water and sanitation in developing countries. Epidemiol Rev 1986;8:i17-28 **

Esrey SA. Feachem RG, Hughes JM. Interventions for the control of diarrhoeal diseases among young children: improving water supplies and excreta disposal facilities. Bull WHO 1985;63(4):757-72**

Feachem R. Priorities for diarrhoeal disease control: water, excreta, behaviour and diarrhoea. Diarrhoea Dialogue 1981;(4):4-5

Feachem R, McGarry M, Mara D, eds. Water, wastes and health in hot climates. London: Wiley, 1977. xvi, 399 p.

Feachem RG, Guy MW, Harrison S, Iwugo KO, Marshall T, Mbere N, Muller R, Wright AM. Excreta disposal facilities and intestinal parasitism in urban Africa: preliminary studies in Botswana, Ghana and Zambia. Trans R Soc Trop Med Hyg 1983;77(4):515-21 **

Feachem RG. Infections related to water and excreta: the health dimension of the decade. In: Water supply and sanitation in developing countries. London: Institute of Water Engineers and Scientists, 1983:25-46

Feachem RG. Infectious disease related to water supply and excreta disposal

1

facilities. AMBIO 1977:6(1):55-8

Feachem RG. The role of water supply and sanitation in reducing mortality in China, Costa Rica, Kerala State (India) and Sri Lanka. <u>In</u>: Halstead SB, Walsh JA, Warren KS, eds. Good health at low cost; proceedings of a conference, held at the Bellagio Conference Center, Italy, 29 Apr-3 May 1985:191-8

Feachem RG. Rural water and sanitation; community participation in appropriate water supply and sanitation technologies: the mythology for the decade. Proc R Soc Lond (B) 1980 Jul; 209(1174):15-29 **

Feachem RG, Burns E, Cairncross AM, Cronin A, Cross R, Curtis D, Khan MK, Lamb D, Southal H. Water, health and development: an interdisciplinary evaluation. London: Tri-Med Books, 1978. 267 p.

Gibbs KR. There is no safe water in rural Banyladesh: so what about the kids? Shishu Diganta (Dhaka) 1980 Dec;(9):25-7 **

The International Drinking Water Supply and Sanitation Decade. Review of regional and global data (as at 31 December 1983). WHO Offset Publ 1986:(92):1-30

Kawata K. Water and other environmental interventions--the minimum investment concept. Am J Clin Nutr 1978 Nov:31(11):2114-23 **

Kuo C. Measures to control diarrhoeal diseases -- environmental sanitation. Regional Meeting on Cholera and Diarrhoeal Diseases, Alexandria, 1-5 Jun 1978. Alexandria: Regional Office for the Eastern Mediterranean, World Health Organization, 1978. 6 p. **

Lee EW. Safe water supply and sanitation in diarrhoeal diseases control. Regional Planning Meeting on Diarrhoeal Diseases Control, Manila, 5-7 Jun 1979. Manila: Regional Office of the Western Pacific, World Health Organization, 1979. 5 p. (WPR/BVD/DDC/79.3) **

Mosley WH, Khan MU. Cholera epidemiology - some environmental aspects. Prog Water Technol 1979;11(1-2):309-16 **

van Zijl WJ. Studies on diarrhoeal diseases in seven countries by the WHO Diarrhoeal Diseases Advisory Team. Bull WHO 1966;35(2):249-61 **

WEANING AND WEANING EDUCATION

Ashworth A, Feachem RG. Interventions for the control of diarrhoeal diseases among young children: weaning education. Bull WHO 1985;63(6):1115-27 **

Rowland MGM. The weamling's dilemma: are we making progress? Acta Paediatr Scand 1986;(suppl 323):33-42 **

Wharton BA. Food for weanling: the next priority in infant nutrition. Acta Paediatr Scand 1986;(suppl 323):96-102

YERSINIA AND YERSINIA INFECTIONS

Baqai R. <u>Yersinia</u> <u>enterocolitica</u> [editorial]. J Pak Med Assoc 1984 Aug; 34(8):232-4 **

Bottone EJ. <u>Yersinia enterocolitica</u>. <u>In: Ellner PD, ed. Infectious diarrheal diseases. New York: Dekker, 1984:13-48</u>

Bottone EJ, ed. <u>Yersinia enterocolitica</u>. Boca Raton, Florida: CRC Press, 1981. 224 p.

Butler T. Plague and other <u>Yersinia</u> infections. New York: Plenum, 1983. 220 p.

Cornelis G, Laroche Y, Balligand G, Sory M-P, Wauters G. <u>Yersinia</u> enterocolitica, a primary model for bacterial invasiveness. Rev Infect Dis 1987 Jan-Feb;9(1):64-87 **

Portnoy DA, Martinez RJ. Role of a plasmid in the pathogenicity of <u>Yersinia</u> species. Curr Top Microbiol Immunol 1985;118:29-51

Vantrappen G, Geboes K, Ponette E. <u>Yersinia</u> enteritis. Med Clin North Am 1982 May;66(3):639-53 **

Wu GX. [Progress of studies on yersiniosis caused by <u>Yersinia enterocolitica</u>]. Chung Hua Lin Hsing Ping Hsueh Tsa Chih 1984 Oct;5(5):309-13

ZINC

McClain CJ. Zinc metabolism in malabsorption syndromes. J Am Coll Nutr 1985; 4(1):49-64 **

Prasad AS. The role of zinc in gastrointestinal and liver disease. Clin Gastroenterol 1983 Sep;12(3):713-41

Tasman-Jones C, Kay RG, Lee SP. Zinc and copper deficiency, with particular reference to parenteral nutrition. Surg Annu 1978;10:23-52 **

ZOONOSES

Goscienski PJ. Zoonoses. Pediatr Infect Dis 1983 Jan;2(1):69-80

AUTHOR SECTION

Abbott SL see Janda JM

Abed FH. Household teaching of ORT in rural Bangladesh. Assign Child 1983; (61/62):249-65

The Bangladesh Rural Advancement Committee (BRAC), through its Oral Therapy Extension Programme, seeks to disseminate before 1990 to all rural Bangladesh households information about oral rehydration therapy. Specifically, the aim of a 3-year program, begun in 1980, is to teach mothers in 2.5 million households (14 million people) in 5 of 20 districts of Bangladesh, how to prepare oral rehydration solutions (ORS) with local ingredients. purpose, BRAC developed a health message called "seven points to remember". that explains how to prepare and use a "lobon-qur" ORS mixture (lobon-sodium chloride: qur=molasses. The resulting solution contains sucrose electrolytes, including potassium in approximately proper proportions). message also includes some relevant nutritional and other knowledge. here are the characteristics of the program, its evolution and organization, recruitment and training of workers, its built-in monitoring and evaluation system, and some available results. Preliminary findings indicate that the program is developing as hoped, and has attained maturity in terms of teaching quality. Illiterate Bangladeshi mothers can understand the health message, and prepare at home a safe, effective ORS, using household ingredients and finger measurement. While the program is vertical and has few links with other sectors, it provides valuable lessons in modes of community participation and in the methodology of face-to-face teaching of mothers.

Abed FH. Household teaching of oral rehydration therapy in rural Bangladesh. J Indian Med Assoc 1987 Jul;85(7):205-9

Acres SD. Enterotoxigenic Escherichia coli infections in newborn calves: a review. J Dairy Sci 1985 Jan:68(1):229-56

Escherichia coli cause two common diseases of newborn calves: septicemia, in which the bacteria invade the systemic circulation and internal organs, and enteric colibacillosis, in which the bacteria exert their pathogenic influence on the mucosa of the small intestine. This paper concentrates on the latter condition, caused by enterotoxigenic \underline{E} . \underline{coli} (ETEC). ETEC diarrhea occurs during the first few days of life. The \underline{E} . \underline{coli} that cause the disease possess special attributes of virulence that allow them to colonize the small intestine and produce an enterotoxin that causes hypersecretion of fluid into the intestinal lumen. These ETEC are shed into the environment by infected animals in the herd and are ingested by newborn calves soon after birth. There is some natural immunity to ETEC; however, it often fails to protect calves born and raised under modern husbandry conditions. Hence, methods have been developed to stimulate protective immunity by vaccination of the dam. The protective antibodies are transferred passively to calves through the colostrum.

Adegbola RA. Review: Bacterial adhesion and pathogenicity. Afr J Med Med Sci 1988 Jun:17(2):63-9

"Bacteria adhere to almost any surface via specific surface molecules of recognition through which a firm union is established for colonization of the host. Studies have shown that adhesion plays an important and critical early role in the pathogenesis of infectious diseases, and a series of adhesins have been well documented in a certain number of strains and species of bacteria of medical importance. Attempts have been made to interfere with, or prevent adhesion of harmful bacteria to the host tissue. using receptor analogues or bacterial adhesin-vaccines as prophylactic measures to protect recipients from specific bacterial diseases. Although much success has been reported from such procedures in laboratory animals and livestock. extensive clinical trials are required to assess the efficacy of such procedures in humans. However, reports from limited studies have shown some encouraging results. Future studies must also be directed to the isolation and characterization of more adhesins and receptors and their interactions, which would provide fuller understanding of mechanisms of bacterial adhesion, especially at molecular level." (Author's abstract)

Aggarwal P, Misra BS, Singh J, Basu RN. Review of cholera in Delhi: a 14 year study. J Commun Dis 1986 Mar;18(1):17-21

Trends in the incidence of cholera in Delhi, India were studied for a 14-year period (1972-1985). Frequency of culture positivity varied from 13 to 23%, highest being in 1983. Predominance of Ogawa serotype was observed throughout the study period. All isolates of <u>Vibrio</u> Ol belonged to biotype El Tor. than 10% of the total cases occurred in infants, except in the year 1977, and more than 50% cases in the children aged under 15. No case of cholera was detected amongst meonates. All other age groups and both sexes were affected similarly. Vibrio cholerae in Delhi was seasonal and the increase in cases was dependent on an increase in rainfall, temperature and relative humidity. Thus, high seasonal incidences were observed in July and August. Persistence level calculated for the years showed a gradual rise from 43.6% in 1976 to 51.2% in 1985. Minimum monthly incidence rate was always zero for the years studied. These results place Delhi in an area of intermediate endemicity. Majority of cholera cases in this study came from the trans-Jamuna area of Delhi which has the highest slum population comprised of laborers who live in poor housing without sewage facilities.

Agwanda R see Kinoti SN

Ahlbom A see Persson PG

Ahmad K, Jahan K, Huq I. Decontamination of drinking water by alum for the preparation of oral rehydration solution. Food Nutr Bull 1984 Jun;6(2):54-7

This is the first report on the use of chemical agents in decontaminating potable water for the preparation of oral rehydration solution (ORS). The study determines whether aluminium potassium sulphate (potash alum), used traditionally for purifying tank, reservoir and household drinking water, would have an antibacterial effect on the total bacterial count in ORS prepared with the piped-supply water, and with pond water of high bacterial count. Vibrio cholerae at concentrations 10^3 and 10^4 per ml was killed between 1 and 2 h in $500~\mu g/ml$ potash alum. Potash alum at a concentration of 1 mg/ml killed V. cholerae in water in less than 1 h. Escherichia coli from stool (10^3 and 10^4 per ml) had the same survival time in presence of $500~\mu g/ml$ of potash alum. The pH of ORS fortified with $500~\mu g/ml$ alum remained at 6.4. ORS made from

well water or pond water, with or without potash alum, did not vary markedly in ionic concentration. Changes in NaHCO $_3$ level were within allowable limits. There were sharp decreases in the total bacterial counts in ORS made with water collected from different sources and when fortified with 500 μ g/ml of potash alum. Since diarrheal diseases are often caused by contaminated water, treating water before drinking or making ORS with potash alum during epidemics in rural or urban areas should decrease diarrheal morbidity and mortality.

Ahmed HS, Molla AM. Rice-based oral rehydration. J Diarrhoeal Dis Res 1987 Mar:5(1):1-6

This work is an exhaustive report on research findings on rice-based oral rehydration. The review covers studies carried out from 1949 to the present. The gradual development of an oral rehydration solution (ORS), constituted with rice through continuous research done worldwide, has led to the formulation of a more perfect super ORS which is cheaper, more effective and can be prepared It was found that glucose is with readily available home ingredients. expensive and not easily available in many less-developed countries where diarrhea is common. The use of molasses instead of glucose was tried in the preparation of ORS in Bangladesh. This solution, which can be easily prepared at home, was found effective as well as tasty. Research has continued to simplify the ORS formula, reduce its costs, and enhance nourishment during diarrhea. The use of chicken soup with glucose or cereal starch has been suggested by some scientists for treating diarrheal dehydration. The ORS with rice water (left-over water after boiling rice) was found to be more effective in reducing stool output than the glucose-based ORS. These findings have stimulated a vigorous search for new and better ORS based on rice and other dietary staples. In rice ORS, rice powder is used instead of glucose. The starch found in rice consists of polymers of glucose. Rice contains 7-10% protein. Rice ORS is safe, and no adverse effects have yet been detected. Several clinical and field trials have been done with rice ORS, and it was found to be more effective than other forms of ORS in reducing stool output, ORS intake, vomiting, and duration of diarrhea. Rice ORS provides extra nutrition for malnourished patients. Less supervision by nursing staff and physicians is required while giving the rice ORS to hospital patients. Young children can digest and absorb this solution. With this ORS, it has been observed that bacteria may grow quickly if the prepared solutions remain unused for a long time in a warm environment. At the ICDDR, B, it was found that rice ORS, once prepared, remains good for use at ambient temperature for 8 to 12 h. The rice-based ORS is still undergoing clinical and field trials, although in certain places, this formulation is already in use in diarrhea treatment centers. Further simplification of its formula is necessary so that people can make it easily at home and preserve it for a longer time after its preparation.

Ahmed QS see Huq MI

Ahmed QS see Khan MU

Ahmed ZU, Sack DA, Sarker MR, Haider K. Possible approaches to the development of a vaccine against shigellosis. <u>In</u>: Ahmed ZU, Choudhury N, eds. Proceedings of the International Seminar on Biotechnology and Genetic Engineering, Dhaka, 25-27 Jan 1986. Dhaka: Bangladesh Academy of Sciences, 1987:195-204

Current knowledge of the pathobiology of <u>Shigellae</u> and the molecular mechanism involved in the development of the disease is inadequate. That mucosal

immunity and intestinal IgA may be of considerable importance in protecting against intestinal infections has generated interest in orally administered live vaccines that could adequately stimulate intestinal immunity and provide protection. Such a vaccine can be developed by genetic modification of the pathogen by removing its pathogenicity while retaining its protective potential, or by transferring the pathogen's protective potential to a harmless carrier bacterium. These can be approached by using the techniques of either conventional genetics or recombinant deoxyribonucleic acid (DNA) methods. Neither of these methods has, at the present time, a significant margin of advantage in the development of a suitable live vaccine. It is, therefore, worthwhile to pursue the promising recombinant DNA approach but also that of conventional genetics. Although currently going through a phase of relative disinterest, the latter remains a productive route to microbial strain construction. A promising approach to developing potential vaccine strains of Snigella would be to isolate attenuated mutants. An attenuation method that has not yet been reported to have been applied to Shigellae involves the selection of two independent genetic blocks which will cause impaired cell proliferation in the gut. Isolation of such strains and testing their vaccine potential are considered worthwhile and are likely to aid significantly to the development of a live vaccine against shigellosis. (Modified authors' abstract)

Aiba S see Sudo R

Akin EW, Jakubowski W. Drinking water transmission of giardiasis in the United States. Wat Sci Technol 1986;18(10):219-26

"For about a century after Giardia was named by Lambl in 1859, the occurrence of the organism in humans was not widely considered significant in explaining the etiology of disease. This organism is now recognized as a cause of disease that ranges from mild-to-severe and debilitating gastroenteritis. Beginning in 1965, drinking water became increasingly implicated as an important route of transmission. An outbreak occurred in a Colorado ski resort in that year. A survey of visitors to the resort revealed that 123 persons experienced acute enteritis, possibly giardiasis. Sewage contamination of the well-water supply was thought to be the source of the organism. Further evidence for waterborne transmission of Giardia came in 1975 with the first isolation of the organism from a water supply implicated in an outbreak. A cyst was isolated from the water supply during an extended outbreak that resulted in 359 confirmed cases over a 7-month period. Subsequent outbreaks have clearly established the role of water in Giardia transmission. From 1965 through 1984, 90 waterborne outbreaks with 23,776 cases of giardiasis were reported in the United States. This report discusses the etiologic agent and examines drinking water outbreak occurrence and control." (Authors' abstract)

Albert MJ. Enteric adenoviruses: brief review. Arch Virol 1986;88(1-2):1-17

In 1978, the World Health Organization initiated a program for global prevention and control of childhood diarrheas. As a result, the relative importance of various pathogens in the etiology of diarrhea in many parts of the world has been recognized. Rotavirus ranks as the most prevalent viral pathogen in childhood diarrhea. After a decade of intense research since its discovery, many vaccines for its control are in sight. Several studies have now established enteric adenoviruses as definite pathogens causing diarrhea. They probably rank as the second most important viral pathogen. Structural

study of enteric adenovirus strains has shown an icosahedron capsid with a diameter of 70-80 nm and fibers 28-33 nm long, which are typical of established adenoviruses. The growth characteristics, taxonomy, genome profiles, cloning and mapping of genomes are discussed. No systematic study of the antigenic structure of enteric adenovirus has so far been undertaken. However, some information about their antigenic structure can be obtained serological methods used to identify them, and by analogy with established adenoviruses. As a group, adenoviruses possess several structural proteins which can be resolved by SDS-polyacrylamide gel electrophoresis. The leading symptom of enteric adenovirus infection is diarrhea. It may be associated with fever, vomiting, or respiratory problems. The disease is usually mild, but fatal cases have also been reported. Enteric adenoviruses are excreted in large quantities of up to 10"-particles per gram of stool at the acute stage of the disease, suggesting that they multiply actively in the gastrointestinal tract, most likely in the small intestinal mucosa. The epidemiology of the disease, including seroepidemiology, seasonal variation, outbreaks, and mode of spread, are described, and detection methods are outlined. Type-specific enzyme-linked immunosorbent assay has been recommended as the most suitable method for the diagnosis of enteric adenovirus-associated gastroenteritis.

Alexandrescu M. Coman G. Ene L. Manuca M. [Human infection with Campylobacter jejuni/coli]. Rev Ig [Bacteriol] 1984 Jul-Sep;29(3):245-57

Allason-Jones E, Mindel A. Sex and the bowel. Int J Color Dis 1987 Feb;2(1): 32-7

Alon U see Korman SH

Alouf JE. [Anti-toxin vaccines]. Ann Inst Pasteur Microbiól 1985 Nov-Dec; 136B(3):309-21

Altman DF. Gastrointestinal cryptosporidiosis and cytomegalovirus enterocolitis. Front Radial Ther Oncol 1985;19:88-90

Anderson RM. The population dynamics and epidemiology of intestinal nematode infections. Trans R Soc Trop Med Hyg 1986;80(5):686-96

"The paper reviews recent studies on the population biology, transmission dynamics and epidemiology of intestinal nematode infections. Particular attention is given to patterns of change in average intensity of infection with age, the possible role of acquired immunity assessed via reinfection studies after chemotherapy, evidence of predisposition to heavy (or light) infection by single and multiple species of parasites and control by mass, selective and targetted chemotherapy." (Author's abstract)

Andres JM. Advances in understanding the pathogenesis of persistent diarrhea in young children. Adv Pediatr 1988;35:483-98

Angus KW. Cryptosporidiosis in man, domestic animals and birds: a review. J R Soc Med $1983 \, \mathrm{Jan}; 76:62-9$

Cryptosporidia are coccidian parasites which can infect many species of mammals, birds, and reptiles. Their small size and moderate excretion rate make them difficult to detect in feces or tissue sections. Under experimental conditions, the life cycle in specific pathogen-free lambs, infected on the

first day of life, may be as short as 72 h, a period substantially shorter than that of other enteric coccidia. This capability of Cryptosporidium has important enidemiological implications. A random survey showed that sera from 10 mammalian species, including man, contained detectable antibodies to the organism. Transmission experiments show that Cryptosporidium can cause diarrhea and severe pathological lesions in the intestines of calves. and piglets. The organism has been associated with outbreaks of diarrhea in calves, lambs, red deer, and goats in the absence of other known enteropathogens, and may represent a serious complication of viral diarrheas in Cryptosporidia have also been associated with outbreaks of these species. respiratory disease in some species of diarrhea or serious Demonstration of occysts in methanol-fixed smears of feces stained either by Giemsa's method or by a modified Ziehl-Neelsen technique is now used routinely in diagnosis of cryptosporidiosis in farm livestock. Diagnosis of acute but transient cases of cryptosporidiosis in man has been made by demonstration of oocysts in stool samples by stained smears or by flotation, and confirmed by transmission to mice. Attempts at treatment or prophylaxis of cryptosporidial infections with anticoccidial or other drugs have proved unsuccessful, and the infective stages in the feces have been shown to be highly resistant to the action of a disinfectant. The economic significance of cryptosporidiosis in livestock is possibly considerable. A pathogenic coccidian capable of crossing species barriers, with a short-life cycle, which permits rapid build-up of infection in enclosed premises, constitutes a potential hazard for susceptible human individuals. The importance of cryptosporidiosis as a grave complication in immunocompromised patients cannot be over-emphasized.

Arasu TS, Wyllie R, Fitzgerald JF. Chronic diarrhea in infants and children. Am Fam Physician 1979 Apr;19(4):87-94

"A useful clinical classification of chronic diarrhea is based on the character of the stool - watery, fatty, or bloody. Pathophysiologic mechanisms include osmotic and secretory diarrheas, bacterial overgrowth leading to excess colonic bile acids and fatty acids, defective anion exchange systems, mucosal damage and abnormal motility and transit. Evaluation may require hospitalization. Antidiarrheal agents are not used. Antibiotics are generally not indicated. Parenteral hydration and feedings of special formulas are carefully monitored." (Authors' abstract)

Arbo A, Santos JI. Diarrheal diseases in the immunocompromised host. Pediatr Infect Dis 1987 Uct:6(10):894-906

Arfaa F. Selective primary health care: strategies for control of disease in the developing world. XII. Ascariasis and trichuriasis. Rev Infect Dis 1984 May-Jun;6(3):364-73

"Ascariasis and trichuriasis are the most prevalent and widespread intestinal helminthiases. Transmission of disease occurs by ingestion or inhalation of Ascaris and Trichuris eggs embryonated in the soil. During the migration of Ascaris to the lungs, pneumonic symptoms may develop. The intestinal stage of the parasite can cause severe symptoms and complications when the worm burden is high. High numbers of Trichuris may cause diarrhea. The poor standard of hygiene, the biology of the parasite (which is such that a high number of very resistant eggs are produced), and the habits of the host (such as the use of night soil) are factors causing a high prevalence of infection in many countries in Asia, Africa, South America, and Europe. The mechanism of the

transmission of <u>Ascaris</u> varies in different communities. The peak of infection is among children aged four to 14 years. Worm burden is normally low, and only a small segment of the population harbors a high proportion of the worms present in a community. Control of infections due to <u>Ascaris</u> and <u>Trichuris</u> is feasible by a combination of mass chemotherapy with the effective drugs now available, safe disposal of excreta, destruction of the eggs, and health education." (Author's abstract)

Armstrony M. Cryptosporidiosis. Med Lab Sci 1987 Jul;44(3):280-4

In 1907, a previously unrecognized coccidian parasite was found in the stomach of the common mouse and was named Cryptosporidium muris, and subsequently a further species, C. parvum, was identified in the small intestine of the same host. The parasite remained undetected in man for almost 70 years, and by 1981, only 7 cases of human cryptosporidiosis had been described. In the last 5 years, there have been over 200 publications describing its detection, identification, incidence and occurrence in both man and animals. Cryptosporidium occurs in a wide variety of host species. The parasite is a newly recognized cause of human gastroenteritis affecting both immunocompetent and immunocompromised individuals. Symptoms are often protracted, lasting for up to 14 days, and may include diarrhea, vomiting, fever, nausea, anorexia, abdominal cramps, lassitude, and subsequent significant weight loss. have shown a higher incidence of infection in children, especially those in the developing countries. Immunosuppressive therapy for leukemia and other life-threatening illnesses has been reported as contributing factors for prolonging the course of cryptosporidial enteritis. Until 1980, the diagnosis of human cryptosporidiosis was by histological demonstration of the parasite in close association with the microvillus border in jejunal and ileal biopsy sections. Confirmation bу electron microscopy characteristic showed morphology. Rapid, sensitive and simple diagnostic tests have now been The resistance of cryptosporidial oocysts to drugs environmental conditions has been reported. Antimicrobial agents and anticoccidial drugs have been shown to be ineffective in preventing in experimental animals. cryptosporidial infection The incidence of cryptosporidiosis reported from the UK, and around the world, clearly demonstrates that the parasite is a frequent cause of enteritis. cryptosporidiosis was considered as а zoonotic Cryptosporidium is now generally accepted as a member of the group of pathogenic organisms responsible for causing travelers' diarrhea. The seasonal pattern of cryptosporidiosis is difficult to establish. Reports from some developing countries describe a higher frequency during the rainy months.

Ashworth A. International differences in child mortality and the impact of malnutrition. Hum Nutr Clin Nutr 1982;36C(4):279-88

In the industrialized countries, 1-4-year mortality continues to decline even though levels are already low, in most cases being below one per 1,000. Rates of decline in child and infant mortalities are similar. The main causes of child death are accidents and congenital anomalies which are difficult to control. Indications are that 1-4-year mortality rates in the majority of less-developed countries are still very high. Limited data suggest that 1-4-year mortality is declining more rapidly than infant mortality and that rates of decline are faster than in industrialized countries. Diarrheal disease and measles are the main causes of 1-4-year mortality with malnutrition being a contributory cause in 60% of the cases. The majority of child deaths

occur in the second year of life. Health planning and evaluation, particularly in Africa and Asia, are severely hampered by totally inadequate death registration data. (Modified author's abstract)

Ashworth A. International differences in infant mortality and the impact of malnutrition; a review. Hum Nutr Clin Nutr 1982;36C(1):7-23

"In the industrialized countries infant mortality continues to decline even though mortality is already at a low level. The greatest number of infant deaths occur in the neonatal period, especially during the first day of life and the main determinants of infant death are perinatal causes and congenital anomalies. In less-developed countries infant mortality remains high, particularly in Africa, the Indian subcontinent and South-East Asia. Observed rates of decline during the last 20 years have been no faster than in the industrialized countries and may indeed have been slower. Although a greater proportion of infant deaths occur postneonatally compared with industrialized countries, the majority nevertheless occur within the first 3 months of life. The main causes of infant mortality are low birth-weight and diarrhoeal diseases. The implications in terms of health and nutrition policies are discussed." (Author's abstract)

Ashworth A, Feachem RG. Interventions for the control of diarrhoeal diseases among young children: prevention of low birth weight. Bull WHO 1985;63(1):165-84

The effects of low-birth weight on diarrheal morbidity and mortality are analyzed, and interventions to increase birth weights are reviewed. authors have located no satisfactory data on low-birth weight as a determinant of diarrheal mortality or morbidity. The strong association between low-birth weight and mortality, however, makes it likely that there is an association between low-birth weight and diarrheal mortality in developing countries where diarrhea is a major cause of infant deaths. Poor maternal nutrition, certain infections, pre-eclampsia, arduous work after mid-pregnancy, short-birth intervals, and teenage pregnancy are likely to be casually associated with low-birth weight in developing countries. Of the interventions examined, maternal food supplementation has been the most studied. If targeted to mothers at nutritional risk, and if the food is consumed in addition to the usual diet, the prevalence of low-birth weight can be expected to be reduced. However, food supplementation can be expensive, and the results from carefully supervised feeding trials may be better than those that can be achieved in national programs. The effect of supplementation with iron, zinc, or folate requires further study. If it were possible to intervene in maternal nutrition, health and life-style in a developing country in a way that reduced the prevalence of low-birth weight from around 30% to around 15%, a fall in the infant mortality rate of around 26% would be expected. The fall in infant diarrheal mortality rate might be similar. The scarce data on relative risk of morbidity by birth weight do not allow any comparable computations for morbidity reductions to be made. This review confirms that whatever its association with diarrhea, low-birth weight is an important determinant of infant mortality. For the more general goal of reducing infant mortality it is necessary to know more about the nature, etiology, and prevention of low-birth weight in developing countries. (Modified authors' abstract)

Ashworth A, Feachem RG. Interventions for the control of diarrhoeal diseases among young children: weaning education. Bull WHO 1985;63(6):1115-27

"A review of data from 12 developing countries suggests that it is possible, even in poor communities, to improve substantially the nutritional status of infants and young children by weaning education. Face-to-face communication by locally recruited workers, reinforced by radio and other mass media, may be the most effective channels for weaning education. It is estimated that, through its effect on nutritional status, weaning education may reduce the diarrhoea mortality rate among children under b years of age by 2-12%. The possible impacts of weaning education on food hygiene and on feeding during and after diarrhoeal illness are not considered in this paper. Preliminary estimates of cost-effectiveness suggest that weaning education may be an economically attractive intervention for reducing diarrhoea mortality in some countries. Several important aspects of weaning education and its relation to diarrhoea need to be clarified by research." (Authors' abstract)

Atkinson M, Hosking DJ. Gastrointestinal complications of <u>Diabetes</u> <u>mellitus</u>. Clin Gastroenterol 1983 Sep;12(3):633-60

Auclair C see Leung AKC

Auricchio S, Cucchiara S, D'Antonio AM, De Ritis G, De Vizia B, Follo D, Iaccarino E. Gastrointestinal allergy or intolerance to multiple foods in severe chronic diarrhea in early infancy. In: Lebenthal E, ed. Chronic diarrhea in children. New York: Raven Press, 1984:425-34

Auricchio S. Gluten-sensitive enteropathy and infant nutrition. J Pediatr Gastroenterol Nutr 1983;2(suppl 1):S304-9

Auricchio S. Peptide digestion and absorption in the small intestinal mucosa during acute and chronic diarrhea. <u>In</u>: Lebenthal E, ed. Chronic diarrhea in children. New York: Raven Press, 1984:179-91

Awouters F, Niemegeers CJE, Janssen PAJ. Pharmacology of antidiarrheal drugs. Annu Rev Pharmacol Toxicol 1983;23:279-301

A pharmacological study of potential antidiarrheal drugs is based on the measurement of inhibition of diarrhea in laboratory animals. The castor test in rats has been used for detecting and comparing the antidiarrheal activity of compounds. In this test, morphine-like antidiarrheals, and also various drugs of other pharmacological classes showed changes in their excretory pattern induced by castor oil. Most drugs were active in the castor oil test at a dose that exceeded the effective dose for their characteristic drugs, Anticholinergics, aspirin-like pharmacological action. clonidine-like drugs, which had pronounced intestinal action, had low antidiarrheal specificity. Loperamide was found to have high antidiarrheal specificity. Loperamide and its metabolites are confined to an enterohepatic circuit in the organism. The local action of loperamide is partially mediated by opiate receptors and results in inhibition of both excessive propulsive and secretory activity of the intestine. Loperamide was compared with several other antidiarrheal preparations in terms of their efficacy and modes of action in the gastrointestinal tract. The superiority of loperamide in controlling diarrhea over a range of other opiate derivatives is shown.

Axon AT. Functional diarrhoea. Int J Color Dis 1986 Jan:1(1):49-53

Axton JH. Measles and the state of nutrition. S Afr Med J 1979 Jan 27;55(4): 125-6

"The severity of an attack of measles is largely determined by the underlying state of nutrition at the time of the attack. Evidence is presented which suggest that, conversely, measles may be responsible for the precipitation of malnutrition in undernounished children, by a combination of several different mechanisms." (Author's abstract)

Aziz KMS see Huq I

Aziz KMS see Samadi AR

Bachman BA see Boyd WP. Jr.

Bachmann BJ. Linkage map of <u>Escherichia</u> <u>coli</u> K-12, edition 7. Microbiol Rev 1983 Jun:47(2):180-230

Bagchi NG see Mahalanabis D

Baker RW, Peppercorn MA. Enteric diseases of homosexual men. Pharmacotherapy 1982 Jan-Feb;2(1):32-42

Certain enteric ailments—are particularly common—among homosexual men. They are primarily infectious—diseases and—include not only—such common—venereal diseases as gonorrhea and syphilis but also infections not usually regarded as being sexually transmitted. Among the latter are snigellosis, salmonellosis, giardiasis, and amebiasis. Patients' symptoms are nonspecific and seldom helpful in diagnosing particular diseases. Gonorrhea is probably the most common bacterial infection in gay men. Carriage rates as high as 50% have been reported, and extragenital carriage is common; this necessitates culturing the urethra, rectum, and pharynx. In contrast to other venereal diseases, syphilis may have a characteristic proctoscopic presentation. Lymphogranuloma venereum causes penile lesions and inquinal lymphadenitis in heterosexual men, whereas homosexual men are more prone to proctitis. The disease may mimic Cronn's Recommended disease. treatment includes. tetracycline trimethoprim-sulfamethoxazole. Shigellosis usually presents as an diarrheal illness. Patients generally require only supportive treatment with fluids. Herpes simplex viral infection is difficult to diagnose and has several different presentations, including lumbosacral radiculomyelopathy. Symptomatic treatment with sitz-baths, anesthetic ointment, and analgesics is recommended. Venereal warts are believed to be caused by the same virus that causes verrucous warts; they are usually found in the anal canal or around the anal orifice. They are commonly treated with 25% podophyllin solution. Parasitic infections include giardiasis, amebiasis, and pinworm infections. Metronidazole may be used in the treatment of symptomatic giardiasis and amebiasis, but it is not approved for the former indication; quinacrine is approved for giardiasis. Pinworm infestation may be treated with pyrantel pamoate or mebendazole. Cure of enteric diseases in homosexual men must be documented, (Modified authors' abstract)

Baldini MM see Kaper JB

Balistreri WF, Heubi JE, Suchy FJ. Bile acid metabolism: relationship of bile acid malabsorption and diarrhea. J Pediatr Gastroenterol Nutr 1983;2(1):105-21

Balligand G see Cornelis G

Baloda SB see Wadstrom T

Banatvala JE. The role of viruses in acute diarrhoeal disease. Clin Gastroenterol 1979 Sep;8(3):569-98

Banerjee H see Sen PC

Banwell JG. Treatment of travelers' diarrhea: fluid and dietary management. Rev Infect Dis 1986 May-Jun;8(suppl 2):S182-7

Travelers' diarrhea is associated with a mild or moderately severe loss of fluid and electrolytes. Severe fluid deficits are encountered only rarely. Mortality associated with fluid deficits is very rare; significant morbidity occurs only in older adults or in patients with chronic intestinal diseases or other chronic diseases (cardiac, pulmonary, or renal). Treatment of fluid and electrolyte deficits may be effectively achieved by rehydration with oral rehydration solution (WHO) or with a commercial solution of similar composition. Dietary recommendations should emphasize the necessity resuming a normal diet once a reduction in symptoms makes this feasible. small proportions frequently and omitting caffeine lactose-containing food and drink may be advantageous. (Modified author's abstract)

Baqai R. Rotavirus gastroenteritis [editorial]. J Pak Med Assoc 1983 Oct; 33(10):240-2

This editorial discusses the importance of rotavirus as an etiological agent of diarrhea. It includes the morphology, physiopathology, incidences in different age groups, immunology, laboratory detection and management of rotavirus infection. The author suggests that rapid diagnostic methods should be available; and that development of an effective vaccine could reduce morbidity and mortality due to rotavirus in infants and children.

Baqai R. <u>Yersinia enterocolitica</u> (editorial). J Pak Med Assoc 1984 Aug; 34(8):232-4

 $\frac{\text{Yersinia}}{\text{outlines}} \stackrel{\text{enterocolitica}}{\text{the clinical}} \text{ is a newly recognized enteropathogen.} \quad \text{This editorial outlines} \\ \frac{\text{the clinical}}{\text{the clinical}} \text{ pattern, complications, transmission, diagnosis and treatment of } \\ \frac{\text{Yersinia}}{\text{treatment of organism}} \text{ infections.} \\ \text{Since } \frac{\text{Y. enterocolitica}}{\text{treatment of organism}} \text{ of diarrhea} \\ \text{from many countries, diarrheal stools from children and adults in Pakistan should be examined for this organism.} \\ \\ \text{This editorial complete of the clinical pattern, complications, transmission, diagnosis and treatment of the clinical pattern, complications, transmission, diagnosis and treatment of the clinical pattern, complications, transmission, diagnosis and treatment of the clinical pattern, complications, transmission, diagnosis and treatment of the clinical pattern, complications, transmission, diagnosis and treatment of the clinical pattern, complications, transmission, diagnosis and treatment of the clinical pattern, complications, transmission, diagnosis and treatment of the clinical pattern, complications, transmission, diagnosis and treatment of the clinical pattern, complications, transmission, diagnosis and treatment of the clinical pattern, complications, transmission, diagnosis and treatment of transmission pattern, complications, transmission, diagnosis and treatment of transmission pattern, complications, transmission pattern, complications, transmission pattern, complications, transmission pattern, complications, complications, transmission pattern, complications, com$

Barness LA. Chronic diarrhea in children. Postgrad Med 1979 Feb;65(2):163-6, 168-9

Pathogenic and clinical features of chronic diarrhea in children are described. Several mechanisms have been implicated as causes of diarrhea. Chronic diarrhea results in malnutrition, malabsorption and other complications. Less common causes include inflammatory responses, enzyme deficiencies, metabolic, anatomic and physiologic abnormalities, and endocrine disorders. Ingestion of excess calories may also cause diarrhea. The use of antibiotics sometimes causes an inflammatory response in the bowel. Escherichia coli strains are often responsible for diarrhea through production of enterotoxins. Alterations in prostaglandins or acetyl choline levels may cause chronic diarrhea, while parasites like Giardia may also cause diarrhea. Chronic diarrhea accompanies immunoglobulin deficiency. The recommended hydrating solution consists of 75 to 90 mmol/l of sodium chloride administered parenterally with 40 mmol/l of

potassium in case of dehydration due to diarrhea. Oral replacement of fluids can be increased gradually. Severe chronic diarrhea may require hyperalimentation with parenteral aminoacids, sugar, and fat. Nonspecific treatment includes a diet of clear liquids with elimination of milk, cereals, and vegetables. Medications containing atropine or morphine should be avoided. Diagnosis of the underlying cause of chronic diarrhea is desirable before definite treatment is begun, and physical examinations should be done carefully. Stools should be examined, and sweat tests should be made. Various laboratory tests should be carried out to rule out cystic fibrosis, anatomic and inflammatory abnormalities, and celiac diseases. Treatment should be directed to the underlying disease.

Barnett B. Viral gastroenteritis. Med Clin North Am 1983 Sep;67(5):1031-58

Rotaviruses and Norwalk-like viruses are the two major groups of viruses associated with gastroenteritis. This paper reviews the properties of these viruses, their epidemiology, clinical manifestations, laboratory diagnosis, immunology, and treatment of infections with rotavirus and Norwalk-like virus. A brief account of miscellaneous viruses associated with gastroenteritis (adenovirus, astrovirus, enterovirus, coronavirus) is also Rotaviral infections are seasonal, occurring mainly in the winter months, and are the major cause of viral gastroenteritis in infants and children. Diarrhea, vomiting, and fever are the most common clinical features. gastroenteritis may lead to severe dehydration. Norwalk-like viruses are a major cause of explosive outbreaks of acute gastroenteritis in older children and adults. These outbreaks are frequently associated with a vehicle, such as culinary water, raw vegetables, or raw seafood. Long-term resistance to Norwalk-like viral infections appears to be mediated by nonantibody factors. Other than rotaviruses, adenoviruses are the most frequently observed viruses in stools from children with gastroenteritis. Other symptoms of enteric adenovirus-associated gastroenteritis may be upper respiratory problems, fever, and vomiting. Several outbreaks of gastroenteritis in infants, children, and adults have been associated with astroviruses. While enteroviruses do not appear to play a major epidemiologic role in gastroenteritis, enteroviral disease is frequently severe in infants. Coronaviruses have been detected in the feces of persons with gastroenteritis. The gastroenteritis viruses are generally fastidious, and thus traditional cell culture isolation and detection procedures are not applicable. Electron microscopy and immune-electron microscopy remain among the most efficient techniques. The enzyme-linked immunosorbent assay is currently the method of choice for rotavirus detection. On the other hand, a radioimmunoassay for the Norwalk virus and its antibodies has been a powerful tool for epidemiological studies. The cultivation of these viral agents in vitro will facilitate the development of diagnostic reagents and the development and evaluation of vaccines. Most cases of viral gastroenteritis are self-limiting and can be effectively treated by rehydration therapy.

Baron LS see Kopecko DJ

Barrett EL, Clark MA. Tetrathionate reduction and production of hydrogen sulfide from thiosulfate. Microbiol Rev 1987 Jun;51(2):192-205

This paper analyzes the literature concerning tetrathionate and thiosulfate reductions by nonsulfate-reducing bacteria and provide a foundation for future studies of these processes. A brief summary of these reductions as performed

by the sulfate reducers is included. The distribution of the ability to reduce tetrathionate or thiosulfate has been examined, and reasons for the many discrepancies among reports of these abilities are discussed. Studies on dissimilatory tetrathionate and thiosulfate reductions as performed by members of the Enterobacteriaceae are reviewed with a focus on the biochemistry. regulation, and genetics of the associated electron transport systems. reported results include evidence for and against the hypothesis that these Assimilatory reductions constitute pathways of anaerobic respiration. thiosulfate reduction has been examined in the context of possible parallels with the dissimilatory reactions. Reports of plasmid-mediated transfer of the ability to reduce thiosulfate to hydrogen sulfide among the Enterobacteriaceae Findings regarding anaerobic tetrathionate are summarized briefly. thiosulfate reductions by thiosulfate-oxidizing bacteria and by Pseudomonas aeruginosa are summarized with a focus on similarities with comparable processes in the Enterobacteriaceae. The collective results presented in this review suggest that the ability to reduce tetrathionate and thiosulfate is widespread, but these reductions as performed by nonsulfate reducers, sulfate reducers are fundamentally different. (Modified authors' abstract)

Bart KJ see Hinman AR

Bartlett AV see Pickering LK

Bartlett JG. Antibiotic-associated colitis. DM 1984 Dec;30(15):1-54

Bartlett JG see Quinn TC

Barua D see Manalanabis D

Basu RN see Aggarwal P

Bayless TM see Ravich WJ

Befus AD see Elson CO

Befus D. Immunity in intestinal helminth infections: present concepts, future directions. Trans R Soc Trop Med Hyg 1986;80(5):735-41

"Although intestinal helminth and protozoan infections are prevalent throughout the world, their impact is poorly known. Nevertheless, the morbidity and mortality that occur have stimulated research into host resistance and pathogenesis. Unfortunately, despite an increasing knowledge base, the actual effector molecules which lead to parasite loss are unknown. IgA antibody, intraepithelial leukocytes and mucosal mast cells are thought to be involved. The role of these and other responses in making the intestinal microenvironment hostile to the parasite through effects of neurotransmission, epithelial cell differentiation and function, smooth muscle activities, and local inflammatory responses must be studied. Future research will include: further definition of immunologic repertoire of the and molecular the cellular identification of local effector molecules and investigation of interactions between antigen-specific immune responses and intestinal physiology. of human infections will be more restricted but must include: assessment of parasite-specific local immunologic responses, inflammatory events in the intestine, and development of relatively non-invasive techniques to study gastrointestinal physiology during parasitic infection. Perhaps most

importantly, research facilities must be established in developing countries to investigate intestinal immunological, inflammatory and physiological responses during infection. Through such investigations, risk factors for susceptibility and disease severity may be identified and therapeutic or prophylactic strategies developed." (Author's abstract):

Behar M. The role of feeding and nutrition in the pathogeny and prevention of diarrheic processes. Bull Pan Am Health Organ 1975;9(1):1-9

The combined interaction of diarrhea and inadequate nutrition poses a grave health problem for millions of children in Latin America. This paper explores this interaction by examining two of its main components: the protection against diarrhea conferred by breast feeding and the increased vulnerability to diarrhea created by malnutrition. Breast feeding helps prevent enteric infections in several ways. The mother's colostrum and milk contain antibodies against some enterobacterial antigens. The presence in human milk of a substance which favors the development of Bifidobacterium helps discourage growth of pathogenic enterobacteria in the intestinal lumen. Further, children living in unhealthy surroundings become heavily exposed to common bacteria when breast feeding is discontinued, a circumstance deemed largely responsible for weanling diarrhea. Diarrhea tends to be more common and severe among malnourished children. Processes that could contribute to this problem include morphological alterations of the intestinal mucosa in malnourished children, poor intestinal absorption of fats and other nutrients, irritation caused by increased concentrations of free bile acids, and changes in the composition of the intestinal flora. Diarrhea control alone could greatly improve children's nutritional status, while breast feeding in the early months of supplemented later, and followed by a sound diet after weaning considerably reduce the danger and damage caused by diarrheic infections.

Bender BS see Quinn TC

Bengoa JM see Rosenberg IH

Bennet J. A review of antidiarrhoeal compounds. <u>In:</u> Gough D, ed. The control of diarrhoea in clinical practice. London: The Royal Society of Medicine, 1978:1-8 (Royal Society of Medicine International Congress and Symposium series, 5)

Berant M see Korman SH

Berger SA see Pitl1k S

Berrut C, Loizeau E. [Chronic diarrhea: current aspects]. Ther Umsch 1984 Sep;41(9):618-24

Bhattacharya AK see Mahalanabis D

Bhattacharya SK. Acute diarrhoeal diseases $_{\parallel}$ in newborn. J Indian Med Assoc 1987 Jul;85(7):213, 209

Binder HJ see Dobbins JW

Binderelf A see Neilands JB

Black RE. Pathogens that cause travelers' diarrhea in Latin America and

Africa. Rev Infect Dis 1986 May-Jun;8(suppl 2):S131-5

With the advent of rapid and convenient means of transportation, approximately 12 million persons travel each year from industrialized to developing countries in the tropics and subtropics. These travelers are at risk for a variety of infectious diseases that are endemic in these areas; the most frequently occurring of these is diarrhea. Studies of groups of travelers to Latin America and Africa have found that approximately one-half develop diarrhea during their stay abroad. Etiologic investigations of these illnesses have demonstrated that the important agents that cause travelers' diarrhea are similar to those that cause diarrhea in children in the developing countries. One-third of the cases are associated with enterotoxin-producing strains of Escherichia coli. Smaller proportions appear to be due to rotavirus, Norwalk virus, Shigella, Salmonella, Giardia lamblia, and Entamoeba histolytica. Although they have not been fully evaluated in travelers' illnesses in Latin America or Africa, <u>Campylobacter jejuni</u>, <u>Aeromonas hydrophila</u>, viruses, and <u>Cryptosporidium</u> probably cause some of the currently unexplained cases of diarrhea. Although fluid replacement of diarrheal stool losses does not depend on the etiology of diarrhea for the patient in question, more specific prophylactic or therapeutic measures may. Knowledge about the enteropathogens associated with travelers' diarrhea and about the changes over time in their identity should greatly facilitate the assessment of the need for, and the likelihood of success of, attempts to prevent or manage this illness. (Modified author's abstract)

Black RE. The prophylaxis and therapy of secretory diarrhea. Med Clin North Am 1982 May; 66(3):611-21

Black RE. Viral diarrheas. <u>In</u>: Strickland GT, ed. Hunter's Tropical medicine. 6th ed. Philadelphia: Saunders, 1984:124-31

Black RE see Levine MM

Black RH. Invited discussion of Dr R M Glasse's paper. <u>In</u>: Proceedings of the Cholera Research Symposium, Honolulu, Hawaii, 24-29 Jan 1965. Washington, D.C.: U S Government Printing Office, 1965:340

In a discussion generated over R M Glasse's paper on "Cultural aspects of the transmission of cholera" (In: Proceedings of the Cholera Research Symposium, Honolulu, Hawaii, 24-29 Jan 1965:337-9), the author makes several comments on plausible reasons for the difference in the incidence of cholera among communities and among socioeconomic classes. The role of the epidemiologist and that of an anthropologist are clearly defined and demarcated. The author points out that if person-to-person contact in the spread of cholera is emphasized, then the low incidence of cholera at the height of the wet season might be explained by the relatively small amount of movement of people between the small but packed isolated communities. The author agrees with Glasse that, in any anthropological or epidemiological study, the features of family size and structure and their spatial distribution should be taken into account, and these factors need to be appropriately differentiated from Western family structures. It is stated that a number of observations in Glasse's paper may be relevant to differences in the incidence of cholera amongst members of different socioeconomic classes. These include the use of alum precipitation of water, the use of soap for hand cleansing, the use of latrines, and the stricter observance of Muslim law by the rich regarding cleansing after

defecation. These are rightly the items of possible significance put forward by the anthropologist for consideration by the epidemiologist. The casual defecatory behavior of children has been presented as a possible mechanism for the dissemination of the cholera Vibrio. If direct contact with the feces of vouna children is significant in the transmission of anthropological findings suggest that there 'should be a higher incidence in women and girls, who are more exposed to such contact than males. The author, therefore, emphasizes the need of looking at the problem of transmission of cholera from the point of view of the anthropologist, who is likely to have an assortment of field notes on the behavior of people in a cholera-stricken area. The author lays particular emphasis on the findings of the anthropologist - who studies people in a society. The epidemiologist, later, examines the findings of the anthropologist in an attempt to locate those factors which may be of significance in the transmission of disease - in this case cholera.

Blacklow NR, Cukor G. Viral gastroenteritis. N Engl J Med 1981 Feb;304(7): 397-406

Acute viral gastroenteritis, an extremely common illness, affects all age groups and occurs in both epidemic and endemic forms. Norwalk virus is associated with family and community-wide outbreaks of qastroenteritis among school-age children, family contacts, and adults. This form typically produces an explosive, self-limited illness that usually lasts for 24 to 48 h, and is accompanied by vomiting, diarrhea, nausea, abdominal cramps, low-grade fever, anorexia, malaise, and myalgia. Rotavirus is associated with gastroenteritis which is usually sporadic and occasionally epidemic, occurring predominantly in infants and young children. This form of illness typically produces severe diarrhea lasting for 5 to 8 days, and is usually accompanied by fever and Infants may become severely dehydrated, requiring hospitalization vomiting. for fluid replenishment. The biologic characteristics, diagnostic tests, epidemiology, pathogenesis, and clinical epidemiology of Norwalk virus and rotaviruses have been described. Therapy of viral gastroenteritis is limited to supportive measures. Patients with severe dehydration, particularly elderly, very young, or debilitated persons require parenteral replacement. Oral rehydration with glucose-electrolyte formula has been effective in most infants with 5 to 10% dehydration due to rotavirus diarrhea. The possibility that a vaccine against rotavirus can be developed gives considerable hope for reduction of morbidity and mortality due to this pathogen in future.

Blackshaw AJ, Levison DA. Eosinophilic infiltrates of the gastrointestinal tract. J Clin Pathol 1986 Jan;39(1):1-7

Blackwell TE. Enteritis and diarrhea. Vet Clin North Am [Large Anim Pract] 1983 Nov;5(3):557-70

This paper highlights aspects pertaining to diseases in sheep and goats that are characterized by enteritis or diarrhea. The diseases are discussed under etiologic agents classified as bacterial, viral, and parasitic agents. Detailed clinical examinations, together with sound knowledge of the enteric diseases of sheep and goats, are necessary to establish accurate diagnosis. Symptomatic therapy may be administered while awaiting laboratory results. The most common form of symptomatic treatment, indicated for the majority of gastrointestinal disorders, is replacement of fluid and electrolyte losses. The therapy may be administered intravenously, subcutaneously, or orally.

Severe diarrheas in young lambs and kids (Escherichia coli, salmonellosis, rotavirus) often result in exaggerated loss of bicarbonate ions in proportion to other electrolytes. In these cases, "balanced" electrolyte solutions may not be adequate to correct the accompanying acidosis, and additional bicarbonate may be necessary. The role of antibiotics and anthelmintic agents has been discussed. It has been suggested that, in some cases, diarrhea may be a secondary problem. A carefully recorded history and clinical examination of the patient will reveal the true nature of the disease process and will lead to the treatment of the animal for the specific problem rather than the secondary diarrhea.

Blake NJ see Rodrick GE

Blake PA, Weaver RE, Hollis DG. Diseases of humans (other than cholera) caused by vibrios. Annu Rev Microbiol 1980;34:341-67

This review summarizes the current status of knowledge on disease associated with vibrios other than <u>Vibrio cholerae</u> 01. These species include V. cholerae non-01, <u>V. parahaemolyticus</u>, <u>V. vulnificus</u>, <u>V. alginolyticus</u>, and two vibrios of uncertain pathogenicity for humans - group F (EF6) vibrios and <u>V.</u> metschnikovii (enteric group 16). Both Vibrio species (V. cholerae and V. parahaemolyticus) that definitely cause gastroenteritis as well as the other vibrios, can be isolated from stool specimens by direct inoculation of thiosulfate bile salt sucrose agar. Vibrios are facultative anaerobes that are usually oxidase-positive and ferment glucose without forming gas. The methods of identification of different Vibrio species have been outlined. The clinical features, ecology, pathogenicity, and epidemiology of V. cholerae non-01, V. parahaemolyticus, V. vulnificus, and V. alginolyticus have been discussed. Seafoods (eaten cooked or raw) and/or seawater are clearly important vehicles of transmission of these organisms. When seafoods are eaten cooked, preventing the infection is simple; regardless of Vibrio content, such foods will be safe if they are cooked at enough high temperature to sterilize them. of shellfish that have been subjected to fecal contamination should be prevented. Heightened awareness of clinicians, microbiologists. epidemiologists of the existence of pathogenic vibrios and the settings which they inhabit may evolve improved techniques of isolation, control disease transmission, and treatment of the different diarrheas caused by them.

Blaser MJ, Reller LB. <u>Campylobacter</u> enteritis. N Engl J Med 1981 Dec 10;305 (24):1444-52

Campylobacters, which are neither aerobes nor anaerobes, represent a newly recognized class of agents that are infectious to human beings. Background information on what led to the documentation of the importance of Campylobacter jejuni currently known information on Campylobacter enteritis are presented in this review. Evidence syggesting that C. jejuni are diarrheal pathogens which affect human beings has been presented. Avian species are reservoirs of C. jejuni. The organism has been isolated from fresh and salt water and has been shown to survive in fresh water for up to 5 weeks. Transmission of C. jejuni appears to occur by the fecal-oral route through contaminated food and water or by direct contact with fecal materials from infected animals or persons. The prevalence of C. jejuni infection in some of the developing countries may be much greater than that reported from industrialized countries. The epidemiology of infection is quite different in the developed and developing countries; infection occurs very early in life in the developing countries.

Most illnesses have occurred from 1 to 7 days after exposure to a known vehicle; longer incubation periods may be due to smaller inoculum size. Organisms surviving the gastric acid barrier reach the bile-rich, micro-aerobic upper small intestine. The sites of tissue injury include the jejunum, ileum, and colon, with similar pathological features in each. symptoms of the affected persons are diarrhea, abdominal pain, malaise, fever, nausea, vomiting, and constitutional problems. Most patients recover in less than a week, but 20% may have a relapse or a prolonged or severe illness. Simplification of culture techniques now allows the diagnosis of Campylobacter enteritis to be carried out with considerable ease at almost Therefore, C. jejuni should be sought clinical-microbiology laboratory. routinely in fecal specimens from patients with diarrhea. Enteric infection with <u>C. jejuni</u> is frequently self-limited. Anecdotal reports indicate rapid resolution of symptoms and fewer relapses after antimicrobial therapy. Erythromycin is the drug of choice on the basis of the frequency with which the organism is susceptible, the ease of administration, the high tissue levels. and the lack of serious toxicity. Alternative agents include tetracycline, doxycycline, and clindamycin. The need for handwashing to prevent C. jejuní infection has been stressed.

Blaser MJ. Environmental interventions for the prevention of travelers' diarrhea. Rev Infect Dis 1986 May-Jun;8(suppl 2):\$142-50

The diarrheal illnesses affecting travelers to areas with low standards of hygiene are due to exposure to microbial agents not in wide circulation in the travelers' own home area. A major objective for the prevention of travelers' diarrhea should be to minimize exposure to these infectious agents. Studies of sporadic and epidemic travelers' diarrhea have shown that contaminated food and water are usually the most important vehicles for transmission of these agents. Travelers must know which foods and water sources to avoid and which they may reasonably be assured as safe. Also, methods for disinfecting potentially contaminated sources must be simple and practical. Acceptable methods for ensuring the safety of food and drink are amply documented in the literature. However, although the consumption of certain foods and beverages is clearly associated with an increased risk of developing travelers' diarrhea as demonstrated by some retrospective studies, an adherence to strict dietary rules generally did not appear to diminish the incidence. Despite these findings, whose validity may have been weakened by study-design flaws, careful attention to the preparation and choice of food and beverage is recommended for prevention of both diarrheal and nondiarrheal illnesses. (Modified author's abstract)

Blaser MJ, Taylor DN, Feldman RA. Epidemiology of <u>Campylobacter jejuni</u> infections. Epidemiol Rev 1983;5:157-76

Campylobacter jejuni are now recognized as a group of pathogenic bacteria which cause gastroenteritis in humans. This paper presents an overview of the clinical syndrome of Campylobacter enteritis and discusses the reservoirs of C. jejuni and the mechanisms of transmission to humans. The data on the prevalence of this infection have been reviewed to determine which groups are at significant risk. Because infection does not always lead to illness, the findings that some groups have lower illness-to-infection ratios than others have also been reviewed. As with Salmonellae, the major identified reservoirs for Campylobacter are animals and products obtained from animals. Although milk most frequently has been shown to be a vehicle for Campylobacter, future

investigations may identify poultry, poultry products, and meats obtained from other animals as major vehicles. It is unlikely that pets will be the major vector, although Campylobacter from puppies and kittens may infect some humans. The seasonality of Campylobacter infections in humans may relate to the frequency of contamination of animal products used for human food, but may also relate to the fact that, in warmer weather, larger doses are ingested, resulting in higher illness-to-infection ratio. It is probable that, for some little variation Campylobacter serotypes, there may be in distribution. Although serotypes of Campylobacter have not yet been identified that are specifically associated with vehicles or with the magnitude of clinical illness, such associations have been found for Salmonellae, and may be expected for campylobacters. Campylobacters have been found frequently in the developing countries, while <u>Salmonellae</u> are less frequently found. This phenomenon deserves exploration, since it suggests differences between reservoirs or transmission patterns of Campylobacter and Salmonella not yet identified. In the developed countries, Campylobacter infection is a common cause of acute diarrhea; however, deaths appear to be uncommon. In contrast, the prevalence of Campylobacter infection is seen in young children aged under 2 in developing countries.

Blaser MJ. Gastric <u>Campylobacter-like</u> organisms, gastritis, and peptic ulcer disease. Gastroenterology 1987 Aug: 93(2): 371-83

Blaser MJ. Infectious diarrheas: acute, chronic, and iatrogenic [editorial]. Ann Intern Med 1986 Nov;105(5):785-7

Blodgett RC see Rocha MP

Bloom SR see Nicholl CG

Bockermuhl J see Meyer-Kawohl R

Bodart P see Pringot J

Bohach GA see Cavalieri SJ

Bohl EH. Rotaviral diarrhea in pigs: brief review. J Am Vet Med Assoc 1979 Mar 15;174(6):613-5

A rotavirus has recently been incriminated as a cause of diarrhea in young pigs. Only limited information is available on the natural occurrence and significance of rotaviral infections in swine. This report gives a brief review of some of the known and probable characteristics of rotaviral infections in swine. Rotavirus is a name given to a group of viruses that have similar characteristics and are generally capable of causing diarrhea in the young. Infection of pigs with porcine rotavirus is common and widespread and can result in diarrhea, especially in 1- to 4-week-old pigs. This virus is frequently associated with a diarrheal syndrome popularly known as "white scours", "milk scours", or "3-week-old scours". Pigs less than I week old are infrequently infected, presumably because of adequate passive immunity. The infection resembles enzootic transmissible gastroenteritis. Diagnosis can be made by immunofluorescent staining of mucosal scrapings from the small intestine. (Modified author's abstract)

Bondarenko VM see Zhalko-Titarenko VP

Boosinger TR see Riviere JE

Booth CC. Diarrhoea due to intestinal malabsorption. Proc R Soc Med 1963 Dec;56(12):1068-70

Intestinal malabsorption causes two main problems: (1) diarrhea associated with steatorrhea that results from fat malabsorption; and (2) malnutrition caused by impaired absorption of essential nutrients. Patients with malabsorption. therefore, may present with diarrhea, nutritional deficiencies or both, Dader summarizes briefly the types of malabsorption and nutritional deficiencies that occur in patients with idiopathic steatorrhea, surgical conditions associated with malabsorption, pancreatic disease, and chronic tropical sprue. Of the 55 patients hospitalized with idiopathic steatornea. 26 had diarrhea. There was little or no diarrhea in 21 other patients who had nutritional deficiencies. Three main surgical conditions cause malabsorption: intestinal resection, blind loop syndrome and Crohn's disease, conditions most frequently involve the distal small intestine, and cause total malabsorption of vitamin B_{12} , and partial fat and protein malabsorption. The striking feature of pancreatic disease is that malabsorption may cause little diarrhea, even when pancreatic secretion is completely absent. Tropical sprue - a type of intestinal malabsorption associated with nutritional deficiencies - occurs in tropical areas, such as India, Burma, Malaysia, Hong Kong, Indonesia, and certain West Indian islands.

Booth IW, Cutting WAM. Current concepts in the management of acute diarrhoea in children. Postgrad Doc - Middle-East 1984 Jul:418-24

The essential treatment for children with acute diarrhea is the intake of extra fluids by mouth. Domestic drinks are satisfactory for mild cases, but moderate dehydration calls for carbohydrate-electrolyte solutions which enhance absorption. Drugs should not be used routinely in acute infectious diarrhea, but there are specific indications for antibiotics, particularly during cholera and Shigella dysentery. Antimotility drugs do more harm than good in children; the available antisecretory drugs have significant side-effects, and kaolin has no proven value. Withholding food during diarrhea does not speed up recovery, and in undernourished children contributes to the diarrhea-malnutrition syndrome. Breast feeding during diarrhea is beneficial. Early reintroduction of diet after initial rehydration and extra food during convalescence counteract the negative nutritional balance caused by the disease. (Modified author's abstract)

Booth IW, Harries JT. Inflammatory bowel disease in childhood. Gut 1984 Feb; 25(2):188-202

Infants and children are not only growing but also developing, both physically and intellectually, and these differences explain why inflammatory bowel disease produce differing symptoms and management problems compared with adults. This review highlights these differences rather than to provide a totally comprehensive account of inflammatory bowel disease in childhood. Many of the views in this study reflect experience and practice which are essentially empirical and pragmatic where there are no scientifically well founded guidelines available. The incidence, clinical manifestation (Crohn's disease and ulcerative colitis), laboratory assessment, colonoscopy, and management of inflammatory bowel disease have been discussed. It is suggested that the adoption of a team approach to the management of inflammatory bowel

disease in children is important, with the involvement of physician, surgeon, psychiatrist, medical social worker, and stoma therapist. Not only will this type of approach improve the patient's sense of wellbeing but may also reduce the number of relapses in ulcerative colitis.

Booth IW, Levine MM, Harries JT. Oral rehydration therapy in acute diarrhoea in childhood. J Pediatr Gastroenterol Nutr 1984 Sep;3(4):491-9

A controversy is alive concerning the concentration of sodium in oral rehydration solutions (ORS). This paper summarizes the rationale for ORS, some practical aspects of administration and the likely future role of this form of management of acute diarrhea and vomiting. The glucose-coupled sodium transport, and the composition of a multi-purpose ORS are discussed. It has been found that oral rehydration therapy (ORT) with early refeeding may have a beneficial effect on nutritional status, following an episode of acute diarrhea. The use of a rice-powder and a glycine-glucose ORS represents imaginative ways of increasing the practicability, nutritional value and efficiency of ORS. Evidence is accumulating that appropriate administration of an ORS, containing 90 mmol/l sodium, may be equally as safe and effective in moderate-to-severely dehydrated, well-nourished children as in malnourished patients in developing countries, irrespective of the serum sodium level on admission. However, in Western industrialized countries, such as the UK, any form of ORT is probably underutilized by those who provide primary health care. This indicates that much remains to be done to ensure the delivery of ORT to children in the west as effectively as has been done in many parts of the developing world.

Booth IW, Candy DCA. Practical problems in protracted diarrhoea. J Trop Pediatr 1987 Apr;33(2):69-74

The practical problems of protracted diarrhea of infancy have been examined to focus on the causation mechanisms of diarrheal illnesses and their management. and to define areas for future research. While novel causes of protracted diarrhea of infancy continue to be identified, malnutrition appears to play a central role in the initiation and perpetuation of a wide variety of disturbances of gastrointestinal structure and function which characterize the patient with protracted post-gastroenteritis diarrhea. Although the exact pathogenesis of protracted diarrhea is rarely fully understood in any given protracted patient, it is clear that breaking the vicious cycle of diarrhea/malnutrition by appropriate nutritional measures is the most important single factor in successful management. Successful dietary management is usually a compromise between the pressing nutritional demands of the patient, and the limitations imposed upon meeting these demands by impaired small intestinal solute transport. At present, further information about the net absorption of a variety of nutrients in protracted diarrhea in developing countries is urgently required, to found management on a more rational footing. In particular, a formal evaluation of human breast milk and modular, possibly meat-based feeds is needed, in settings where the problem of protracted diarrhea is most pressing. The routine use of antisecretory/antimotility drugs has not been recommended.

Bottone EJ. <u>Yersinia enterocolitica</u>. <u>In</u>: Ellner PD, ed. Infectious diarrheal diseases. New York: Dekker, 1984:13-48

Bottone EJ, ed. <u>Yersinia enterocolitica</u>. Boca Raton, Florida: CRC Press, 1981. 224 p.

Boyd WP, Jr., Bachman BA. Gastrointestinal infections in the compromised host. Med Clin North Am 1982 May:66 (3):743-53

Bradley DJ see Feachem RG

Bradshaw MJ, Harvey RF. Antidiarrhoeal agents: clinical pharmacology and therapeutic use. Curr Ther 1983 Feb:65-73

An appropriate treatment for diarrhea depends on determining the cause of the condition, and instituting treatment to correct the underlying pathological process. Whether the diarrhea is acute or chronic, life-threatening or merely inconveniencing, how much the diarrhea interferes with the patient's ability to lead a normal life will determine the type of approach best suited to the individual problem. Pathophysiological processes leading to an excess of water in the stool, particularly the mechanisms of water and electrolyte absorption and secretion within the bowel, are discussed. Changes in intestinal motility are now thought to be secondary to fluid accumulation in the lumen in producing diarrhea, but reduced segmenting activity by fluid luminal contents may influence its severity. Based on the pathological process of a case of diarrhea, the application of antidiarrheal agents are divided into categories: (1) agents that absorb water; (2) agents that adsorb toxins stimulating secretion; (3) agents altering electrolyte transport or secretion; and (4) agents altering intestinal motility. There is, however, little evidence to show that general adsorbents adsorb toxins, viruses, and bacteria effectively. In trials, which allowed a 'flexible dosage, codeine phosphate, loperamide, and diphenoxylate, all reduced stool frequency to the same extent, with loperamide and codeine being more effective in relieving urgency and incontinence. Treatment of acute diarrheas, such as Campylobacter enteritis, typhoid fever, travelers' diarrhea, shigellosis, cholera, enterocolitis, giardiasis, and pseudomembranous colitis, is outlined. Specific problems, including irritable bowel syndrome and steatorrhea, are also focused with reference to current patient management techniques that use antibiotics and other agents.

Brandborg LL see Owen RL

Brasitus TA. Parasites and malabsorption. Clin Gastroenterol 1983 May;12(2): 495-510

Association of intestinal parasites (both protozoa and helminths) with nutrient malabsorption is reviewed. It appears that giardiasis, coccidiosis, cryptosporidiosis, strongyloidiasis, capillariasis and perhaps <u>Plasmodium falciparum</u> malaria are the only parasitic diseases which cause malabsorption of many nutrients. <u>Diphylobothrium latum</u> and <u>Ascaris lumbricoides</u> interfere with, respectively, vitamin B₁₂ and vitamin A absorption. In view of the increasing use of immuno-suppressive therapy, it is likely that malabsorption due to intestinal parasites may become even more evident in the future.

Brenner U see Muller JM

Brinton CC, Jr. see Levine MM

Briscoe J. Intervention studies and the definition of dominant transmission routes. Am J Epidemiol 1984 Sep;120(3):449-55

"A common approach to assessing the relative importance of different

transmission routes is to eliminate transmission through one route and assume that the ratio "number of cases eliminated: number of residual cases" measures the relative importance of the eliminated route vis-a-vis the residual transmission route. A quantitative model is used to generate synthetic data similar to those analyzed by epidemiologists. These data are analyzed using this conventional procedure and the inferences drawn from the synthetic data compared with the causal relationships structured into the model. The implications for the analysis of real-world data are analyzed by examining data on the importance of water and other transmission routes for cholera in Bangladesh." (Author's abstract)

Briscoe J, Feachem RG, Rahaman MM. Measuring the impact of water supply and sanitation facilities on diarrhoea morbidity: prospects for case-control methods. Geneva: World Health Organization, 1985. 71 p. (WHO/CWS/85.3; CDD/OPR/85.1)

"Epidemiological studies of the effect of water supply and sanitation facilities on diarrhoeal disease using the conventional prospective methodologies face formidable problems. They require very large sample sizes, require many years to complete and face a variety of serious validity problems. An alternative approach, using a case-control study design, is proposed. Methods for dealing with some of the major potential problems due to misclassification and selection biases are outlined. It is concluded that the approach is feasible and that it overcomes many of the more serious defects in standard health impact evaluation designs. Specifically, the required sample sizes are an order of magnitude less than those required in the standard designs, the studies can be initiated after a project is functioning effectively and is used appropriately by the population, the ethical problems encountered in withholding improved facilities from study populations are absent, the results of the study can be available within a year of initiation of the evaluation, and the resource requirements of the studies are modest. It is proposed that detailed protocols for case-control HIEs (health impact evaluations) of water and sanitation facilities be developed and tested in developing countries, preferably in the context of ongoing diarrhoeal disease research projects. It is hoped that, after the development and testing of such protocols, it will be possible to develop general guidelines for the conduct of case-control studies of the impact of water supply and sanitation facilities on morbidity due to diarrhoeal diseases." (Authors' abstract)

Briscoe J. Public health in rural India: the case of excreta disposal. Cambridge: Center for Population Studies, Harvard University, 1976. xix, 414 p. PhD Thesis. (Research paper, 12)

Briscoe J. The role of water supply in improving health in poor countries (with special reference to Bangladesh). Am J Clin Nutr 1978 Nov;31(11): 2100-13

Findings from a limited set of studies on water supply and diarrheal diseases, conducted in a rural area of Bangladesh, are reported. Some hypotheses have been offered to explain the unexpected and contradictory findings. These hypotheses are: (1) the use of tubewell water for drinking does not protect individuals against cholera; (2) cholera in rural Bangladesh is not primarily a waterborne disease; (3) the small amount of protection afforded by drinking bacteriplogically safe water is overwhelmed by the exposure to polluted surface' water through bathing, food preparation, and utensil washing; (4) in families

who are tubewell users, there may be individuals who do not drink tubewell water, and they constitute the group that is most susceptible to cholera: (5) those who use water from "disconnected" tanks for their surface water requirements are likely to have lower cholera attack rates than those who use canal or river water for drinking, cooking, bathing, and utensil washing. The policy implications of studies on water supply and cholera, the use of cholera as a model for water-related diseases, the use of "intermediate variables" in research and planning, and the specification of water supply standards different water uses have been discussed. In water supply policy. fundamental need appears to be a reorienting and restructuring of decision-making process. The continuation of traditional water use habits even when people of developing countries are presented with alternative sources of ascribed to the "ignorance pure water is denerally of the masses". The prescription then becomes education of the ignorant and the identification of the communication barriers. The importance. ٥f decision-making on issues relating to water improvement program has been stressed.

Briscoe J. Water supply and health in developing countries: selective primary health care revisited. Am J Public Health 1984 Sep:74(9):1009-13

"The inclusion of water supply and sanitation programs as a component of primary health care (PHC) has been questioned on the basis of calculations of the costs of these programs per infant death averted. In this paper, the procedures used in these cost-effectiveness calculations are examined and found to be wanting. The calculations are misleading since gross rather than net costs have been used, and the health impact of these programs underestimated. It is also shown that the methodology used is biased against water supply and sanitation and other programs with multiple outputs. The time constraints facing mothers in implementing PHC programs, as well as the contribution of improved water supplies in alleviating these constraints are outlined. Data are presented to show that, if poor women in developing countries were to choose the mix of activities to be included in PHC programs, improved water supplies would frequently constitute a part of that mix." (Author's abstract)

Brogan MD see Targan SR

Brooks JB. Review of frequency-pulsed electron-capture gas-liquid chromatography studies of diarrheal diseases caused by members of the family Enterobacteriaceae, Clostridium difficile, and rotavirus. J Clin Microbiol 1986 Nov;24(5):687-91

Frequency-pulsed electron-capture gas-liquid chromatography (FPEC-GLC) studies of diarrheal diseases have produced some interesting findings which could be useful to improve diagnosis to study changes in metabolic patterns that occur in the gut during the diarrheal state, and to detect specific compounds that are potentially physiologically active or carcinogenic. The purpose of this review is to summarize information obtained thus far in FPEC-GLC studies of diarrheal disease associated with membranes of the family Enterobacteriaceæe, Clostridium difficile, and rotavirus. Extraction at different pHs and derivatization with specific functional group reagents increase the chances for the correct labeling of chemical compounds detected by GLC analysis. Gas chromatography analysis, and aids to data interpretation, and automation are discussed. The data indicate that FPEC-GLC analysis of stools might be a rapid way to distinguish the most commonly encountered members of the

Enterobacteriaceae, C. difficile, and rotavirus associated with acute diarrhea in infants. A limited number of organisms have been studied by FPEC-GLC; therefore, comparisons for the purpose of identification among the Enterobacteriaceae must be made with organisms that were previously studied by FPEC-GLC. The time for analysis with a 2-column instrument is about 4 h. Automatic sample injection takes advantage of off-duty hours and reduces the cost of analysis. Laboratory managers interested in establishing this FPEC-GLC technique should begin with the purchase of a quality gas chromatogram equipped with dual FPEC-GLC detectors and make-up gas. The FPEC-GLC system could also be used for the rapid determination of tuberculous meningitis and perhaps other types of diseases by analysis of cerebrospinal fluid and serum.

Broor S, Singh V. Viral gastroenteritis. Indian J Gastroenterol 1984 Oct;3 (4):225-9

Two agents which cause two distinct epidemiological and clinical forms of viral gastroenteritis are rotavirus and Norwalk virus. The 70-nm rotavirus is the single most important worldwide cause of diarrhea in infants and young children, occurring usually in sporadic form with occasional epidemics. 27-nm Norwalk and Norwalk-like viruses characteristically cause epidemic viral qastroenteritis in families or community settings affecting mainly school-age children and adults. The incubation period of rotavirus diarrhea ranges from 1 to 7 days but usually less than 48 h. In infants and young children, the onset is usually explosive with vomiting and diarrhea. Mild fever is seen in 63-100% of the cases. Average duration of illness is 5-7 days, but virus shedding continues for about 10 days. Mild-to-moderate dehydration is seen in about 80% of the cases. Rotavirus diarrhea causes mucosal abnormalities in the form of blunting of villi and replacement of columnar epithelial cells by immature cuboidal cells. Functional alterations of duodenal mucosa include decrease in disaccharidase level and impaired xylose absorption. Rotavirus is excreted in large numbers in feces and can be easily identified under an electron microscope. However, the enzyme-linked immunosorbent assay is the most microscope. practical method, and is used widely for the diagnosis and epidemiological The incidence of rotavirus infection is studies of rotavirus infection. highest in infants aged 9-12 months. In temperate climates, it is more prevalent during the cooler months, while in the tropics, the seasonal variation is not so clear. Immunity to rotavirus and possibility of prevention through vaccines or passive immunization have been discussed. Epidemic viral gastroenteritis, caused by Norwalk and related agents, is a self-limiting disease lasting for 24-48 h. Average incubation period is 12-24 h. The clinical features include nausea (85%), vomiting (84%), abdominal cramps (52%), lethargy (57%), diarrhea (44%), fever (32%), and chills (5%). Immune-electron microscopy of fecal filtrates is the only reliable technique available for identifying Norwalk viruses in stool. Other viral agents that may cause acute gastroenteritis, such as adenoviruses, astroviruses, caliciviruses, coronaviruses, are also described.

Brown KH, MacLean WC, Jr. Nutritional management of acute diarrhea: an appraisal of the alternatives. Pediatrics 1984 Feb;73(2):119-25

Recent evidence has convincingly demonstrated an inverse relationship between the prevalence of diarrheal disease and the growth of children in less-developed countries. Growth failure, secondary to diarrhea, has been attributed to the negative impact of diarrhea on dietary intake and to impaired intestinal absorption during and after enteric infections. Catabolic responses to infection also exact a nutritional toll, especially in febrile illnesses. The relative importance of these mechanisms has not been conclusively determined, but a renewed appreciation of the complexities of the relationships among enteric infection, dietary therapy, and nutritional status following diarrhea has led to serious rethinking of the most appropriate way to minimize or replace both macronutrient and micronutrient losses induced by diarrhea. The approach to diarrhea by most pediatricians in the USA and elsewhere consists of a variable period of food reduction or, more likely, fasting - to malabsorption clinical complication of food-induced avoid the compensatory "overfeeding" during the recovery period. Pediatricians also advocate continued feeding during diarrhea. Although the correct therapeutic approach is currently debated, well-controlled studies of the alternatives are, for the most part, lacking. The purpose of this paper is, therefore, to examine the possible advantages and disadvantages of each of these approaches to the nutritional management of childhood diarrhea and to review the limited data available in the literature. Whether fasting during an episode of acute diarrhea is necessary or desirable has also recently been questioned. principal argument advanced for limited fasting is the avoidance of the consequences of malabsorption, namely acidosis, excessive fluid losses, depletion of the bile acid pool, and possible mucosal injury from unabsorbed foods. Advocates of continued feeding during acute diarrhea suggest that the practice will prevent deficits of intakes of protein and calories, maintain or stimulate repair of the intestinal mucosa, and sustain breast feeding in the breast-fed infant. There are only a limited number of clinical studies that address the issue. Available evidence suggests that, in most cases, current practice should be modified to minimize food withdrawal.

Brown KR, Phillips SM. Tropical diseases of importance to the traveler. Adv

Bryant RG see Janda JM

Buchino JJ, Suchy FJ, Snyder JW. Bacterial diarrhea in infants and children. Perspect Pediatr Pathol 1984 Summer;8(2):163-80

This paper reviews both the expanded concepts of the pathophysiology of bacterial diarrhea and the recognition of pathogenic organisms that have taken place in the last two decades, specifically as they reflect the experience of both the Kosair-Children's Hospital, Kentucky and the Children's Hospital Medical Center, Ohio, both in the USA. Host defense mechanisms play a vital role in the determination of susceptibility to gastrointestinal pathogens. Since many of the mechanisms may be different or not fully developed in children, as compared with adults, the recognition of those mechanisms becomes essential to understanding the pathogenesis of infectious diarrhea. From January 1976 to December 1980, 16,779 fecal cultures were processed at Kosair-Children's Hospital and 34,192 at Children's Hospital Medical Center. Despite the difference in number of total cultures performed, the yield of positive isolates (2.8% at Kosair-Children's Hospital and 3.5% at Children's Hospital Medical Center) are quite similar. Also, the relative frequency of isolations for each organism varied negligibly between the 2 institutions. The major pathogenic organisms -- Salmonella, Shigella, Campylobacter, Yersinia, Escherichia coli, and Clostridium difficile -- have been discussed and reviewed. Currently, many laboratories identify pathogens in more than 60% of cases. It is not practical to examine stool specimens for all potential pathogens. Clinical data, such as patient's age, history of food and water

intake, history of travel and recent antibiotic therapy, can be helpful to the laboratory in deciding which specific pathogen to investigate. Specimen collection and transport, primary culture processing and culture characteristics and identification have been discussed at length. A minimum of 3 consecutive fecal specimens (one per day) has been recommended for the diagnosis of both acute diarrhea and the carrier state. Specimens should be transported and processed with minimal delay, while laboratory reports should specify the extent to which the specimen has been examined.

Bukhave K see Rask-Madsen J

Bundy DAP. Epidemiological aspects of <u>Trichuris</u> and trichuriasis in Caribbean communities. Trans R Soc Trop Med Hyg $\overline{1986;80(5)}:706-18$

This review argues for a reappraisal of the health significance of the human whipworm, Trichuris trichiura. Infections with this geohelminth are at as prevalent as Ascaris lumbricoides in many localities, and are associated with significant morbidity. Infection may result in severe trichuriasis syndrome or, more frequently, in a chronic colitis associated with growth stunting. Under-reporting of the chronic manifestations of the disease has resulted in a gross under-estimation of the health impact of trichuriasis. Furthermore, estimation of the parameters of the population dynamics of T. trichiura transmission suggests that whipworm infections are intrinsically more resistant to control than those of other common geohelminths. A major determinant of the transmission dynamics and morbidity characteristics of this helminthiasis is the aggregation of worm burden in certain predisposed individuals and age groups. It is suggested that improved understanding of the factors generating this distribution of infection intensity is a prerequisite for effective control of both infection and morbidity. (Modified author's abstract)

Burchard GD. [Clinical importance, epidemiology and laboratory diagnosis of intestinal cryptosporidia infection]. Immun Infekt 1986 Apr:14(2):51-7

Burdon DW. Treatment of pseudomembranous colitis and antibiotic-associated diarrhoea. J Antimicrob Chemother 1984 Dec;14(suppl D):103-9

"Pseudomembranous colitis, is caused by release of toxins from <u>Clostridium difficile</u> when it colonizes the large intestine. This clostridium is <u>susceptible</u> to concentrations of vancomycin which are readily attained in the colon after oral administration. When vancomycin is given orally to infected patients in a dose of 125 mg every 6 h, a rapid clinical cure can be expected. Some patients may relapse after the vancomycin is stopped, but a further course of treatment will control symptoms." (Author's abstract)

Burek JD see Nime FA

Bunke V see Gracey M

Burman D. Iron deficiency in infancy and childhood. Clin Haematol 1982 Jun; 11(2):339-51

Burns E see Feachem RG

Burrichter PJ see Rocha MP

Burrows CF. Chronic diarrhea in the dog. Vet Clin North Am [Small Anim Pract] 1983 Aug;13(3):521-40

A specific understanding of the cause of chronic diarrhea in dog is essential to ensure a specific therapy. A specific diagnosis is based on an understanding of the pathophysiology of diarrhea, on a carefully taken history, and on the logical application of appropriate diagnostic or gastrointestinal function tests. This article describes how the logical and sequential application of this knowledge can be used to diagnose the cause of chronic diarrhea. It is suggested that, once the disorder has been localized, a specific diagnosis can be made by specific gut function studies, by a logical therapeutic trial, or in the large bowel diarrhea, by colonoscopy and biopsy. The possibility of referral of problem cases should never be excluded. It is also indicated that symptomatic therapy cannot be a substitute for more appropriate and more logical treatment which will achieve a specific cure.

Burton P see Tulloch J

Butler T. Playue and other $\underline{\text{Yersinia}}$ infections. New York: Plenum, 1983. 220 p.

Butler TC. Viral diarrhoeas [editorial perspective]. J Diarrhoeal Dis Res 1984 Sep:2(3):137-41

With an estimated 500 million cases of viral diarrhea annually, rotavirus accounts for 10% of all diarrhea cases. Therefore, viral diarrheas deserve attention as a leading cause of morbidity and mortality in the world today. Although many viruses inhabit the human intestinal tract, the two most important diarrheal pathogens are rotavirus and Norwalk virus. belongs to the Reoviridiae family. They are 70-nm in diameter, and can be detected by enzyme-linked immunosorbent assay. Norwalk virus, probably a calicivirus, is smaller (27-nm) and has been detected in stool bν immune-electron microscopy. The rotavirus nucleic acid is known to be RNA, with 11 double-stranded segments. The nucleic acid of Norwalk virus is also likely to be RNA. Both agents occur worldwide and cause disease predominantly in children. The major rotavirus reservoir is believed to exist asymptomatic adults. Children may acquire infection from their parents and other adults. Moreover, rotavirus can be spread within hospitals or other environments. For Norwalk virus, food and water as transmission vehicles have been suggested by epidemiological evidence. Inadequate sanitation has been identified as a transmission risk factor. While they occur year-round, rotavirus infections predominate in the winter. Antibody incidence increases in children aged 2 months to 4 years, and was higher for rotavirus than for Norwalk virus in all age groups. In general, adults have serum antibodies against both viruses, but the titers decline somewhat with age. antibodies do not necessarily protect against reinfection. Diarrhea, usually watery, is the most common symptom of rotavirus diarrhea. It is likely to begin suddenly and to be explosive. The mechanism of rotavirus diarrhea may involve infection of epithelial cells, multiplication of virus particles within the epithelial cells, morphological changes, epithelial cell death, migration of crypt cells up to the villi, etc. Rotavirus diarrhea is treated primarily by rehydration; the virus infection is self-limiting and requires no other therapy. There are two immunological approaches to preventing rotavirus infection which may become future therapeutic and vaccine strategies: give passive immunity to high risk children; and/or give active immunity, using live

oral vaccines. Various approaches to vaccine development have been outlined.

Butterworth AE. Cell-mediated damage to helminths. Adv Parasitol 1984;23: 143-235

Butzler JP, Skirrow MB. <u>Campylobacter</u> enteritis. Clin Gastroenterol 1979 Sep;8(3):737-65

<u>Campylobacter</u> enteritis 15 reviewed with emphasis Literature on pathogenesis, clinical manifestations, pathology, laboratory chemotherapy, epidemiology, and typing of related campylobacters. discussion on nomenclature and classification is presented. This review on Campylobacter enteritis is concerned only with related campylobacters, which are fully capable of infecting healthy people. In a typical clinical attack of Campylobacter enteritis, the average incubation period is 3 to 5 days, but the range extends from one-and-a-half to 7, or even 10 days. In about half of the patients, diarrhea is preceded by a febrile prodromal period with some or all of the following symptoms: malaise headache, dizziness, backache, myalgia, abdominal pain, and sometimes rigors. The feces of patients who are not given chemotherapy usually remain positive for about 2 to 5 weeks after an attack of Campylobacter enteritis. Children tend to be less severely affected than adults, but they sometimes suffer from a relapsing or persistent type of illness. Complications, such as false intussuception and reactive arthritis, have been reported. The principal site of infection seems to be the jejunum and ileum, but there is a growing evidence that the infection is not limited to the small intestine. The passage of bright red blood in the stools as the disease progresses suggests involvement of the colon, and this has been confirmed in some cases by sigmoidoscopy or colonoscopy. The peripheral blood reflects the acute inflammatory pathology. Most patients show a moderate neutrophil leukocytosis, and the erythrocyte sedimentation rate is raised. Most patients form specific antibody to their infecting organism, often to high titers. Methods of laboratory diagnosis, including microscopy, stool and blood cultures, and identification of Campylobacter, is outlined. Specific chemotherapy is discussed along with the in vitro susceptibilities of the infecting organisms. Erythromycin, tetracyclines, or a nitrofuran, such as furazolidone, are the agents likely to be most useful for a patient with Septicemic patients and those ill must receive uncomplicated enteritis. special consideration. The distribution of infection according to sex, age, seasonality and geographic location, and the sources of infection discussed. Methods of typing of related campylobacters that will detect and identify strains of bacteria are outlined with particular emphasis on antigenic typing and biotyping. The results of experimental studies on pathogenic mechanisms of <u>Campylobacter</u> infections indicate that related campylobacters are pathogenic mainly by virtue of a direct invasive ability, and the enterotoxin production is a feature common with only a few strains. It remains to be seen whether enterotoxin-producing strains are associated with more severe types of illness than others.

Butzler JP, Dekeyser P, Detrain M, Dehaen F. Related <u>Vibrio</u> in stools. J Pediatr 1973 Mar;82(3):493-5

The authors hypothesize that the incidence of vibrionic infections has been underestimated for technical reasons. Over a 6-month period, the authors searched for common enteropathogens as well as for <u>Vibrio</u> species in the stools of hospitalized patients, including 800 infants and children and 100 adults

with diarrnea and 1,000 children without diarrhea. By means of a filtration technique for coproculture, it is relatively easy to isolate related vibrios from stool samples, using a medium containing antibiotics. Related vibrios were isolated in 52 specimens from 41 children and 4 adults by means of a filtration technique. A systematic search for Vibrio in the stools of children without diarrhea revealed 13 carriers. On three occasions, a related Vibrio was found in association with Shigella, once with Giardia, and once with Enterobius vermicularis. Although adequate media were used, no strains of Vibrio fetus could, however, be isolated.

Buysse J see Kopecko DJ

Cabada FJ see DuPont HL

Cairncross AM see Feachem RG

Caldwell MB see Walker RI

Calef E see Gitler C

Calkins BM, Mendeloff AI. Epidemiology of inflammatory bowel disease. Epidemiol Rev 1986;8:60-91

Inflammatory bowel disease is a collection of diseases affecting the bowel. The most common are ulcerative colitis and Crohn's disease. The onset of the disease is characterized by a gradual insidious development of chronic complaints, such as episodic diarrhea, colicky abdominal pain, weight loss, and blood, pus and/or mucus in the stool. Although the disease is not common or highly fatal, it is important to public health, because 1) its highest incidence is early in life, 2) its therapy involves major surgery, including a curative colectomy for ulcerative colitis, and 3) having the disease increases the risk of developing intestinal cancer, certainly for ulcerative colitis cases and probably for Cronn's disease cases. The first studies of the epidemiology of idiopathic inflammatory bowel disease were published in the late 1950s and early 1960s. This review presents the major problems confronting an epidemiologic investigation of inflammatory bowel disease the distribution and characteristics of these disorders in human populations. This review also presents the methods currently used in the investigation of the epidemiology of specifically ulcerative colitis and Crohn's disease, for which incidence and mortality are low, and genetic and environmental determinants may be multiple. In the two and one half decades during which such studies have been carried out in both hemispheres, researchers have reached agreement on some fundamental characteristics of cases. Improved means of acquiring and recording data on patients are necessary, and further clarification of the diagnostic characteristics of early disease is important to ascertain cases before many changes in antecedent characteristics have occurred. The impact of diagnosis-related groups on the classification of inflammatory bowel disease cases needs evaluation. Verification of incidence trends in various populations is important; researchers must be sure that the primary classifications have been consistent over past decades to determine whether trends in incidence or mortality have occurred. It is concluded that studies of the trends in incidence in age-specific categories and in birth cohorts would be useful. Confirming the risk at specific ages is important to establish or refute the observation of bimodality. Further study of racial. religious, ethnic, and social characteristics is also needed.

epidemiologic establishment of familial aggregation has been attempted without success to date. Families in which multiple cases of the disease occur should intensively be studied by genetic, nutritional, psychologic, and metabolic techniques; family members thought to be unaffected should closely be followed at intervals for possible detection of early disease. Further studies of possible bacterial or viral agents involved in pathogenesis of inflammatory bowel disease are also needed. Biologic markers related to risk need to be defined. Although psychologic factors do not appear to be a fruitful area for etiologic research, these factors may contribute to the establishment of the disease and its pattern of remissions and exacerbations. Carefully planned studies of dietary factors, antecedent to disease onset, are needed, as well as the examination of post-natal feeding factors, for understanding disease in the young and the effect on developmental patterns of the digestive and immune system. In conclusion, the study of inflammatory bowel disease poses some interesting methodological challenges to investigators. The current list of hypotheses has no one item that can be described as having been firmly researched epidemiologically. What is needed in addition to new methodological approaches is simply more basic descriptive epidemiology. Possibly when more is known about the characteristics of groups at risk, hypotheses will suggest themselves to investigators.

Calloway DH. Nutritional requirements in parasitic diseases. Rev Infect Dis 1982 Jul-Aug;4(4):891-5

"Nutritional requirements" means different things in different contexts. Generally, the term refers to national or international standards or allowances of nutrients. Concern here involves the potential need for a change of standards where conditions of disease prevail, because disease increases the nutritional requirements of most individuals. "Nutritional requirements" may also be viewed in terms of food supplies. Analysis of a number of studies indicates that the average growth deficit due to endemic infectious diseases in early life is $\leqslant 20$ kcal per day (calculated as 5 kcal/g of tissue). Increased weight gain following the treatment of intestinal parasites such as Ascaris lumbricoides or Giardia lamblia provides similar estimates, as does measurement of energy and protein absorption. These values are within normal variance estimates. Sick children do not eat well and apparently do not eat enough on healthy days to correct for the accrued food deficit. Research on nutritional requirements of children needs to focus on management of food resources in entire families." (Author's abstract)

Candy DCA, Leung TSM, Phillips AD, Harries JT, Marshal WC. Models for studying the adhesion of enterobacteria to the mucosa of the human intestinal tract. In: Elliott K, O'Connor M, Whelen J, eds. Adhesion and microorganisms pathogenicity. London: Pitman, 1981:72-93. (Ciba Foundation symposium, 80)

Candy DCA <u>see</u> Booth IW

Cantey JR. Infectious diarrhea. Pathoyenesis and risk factors. Am J Med 1985 Jun 28;78(suppl 68):65-75

An understanding of the pathogenesis of infectious, especially bacterial, diarrhea has increased dramatically. New etiologic agents, mechanisms, and diseases have become known. For example, Escherichia coli serogroup 0157 is now known to cause acute hemorrhagic colitis. Also E. coli serogroups that produce Shiga toxin are recognized as etiologic agents in the hemolytic-uremic

syndrome. The production of bacterial diarrhea has two major facets, bacterial-mucosal interaction and the induction of intestinal fluid loss by enterotoxins. Bacterial-mucosal interaction can be described in stages: (1) adherence to epithelial cell microvilli, which is often promoted by or associated with pili, (2) close adherence (enteroadherence), usually by classic enteropathogenic E. coli, to mucosal epithelial cells lacking microvilli, and (3) mucosal invasion, as with Shigelia and Salmonella infections. Further, large strides in the understanding of the mechanisms of infectious diarrhea are likely with the cloning of virulence genes if additional host-specific animal pathogens become available for study. (Modified author's abstract)

Cantey JR. Shiga toxin - an expanding role in the pathogenesis of infectious diseases. J Infect Dis 1985 May:151(5):766-71

Shiga toxin is among the most toxic biological agents known. This is the neurotoxic, enterptoxic and cytotoxic product of the Shiga bacillus or Shigella dysenteriae. It has recently been known that Shiga toxin is produced by a wide variety of bacterial strains from diverse areas, including Shigellae. Salmonella typhimurium, enterotoxic and enteropathogenic Escherichia coli (EPEC), miscellaneous diarrhea-associated E, coli, E, coli not associated with diarrhea, Vibrio cholerae Ol, non-V. cholerae Ol, and V. parahaemolyticus. The evidence that Shiga toxin is important in pathogenesis remains largely circumstantial, but is convincing in diarrhea due to Shigellae, hemorrhagic colitis, and hemolytic-uremic syndrome. Further evidence is required as to its role in infection with EPEC and other diarrheal diseases, including those due to Vibrio species. The presence of Shiga toxin in bacterial species that possess other powerful enterotoxins (e.g. V. cholerae) and in fecal bacteria not associated with diarrhea elicits some skepticism. It has, however, been suggested that the seemingly ubiquitous. Shiga toxin may have been responsible for diarrhea in volunteers given a V, cholerae Ol that did not synthesize cholera toxin, as well as the bloody diarrhea sometimes caused by non-Ol vibrios. The natural diversity of biological systems runs counter to the potentially unifying role of Shiga toxin. Further studies of pathogenesis, using bacteria with and without the minimum number of genes for Shiga toxin expression, animal models, and probably human volunteers, will be required to answer the remaining questions.

Carmichael L see Pollock RV

Carpenter CCJ. Oral rehydration: is it as good as parenteral therapy? [editorial]. N Engl J Med 1982 May 6;306(18):1103-4

Current estimates indicate that in the most densely populated areas of the world, about half the childhood mortality is caused by acute diarrheal illnesses. Thus, acute diarrheal illnesses are probably the major cause of death on a global scale. Acute diarrheal death, regardless of etiologic agents, almost always can be prevented by adequately correcting intestinal fluid losses. For the past half century, intravenous (i.v.) fluid was the treatment of choice to correct fluid losses in acute diarrhea in both children and adults. Recent studies demonstrated that a glucose/electrolyte-containing oral rehydration solution (ORS) is equally effective for most acute diarrhea illnesses, including those caused by Vibrio cholerae, rotavirus, and enterotoxigenic Escherichia coli. The potential of ORS in acute diarrhea is discussed. A recent study demonstrated that WHO-recommended ORS (with 90 mmol Na/1 and 20 mmol K/1) does not cause complications, such as hypernatremia and

hyper- or hypokalemia. ORS also has been proved an effective acute diarrhea therapy in well-nourished children in developed countries. Serious complications were found in patients receiving i.v. fluid therapy, while complications with oral therapy were minor. The author suggests ORS should be accepted not only as an equal, but perhaps as the superior means of treating acute diarrhea, both in sophisticated, sanitized and rural medical facilities.

Carpenter CCJ. The pathophysiology of secretory diarrheas. Med Clin North Am 1982 May:66(3):597-610

"The mechanisms by which bacterial enterotoxins cause secretory diarrheas have been well defined, and the definitions of such mechanisms have been important in developing a consistently successful therapeutic approach. The less common secretory diarrheas, caused by the interaction of hormones of tumor origin with the gut small intestinal mucosa have also been clearly defined, and their pathogenetic mechanisms are similar to those by which the cholera and \underline{E} . \underline{coli} enterotoxins cause secretory diarrhea. The mechanisms by which histamine \underline{I} and gastrin of tumor origin cause gastric hypersecretion are less clearly delineated; secretory diarrhea caused by both of these agents can be stopped by total gastrectomy without removal of the responsible tumor. The secretory diarrhea, caused by villous adenomas of the colon, which does not appear to be related to a distally produced humoral agent, results in the same picture of hypokalemic acidosis that is characteristic of the nonbacterial secretory diarrheas originating in the small intestine and is cured by resection of the responsible tumor." (Author's abstract)

Cartwright C see Gertler S

Casemore DP, Sands RL, Curry A. Cryptosporidium species: a "new" human pathoyen. J Clin Pathol 1985 Nov;38(11):1321-36

Publications describing aspects of the coccidian protozoan parasite, Cryptosporidium, have increased greatly during 1983 and 1984 as a result of not only increasing veterinary interest but also in the role of the parasite in the acquired immune-deficiency syndrome (AIDS). The reports reflect widespread microbiologists, only between clinicians, collaboration, not histopathologists, but also between veterinary and human health care workers. Cryptosporidium was first described in mice in 1907 and subsequently in various other species; it was not described in man until 1976. Several likely putative species have been described, but there is probably little host specificity. Experimental and clinical studies have greatly increased the knowledge of the organism's biology. The parasite undergoes its complete life-cycle within the intestine, although it may occasionally occur in other sites. The main symptom produced is a noninflammatory diarrhea, which, in patients with AIDS and children in Third World countries, may be life-threatening: even in immunocompetent subjects, the diarrhoea is usually protracted. Attempts to effective chemotherapeutic agents have been Epidemiologically, the infection was thought to be zoonotic in origin, but there is increasing evidence of person-to-person transmission. Diagnosis has depended upon histological examination, but simple methods of detection have now been described: more invasive methods need no longer be used. parasite, which is found more commonly in children, occurs in about 2% of examined fecal specimens and seems to be closely associated with production of symptoms. A serological response has been shown. Much remains to be learned about the epidemiology and pathogenic mechanisms. (Modified authors' abstract) Cash RA see Rohde JE

Caspary WF. Diarrhoea associated with carbohydrate malabsorption. Clin Gastroenterol 1986 Jul;15(3):631-55

Castrucci G see Cilli V

Catassi C see Giorgi PL

Catterall RD. Clinical aspects of Reiter's disease. Br J Rheumatol 1983 Nov; 22(4 suppl 2):151-5

"The clinical manifestations and results of investigations in a series of 221 patients diagnosed as suffering from Reiter's disease are described in detail. Attention is drawn to the very varied natural history of the disease, the relapse rate of 60% and the development of serious complications and disability in young people. The failure to establish the cause of the condition or to unravel its relationship with nonspecific urethritis and bacillary dysentery contrasts with the reported presence of the human leukocyte antigen HLA-827 in 76% of the patients. There is no curative treatment but symptomatic treatment will relieve pain and stiffness and may shorten the duration of individual attacks. Prolonged follow-up of established cases, monitoring of activity of the disease by regular measurements of the erythrocyte sedimentation test and prompt treatment of relapses may prevent the development of serious locomotor disability." (Author's abstract)

Cavalieri SJ, Bohach GA, Snyder IS. <u>Escherichia coli</u> alpha-hemolysin: characteristics and probable role in pathogenicity. <u>Microbiol Rev 1984 Dec;</u> 48(4):326-43

Cello JP. Inflammatory and malignant diseases of the small bowel causing malabsorption. Clin Gastroenterol 1983 May;12(2):511-32

Chadwick VS. Small intestinal secretion in disease. Scand J Gastroenterol 1983;18(suppl 87):91-7

"Although secretion is a normal function of the healthy small intestine it is overall a net absorber of fluid and electrolytes with a substantial reserve capacity. Failure to cope with normal volumes of secretions or the increased volumes secreted in some clinical disorders may both result in diarrhoea. The former situation occurs after massive gut resection, in rapid transit states and in various malabsorptive states. The latter may occur in the presence of mucosal damage or in coeliac disease, inflammatory bowel disease, viral and some bacterial infections or in the absence of damage such as in toxigenic, drug-induced or endocrine diarrhoea where intestinal secretory mechanisms may be activated in healthy enterocytes." (Author's abstract)

Chakraborty B. Diarrhoeal diseases in children [editorial]. J Indian Med Assoc 1987 Jul;85(7):193-5

Chandra RK, Greenough WB, Guerrant RL, Martorell R, Mata LJ, Warren KS, Wu C-C. Diarrhea and malnutrition: research priorities. <u>In:</u> Chen LC, Scrimshaw NS, eds. Diarrhea and malnutrition: interactions, mechanisms, and interventions. New York: Plenum. 1983:305-8

To identify the research priorities in diarrhea and malnutrition, a working

group has presented a set of general objectives, which include: (1) investigation of etiologic and epidemiologic factors in acute and chronic diarrhea, (2) study of the nutritional consequences of severity and duration of diarrhea, (3) examination of sociocultural, behavioral and anthropologic factors, and (4) application of present and emerging technologies in the most cost-effective manner. Based on these general objectives, the working group outlined some specific priority topics for research within the area of (a) etiopathogenesis and epidemiology, (b) nutritional impact, (c) outcome, and (d) interventions.

Chandra RK. Nutritional regulation of immunity and infection in the gastrointestinal tract. J Pediatr Gastroenterol Nutr 1983;2(suppl 1):S181-7

Chang EB, Field M. Intestinal electrolyte transport and diarrheal disease.

In: Kern F, Jr., Blum AL, eds. The gastroenterology annual, 1/1983.

Amsterdam: Elsevier, 1983:148-80

This chapter on electrolyte transport in the intestine and diarrheal diseases summarizes the developments that have increased the understanding of the physiology and pathophysiology of intestinal electrolyte transport and how its aberrations can be treated. There has been especially significant developments of understanding in 4 areas: (1) the basic mechanism of intestinal absorption and secretion; (2) the neuroendocrine regulation of intestinal ion transport: (3) the role of inflammation in activatiny intestinal secretion; and (4) experimental pharmacology of secretory diarrheas. Recent studies have been placed in the conceptual and factual context of earlier studies, and the review attempts to be comprehensive as well as contemporary. New approaches to receptor blockade. therapy. such as vaccine development. nutrient-supplemented oral replacement solutions. are discussed. Pharmacotherapy of secretory diarrheas is outlined and several ways of modifying intestinal ion transport are reviewed.

Chang EB, Fedorak RN. Prostaglandins in diarrheal disease [editorial]. Pediatr Gastroenterol Nutr 1985 Jun;4(3):341-4

Several studies have raised the possibility that prostaglandins may be essential mediators of stimulated intestinal secretion not only in inflammatory conditions of the bowel but also in a wide variety of noninflammatory diarrheal diseases. This editorial reviews what is known so far of prostaglandins and diarrheal disease and the problems that have been encountered in establishing the physiologic and pathophysiologic significance of prostaglandins intestinal absorption and secretion of salt and water. Prostaglandins, especially the E and F series, can stimulate net intestinal secretion in vitro and can produce copious diarrhea in experimental animals and human subjects. Their effect on ion transport appears to be mediated by activation of adenylate-cyclase activity in enterocytes. Prostaglandins have also been shown to increase the propulsive activity of the gut, which could contribute to the diarrhea by decreasing contact time between intestinal chyme and the absorptive surface. Certain prostanoids are shown to be secretory stimulants in the colon. These observations suggest a mechanism by which active secretion is stimulated in acute and chronic inflammation of the intestine. The validity of serum prostaglandin levels in predicting prostaglandin-mediated diarrhea is in doubt, because measurements of serum levels are unreliable. Reports of the effectiveness of aspirin in certain diarrheal disorders are encouraging. However, aspirin-like agents should be used only in circumstances where the

pathophysiological process is fairly well understood, or in situations where the possibility of prostaglandin-mediated secretion exists, and the patient has copious diarrhea unresponsive to other forms of therapy. The 5-amino salicylate preparations may be useful in patients with inflammatory diseases of the small intestine.

Chanock RM see Kapikian AZ

Chatterjee HN. Control of vomiting in cholera and oral replacement of fluid. Lancet 1953 Nov 21;2(6795):1063

This historic study involving oral rehydration therapy was done in Calcutta. Attempting to control vomiting in cholera, to reduce dehydration and prevent shock, the author, during two cholera epidemics in 1952 and 1953, used avomine on, respectively, 59 and 127 cholera patients. Of these 186 patients, 33 had "mild" dehydration and 153 "moderately severe" dehydration. As avomine does not reduce diarrhea, the author successfully used the leaf juice of an Indian When vomiting and diarrhea thus had plant, Coleus aromaticus. substantially controlled, lost fluids/electrolytes were restored successfully without need for intravenous (i.v.) fluids by oral rehydration alone in the 33 mildly dehydrated patients (blood sp. gr. 1.062 or less, and a good pulse); and by oral plus rectal rehydration in the 153 moderately severe cases (blood sp. gr. 1.062-1.064). The solution contained 4 q of sodium chloride and 25 q of glucose in 1.000 of ml water. When a free urine flow had been established. 2 g of notassium chloride was added to each liter of fluid. All 186 patients thus treated recovered without i.v. fluids. This landmark study demonstrated the potential usefulness of non-i.v. glucose/electrolyte solutions for treating cholera. (Also see the author's letter, "Therapy of diarrhoea in cholera", published in Lancet, 14 Nov 1953, pp. 1045-6).

Chen L see Cvjetanovic B

Chen LC, Huq E, Huffman SL. A prospective study of the risk of diarrhoeal diseases according to the nutritional status of children. Am J Epidemiol 1981 Aug;114(2):284-92

The aim of this study was to re-examine the hypothesis that protein-calorie malnutrition increases the incidence of diarrheal diseases. The study was undertaken in Matlab where the International Centre for Diarrhoeal Disease Research, Bangladesh was maintaining a longitudinal demographic-epidemiologic surveillance system and diarrheal disease health services. The prospective epidemiologic field data collected from this area was used to examine the effect of malnutrition on the subsequent risk of diarrhea among preschool children in rural Bangladesh. A total of 2,019 children aged 12-23 months were classified according to weight-for-age, weight-for-height, and height-for-age as a percentage of the Harvard median standard. Over a prospective period of 24 months, diarrheal hospitalization rates among the children were matched to the initial anthropometric assessment. No differences were observed in diarrheal hospitalization rates according to initial nutritional status. Another group of 207 children aged under 5 were classified according to weight-for-age, and after the nutritional assessment, their diarrheal attack rate was followed prospectively for one year. Again, no differences in diarrheal attack rates between varying nutritional status categories were observed. The nutritional status of the 207 children was then defined as monthly growth velocity (kg change in body weight, percent change of initial

body weight, and percent change of weight-for-age) and the diarrheal attack rate for the subsequent one month period was observed, but again no difference in attack rates was noted between nutritional groups. The results of the study suggest that the nutritional status of children exerts little influence on subsequent diarrheal incidence. The impact of diarrhea in predisposing and exacerbating malnutrition might be the more important of the bidirectional interactions. It is further concluded that the predominant effect of malnutrition on diarrhea might be through disease duration and mortality rather than through disease incidence.

Chernoff R. Dean JA. Medical and nutritional aspects of intractable diarrhea. J Am Diet Assoc 1980 Feb;76(2):161-9

Diarrhea can be triggered by at least 5 pathogenetic mechanisms. Major examples of each are presented to better illustrate each type of diarrhea and to provide a background for discussion of the nutritional management of each class of diarrhea. Certainly, specific diseases require specific therapies, such as avoidance of gluten in celiac disease, but the major dietary nutritional alterations and subsequent treatment are similar for each class of disease. The feeding of healthy people as well as the diet of the sick cannot be left to chance, guided by appetite, or ruled by tradition, but can be safely directed only according to the mechanisms of digestion and metabolism. (Modified authors' abstract)

Cheromcha DP, Hyman PE. Neonatal necrotizing enterocolitis: inflammatory bowel disease of the newborn. Dig Dis Sci 1988 Mar;33(suppl 3):78S-84S

"Neonatal necrotizing enterocolitis is the most common serious gastrointestinal disorder encountered in neonatal intensive care units. It is a major cause of morbidity and mortality in the newborn, particularly in premature infants. Consistent risk factors are birth weight and prematurity. Polycythemia and hyperviscosity altering blood flow and infectious agents are also implicated. Clinical findings include abdominal distention and diarrhea, and systemic symptoms such as apnea, acidosis, and lethargy. Pneumatosis intestinalis can be demonstrated radiographically. Mucosal ulcerations, hemorrhage, and thrombosis occur early, followed by inflammatory changes. Later still necrosis develops. Ischemia, infection, and enteral feedings are suspected to be involved in the pathophysiology. Eicosanoids, especially thromboxane, platelet-activating factor, and leukotrienes are likely mediators." (Authors' abstract)

Chiapella AM. Treatment of intestinal disease. Vet Clin North Am [Small Anim Pract] 1983 Aug;13(3):567-84

The Child Day Care Infectious Disease Study Group. Considerations of infectious diseases in day care centers. Pediatr Infect Dis 1985 Mar-Apr;4 (2):124-36

Chiodini RJ. Crohn's disease and the mycobacterioses: a review and comparison of two disease entities. Clin Microbiol Rev 1989 Jan;2(1):90-117

"Crohn's disease is a chronic granulomatous ileocolitis, of unknown etiology, which generally affects the patient during the prime of life. Medical treatment is supportive at best, and patients afflicted with this disorder generally live with chronic pain, in and out of hospitals, throughout their

lives. The disease bears the name of the investigator who convincingly distinguished this disease from intestinal tuberculosis in 1932. distinction was not universally accepted, and the notion of a mycobacterial etiology has never been fully dismissed. Nevertheless, it was 46 years after the distinction of Crohn's disease and intestinal tuberculosis before research attempting to reassociate mycobacteria and Crohn's disease was published. Recently, there has been a surge of interest in the possible association of mycobacteria and Crohn's disease due largely to the isolation of .genetically identical pathogenic Mycobacterium paratuberculosis from several patients with Crohn's disease in the United States, the Netherlands, Australia, and France. These pathogenic organisms have been isolated from only a few patients, and direct evidence for their involvement in the disease process is not clear; however, M. paratuberculosis is an obligate intracellular organism and strict pathogen, which strongly suggests some etiologic role. Immunologic evidence of a mycobacterial etiology, as assessed by humoral immune determinations, has been conflicting, but evaluation of the more relevant cellular immunity has not been performed. Data from histochemical searches for mycobacteria in Crohn's disease tissues have been equally conflicting, with acid-fast bacilli detected in 0 to 35% of patients. Animal model studies have demonstrated the pathogenic we I 1 elucidated the complexity isolates as as potential of mycobacterial-intestinal interactions. Treatment of Crohn's disease patients with antimycobacterial agent has not been fully assessed, although case reports suggest efficacy. The similarities in the pathology, epidemiology, and chemotherapy of Crohn's disease and the mycobacterioses are discussed. issue is fraught with controversy, and the data generated on the association of mycobacteria and Crohn's disease are in their infantile stages so that a general conclusion on the legitimacy of this association cannot be made. While no firm evidence clearly implicates mycobacteria as an etiologic agent of Crohn's disease, the notion is supported by suggestive and circumstantial evidence and a remarkable similarity of Crohn's disease to known mycobacterial diseases." (Author's abstract)

Chipman DC see Trust TJ

Chizhov NP. [Mechanising of the formation of viral resistance to .chemotherapeutic preparations]. Vopr Virusol 1985 May-Jun;30(3):266-79

Choudhuri AB see Mahalanabis D

Choudhuri PK. Acute gastroenteritis in children. J Indian Med Assoc 1987 Jul:85(7):214-5

Chowdhury AMR, D'Souza S. A design and field methods for monitoring impact on mortality of an oral therapy programme. Dhaka: International Centre for Diarrhoeal Disease Research, Bangladesh, 1982. 23 p. (ICDDR,B working paper, 27)

Desiring to set up a low-cost surveillance system that can detect changes in mortality due to diarrhea in the 1 to 4-year age group, a design and field method for monitoring the impact of an oral therapy programme on mortality was presented. The design for studying the impact of oral rehydration on mortality envisaged a double stratification of thana, an administrative unit (one geographic and one on the basis of "famine liability"), a sliding selection of two unions from each strata. A baseline survey in each union, followed by retrospective multi-round surveys in the selected unions, has been planned.

The stratification, determination of an adequate sample size and selection of unions were discussed. For the baseline survey, two sets of questionnaires were developed. Field operations, mapping, interview training, data collection and supervision of field work and data processing are also discussed briefly.

Chowdhury MNH. <u>Campylobacter</u> <u>jejuni</u> enteritis: a review. Trop Geogr Med 1984 Sep;36(3):215-22

Campylobacter jejuni has recently been recognized as an important cause of human gastroenteritis in many countries. The clinical features of \underline{c} . <u>jejuni</u> infections vary from those of mild gastroenteritis to severe enterocolitis. The most common symptoms of the disease are fever, abdominal pain, and bloody diarrhea. The small intestine is the main site of infection, but the colon may also be involved. C. jejuni appears to invade the wall of the gut as in salmonellosis. Isolation of the organism from feces requires culture in a selective medium containing antibiotics and incubation under reduced oxygen tension at 42°C. Most cases of Campylobacter enteritis are sporadic, and it is often difficult to confirm their source. Although cross-infection between humans occurs rarely, the disease is mainly a zoonotic one with many possible Human infections have been associated with routes of infection. consumption of contaminated food, especially poultry, unpasteurized milk, water as well as contact with domestic animals, such as dogs and cats. In most cases, <u>Campylobacter</u> enteritis is a self-limiting disease, and, therefore, decision on treatment should be taken on clinical grounds. When considered necessary, erythromycin is the drug of choice. Information about <u>C. jejuni</u> infection has accumulated rapidly in recent years, but much remains to be learned, especially about its epidemiology. (Modified author's abstract)

Chretien JH, Garagusi VF. Current management of fungal enteritis. Med Clin North Am 1982 May;66(3):675-87

Enteritis, caused by fungal infection, has become more common in recent years. In part, this may be because of improved diagnostic methods and increased survival periods of patients whose underlying disease predisposes to secondary infection with opportunistic fungi. In most patients, however, severe disease that results in altered immunologic status or therapeutic measures that also. alter immunity of the compromised host are not the only reasons for the increased prevalence of fungal enteritis. Numerous factors, such as antibiotic use, hormonal medications, environmental exposure, and travel can lead to infection or overgrowth of the intestinal tract and subsequent symptomatic disease in the otherwise normal host. In this article, fungal infection of the intestine is discussed in 3 broad categories that encompass most cases: (1) noninvasive proliferation of the organism in healthy individuals, (2) invasive disease in healthy individuals, and (3) invasive patients. Fungal infections immunosuppressed gastrointestinal tract have risen to higher levels of prevalence in the past Major factors accounting for this increase are social changes, such as the increased ease and frequency of travel, which exposes the individual to environmental conditions that may result in fungal infection; increasing use of antibiotic and hormonal medications by otherwise healthy persons; and improved therapy for other diseases, such as polychemotherapy of cancer with its immunosuppressive effects. Both noninvasive and invasive fungal disease of the intestinal tract in otherwise healthy individuals can be successfully treated. The invasive fungal infections in patients with severe underlying disease are often first diagnosed postmortem, but improvement in serologic techniques now

offers a possibility of earlier diagnosis and therapeutic intervention.

Christensen ML. Human viral yastroenteritis. Clin Microbiol Rev 1989 Jan;2(1):51-89

"During the last 15 years, several different groups of fastidious viruses—that are responsible for a large proportion of acute viral gastroenteritis cases have been discovered by the electron microscopic examination of stool specimens. This disease is one of the most prevalent and serious clinical syndromes seen around the world, especially in children. Rotaviruses, in the family Reoviridae, and fastidious fecal adenoviruses account for much of the viral gastroenteritis in infants and young children, whereas the small caliciviruses and unclassified astroviruses, and possibly coronaviruses, are responsible for significantly fewer cases overall. addition to electron microscopy, enzyme immunoassays and other rapid antigen detection systems have been developed to detect rotaviruses and fastidious fecal adenoviruses in the stool specimens of both nonhospitalized patients and those hospitalized for dehydration and electrolyte imbalance. rotavirus vaccines have also been developed, due to the prevalence seriousness of rotavirus infection. The small, unclassified Norwalk virus morphologically similar viruses are responsible for large and small outbreaks of acute gastroenteritis in older children, adolescents, and Hospitalization of older patients infected with these viruses is usually not required, and their laboratory diagnoses have been limited primarily to research laboratories." (Author's abstract)

Cichowicz-Emmanuelli E. Chronic non-specific diarrhea of infancy. Bol Asoc Med PR 1982 May-Jun;74(5-6):178-81

Cilli V, Castrucci G. Viral diarrhea of young animals: a review. Comp Immunol Microbiol Infect Dis 1981;4(3-4):229-42

Diarrheas of young animals are caused by protozoa, bacteria, and viruses. Rotaviruses, coronaviruses, parvoviruses and parvovirus-like enteropathogenic strains of $\underbrace{\text{Escherichia coli}}_{\text{in the etiology}}$, and $\underbrace{\text{Salmonella}}_{\text{anomally involved}}$ species, seem to be most commonly involved in the etiology of the condition. This review discusses the enteric viral infections of young animals. The general characteristics of rotaviruses, coronaviruses, and parvoviruses are reported, and the different aspects of the disease's caused by them are highlighted. is conceivable that viruses associated with diarrhea remain latent in the recovered animals, and subsequent stress could reactivate the virus, causing the disease to reappear. Furthermore, the possibility for inter-species infection is an additional point worthy of consideration to understand the mode of spread of the disease. Young pigs might represent a potential source of rotaviral infection for calves. Very little is known about the aftermath in calves after recovery from a viral enteric infection except that they undergo a significant decrease in their normal growth rate. The serious changes which affect the mucosa of the small intestine, principally the virus-induced inhibition of the absorptive mechanism of the epithelial cells, could result in the loss of some essential nutritional factors, required for normal Due to the differences in virulence which exist among strains of rotaviruses. studies on the pathogenesis of the infection should be pursued. Such studies might provide information which could be of value in correcting the altered physiology induced by the disease. The selective nature of viral damage in the intestine has to be considered carefully. In the case of transmissible

gastroenteritis of pigs, the villus epithelium is destroyed, while crypt epithelium is spared and regenerates the villi. In contrast, the rotaviruses infect the most mature absorptive cells which are situated at the median and at the apical parts of the villi. On the other hand, the parvoviruses have a predilection for the immature proliferative cells of the crypts, so that as the disease progresses, the crypts become aplastic and fail to replace the villus-absorptive cells, causing the atrophy of the villi to occur. A better knowledge also is needed of the antigenicity of the enteropathogenic viruses to understand the epidemiology of the infections, and to develop effective vaccines against neonatal diarrheas. Some guidelines for the prevention of the disease are given.

Clark JH see Fitzgerald JF

Clark MA see Barrett EL

Clements ML see Levine MM

Clench MH see Mathias JR

Cline BL. Current drug regimens for the treatment of intestinal helminth infections. Med Clin North Am 1982 May:66 (3):721-42

Cohen ML see MacDonald KL

Cohen S, Lake AM, Mathis RK, Walker WA. Perspectives on chronic nonspecific diarrhea: dietary management. Pediatrics 1978 May;61(5):808-9

This paper reviews the role of dietary intake in the cause and management of chronic nonspecific diarrhea. Observations suggest a role of inadequate dietary fat -- fat delays yastric emptying and slow intestinal transit. The clinical experience indicates that a significant subset of patients classified as having chronic diarrhea have symptoms that are dietarily, often iatrogenically, induced. A complete dietary history, with an otherwise normal history and results of physical examination, may help the pediatrician to determine those patients who may benefit from increased quantities of dietary fat (preferably polyunsaturated) to slow intestinal transit time and treat the diarrhea.

Col NF, O'Connor RW. Estimating worldwide current antibiotic usage: report of Task Force 1. Rev Infect Dis 1987 May-Jun;9(suppl 3):S232-43

Colwell RR see Joseph SW

Coman G see Alexandrescu M

Cook GC. The clinical significance of gastrointestinal helminths - a review. Trans R Soc Trop Med Hyg 1986;80(5):675-85

Gastrointestinal helminths (nematodes, trematodes, and cestodes) constitute some of the most common and important infective agents in humans. Whereas many symptoms and signs are confined to the intestine and less often to the associated digestive organs, systemic manifestations are also numerous. Using a clinical classification, these organisms can be broadly separated into those involving the small intestine and those which have a colo-rectal distribution;

of the former, a minority has been causally related to intestinal malabsorption. Not all gastrointestinal helminths are associated with disease, and it is important to be able to separate these two groups; when present at high concentration and especially in infants and children some of the least pathogenic are not, however, entirely asymptomatic. Maintenance of a high index of suspicion is necessary, and this applies especially to 'western' populations' in whom rapid and extensive travel to areas of the world with substandard sanitation and contaminated food and water supplies is common; first evidence of infection in them may result from serious clinical complications. Recent advances have focussed on treatment with benzimidazole compounds (especially albendazole) for nematode and praziquantel for cestode infections. Treatment of strongyloidias still remains unsatisfactory. Mass elimination of gastrointestinal helminths in developing 'Third World' countries remains a major challenge. (Modified author's abstract)

Cooke EM. Escherichia coli - an overview. J Hyg (Lond) 1985 Dec;95(3):523-30

Cooper BT. Diarrhoea as a symptom. Clin Gastroenterol 1985 Jul:14(3):599-613

Diarrheal diseases, particularly of infectious etiplogy, are seen worldwide. causing much morbidity and mortality, especially amonyst children. Although the symptom is almost universally understood, diarrhea is difficult to define because of the wide variation in the bowel habits of normal individuals. Diarrhea is, perhaps, best described as a change in bowel habit from normal with an increase in stool volume and/or fluidity with or without an increase in stool frequency. The disorders causing diarrhea are many and various, but most episodes of diarrhea are mild and self-limiting. However, all cases of chronic diarrhea and all severe cases of acute diarrhea require investigation after the initial clinical assessment. Investigation must be logical and structured can be divided into 3 stages: the initial workup (sigmoidoscopy, stool examination, screening blood tests), anatomical and functional assessment of the gastrointestinal tract, and further, investigation of the difficult case (osmotic or secretory diarrhea, hormone levels, tests for laxative abuse, perfusion studies, laparotomy, etc.). Most cases are diagnosed after clinical assessment or the initial workup. In the remainder, there are clues to the diagnosis or to the area of the gastrointestinal tract which needs to be investigated. Only a small number of cases require extensive investigation including the third stage of workup. Analysis of the symptom of diarrhea requires all the attributes of the good physician: wide clinical experience, careful history and examination, diagnostic and therapeutic acumen, a sound understanding of normal and abnormal physiology, skill and experience in selecting the appropriate investigations and interpreting their results, meticulous attention to detail, and finally, a caring and sympathetic attitude to the patient. (Modified author's abstract)

Coppa GV see Giorgi PL

Corazza GR, Gasbarrini G. Defective splenic function and its relation to bowel disease. Clin Gastroenterol 1983 Sep;12(3):651-69

Cornelis G, Laroche Y, Balligand G, Sory M-P, Wauters G. <u>Yersinia enterocolitica</u>, a primary model for bacterial invasiveness. Rev Infect Dis 1987 Jan-Feb; 9(1):64-87

"Yersinia enterocolitica is now the species of Yersinia most frequently isolated from human and animal infections. The species include pathogens and

ubiquitous strains. Among the human pathogens, those isolated in America are more virulent than those isolated elsewhere, especially in Europe and Japan, and these isolates differ biochemically and serologically. between Y. enterocolitica and Y. pestis only became obvious in 1980 with the discovery that at 37°C Y. enterocolitica requires Ca++, a phenotype described in the 1960s for Y. pestis. This requirement as well as virulence is dependent on a 70-kilobase plasmid found later in \underline{Y} . pseudotuberculosis and \underline{Y} . pestis. Thus, many bacteriologists elected Y. enterocolitica as a model for bacterial invasiveness. However, studies with non-American strains were impeded by the lack of an inexpensive, simple animal test, a difficulty now circumvented by supplying an appropriate siderophore to the bacteria. Catt dependence can be viewed as a transition between free growth and protection against the immune system. In the latter phase, Y. enterocolitica synthesizes and releases large amounts of 6 plasmid-encoded outer-membrane proteins. Most of these are under the control of the plasmid region governing Ca++ dependence. Mutants in this region either lose the Ca++ requirement at 37°C or become unable to grow at 37°C irrespective of the Ca++ concentration. The complex events leading to Ca++ dependence is still not understood. Virulence in Y. enterocolitica also depends on chromosomal genes: the endocytosis in intestinal epithelial cells seems not to be encoded by the pYV plasmid. Studies of \underline{Y} , \underline{p} pseudotuberculosis suggest that this property depends on a single chromosomal locus, the study of which might be particularly important in the understanding of the first step in infection." (Authors' abstract)

Coupar IM. Opioid action of the intestine: the importance of the intestinal mucosa. Life Sci 1987 Aug 24;41(8):917-25

"Drug effects on the intestine are traditionally explained in terms of action on the muscle layers and the nerves that control them. This is particularly true in the case of the opioids but research starting two decades ago has identified the intestinal mucosa as the site of action of the antidiarrheal opioids. Continued research using the intestinal mucosa offers a fresh approach to solving some old problems. For example it could lead to more confident predictions to be made about the wanted and unwanted effects of opioid drugs on the intestine and may help to find better drug treatments for alleviating withdrawal diarrhoea in addicts. Eventually it may help to explain how the general process of opioid dependence occurs at a cellular level." (Author's abstract)

Craun GF. A summary of waterborne illness transmitted through contaminated groundwater. J Environ Health 1985 Nov-Dec;48(3):122-7

This report is a summary of waterborne illnesses transmitted through contaminated ground water occurring in the USA from 1971 to 1982. Three hundred and ninety-nine waterborne outbreaks and 86,050 cases of waterborne diseases were reported during this period. Two hundred and four outbreaks (51%) and 34,337 cases of illness (40%) were caused by contaminated ground water. Contaminated, untreated, or inadequately disinfected ground water caused 65% of the waterborne outbreaks and 66% of the waterborne illnesses, which occurred in noncommunity and individual water systems compared to only 32% of the outbreaks and 31% of the illnesses in community water systems. Illnesses most frequently transmitted through ground water included acute gastroenteritis of undetermined etiology, chemical poisoning, hepatitis A, shigellosis and viral gastroenteritis. Waterborne outbreaks in water systems using untreated well water were caused primarily by the overflow or seepage of

sewerage from septic tanks or cesspools, chemical contamination and surface run-off contamination. An increase in the number of outbreaks resulting from the use of untreated, contaminated well water was noted during the summer months.

Creamer B. The small intestine: a review. Pahlavi Med J 1978 Jan;9(1):50-103

Creese AL. Cost effectiveness of potential immunization interventions against diarrhoeal disease. Soc Sci Med 1986:23(3):231-40

Estimates are made of the costs per death averted and the costs per case prevented by three possible immunization interventions against diarrheal disease in children. These estimates are based on cost information collected from a number of ongoing national immunization programs and from effectiveness estimates reported in previously published reviews. The first part of the paper reviews the state of current knowledge regarding immunization costs and converts data from 9 different studies into a common set of price equivalents. The second section assesses the composition of typical immunization program costs and estimates the likely effect on existing costs of introducing new vaccines. Compatibility between existing EPI activity and the administration schedule of the new vaccine is likely to be a major determinant of increments in cost per fully immunized child. The third section brings together the cost information with estimates of the likely impact of measles, rotavirus and new cholera vaccines on mortality and morbidity from diarrhea.

Cremaschi RE see Dionigi R

Crompton DWT. Nutritional aspects of infection. Trans R Soc Trop Med Hyg 1986;80(5):697-705

Current knowledge on ascariasis, hookworm disease, strongyloidiasis, and trichuriasis and their contribution in human malnutrition are depicted. Results from experiments with related parasites in the laboratory have demonstrated the role of gastrointestinal helminthiases in animal malnutrition. Some evidence shows that, in children, infection with the intestinal stages of Ascaris lumbricoides is associated with reduced growth rate, disturbed nitrogen balance, malabsorption of vitamin A, abnormal fat digestion, lactose maldigestion, and an increased intestinal transit time. The main impact of hookworm infection is its relationship with iron deficiency anemia which may have effects at the community level in terms of work and productivity in adults and learning and school performance in children. More research is needed to extend knowledge of the nutritional impact of ascariasis and hookworm disease to establish their public health significance. Research is needed also to identify the range of nutritional effects on man that occur as a result of trichuriasis and strongyloidiasis. The significance of less prevalent and more localized gastrointestinal helminthiases should not be ignored. (Modified author's abstract)

Cronin A see Feachem RG

Cross R see Feachem RG

Cucchiara S see Auricchio S

Cukor G see Blacklow NR

Cunha BA. The toxigenic diarrheas. Intern Med 1987 Feb;8(2):92-110

"The toxigenic diarrheas affect thousands of people each year and, although not usually fatal, are responsible for much morbidity. These illnesses are caused by a variety of microorganisms that produce toxins. The majority of the microorganisms responsible for toxigenic diarrheas are foodborne and are ingested by the affected individual. The microorganism and its preformed toxin may be ingested in the contaminated food item, or the organism may produce toxins in vivo." (Author's abstract)

Cunha BA see Schoch PE

Curry A see Casemore DP

Curtis U see Feachem RG

Cusolito S see Donowitz M

Cutting WAM, Hawkins P. The role of water in relation to diarrhoeal disease. J Trop Med Hyg 1982 Feb;85(1):31-9

Behavioral and social factors are important in respect of the water use for washing, and more relevant when considering the use of excreta disposal facilities. Obviously cultural, educational and economic considerations are closely related and important too. No community has reduced diarrhea to a minor health problem without having adequate systems for sewage disposal, food hygiene and health education as well as adequate water supplies. A whole package of inputs is required if better water is to benefit the community. Increased usage of water probably requires behavioral changes which are very difficult to introduce once an individual and family have adopted a particular pattern of bathing and water use. These changes cannot occur until their social and economic determinants change. Improvements in the water quality, the quantity used and the mode of supply are unlikely to have beneficial effects unless they are part of a larger package of social and economic improvements.

Cutting WAM see Booth IW

Cvjetanovic B. Epidemiological models of diarrhoeal diseases [editorial]. J Diarrhoeal Dis Res 1985 Jun;3(2):63-4

Cvjetanovic B. Health effects and impact of water supply and sanitation. World Health Stat Q 1986;39(1):105-17

A review on selected aspects of the health benefits of water supply and sanitation, and analyses of the concepts, methodologies and interpretations of the results of studies on health effects are included in this paper. The findings of various studies point to the variety of local factors, which make some water supply and/or sanitation projects more (or less) effective than others in the control of specific diseases. However, the provision of water supply alone is considerably less effective than when coupled with health education programs. It is concluded that the impact of water supply and sanitation on health depends on the quality and quantity of water supply and the type of sanitation system, the proportion of the population covered, and the utilization of available water and sanitation facilities by the population.

Water supply and sanitation have proved to be a cost-effective strategy in the control of enteric and diarrheal diseases.

Cvjetanovic B, Chen L, Kronmall R, Rohde C, Suskind R. Measuring and evaluating diarrhea and malabsorption in association with village water supply and sanitation: a review of the Food Wastage/Sanitation Cost Benefit Methodology Project (Guatemala). Arlington, Virginia: Water and Sanitation for Health Project, 1981. 36 p. (WASH technical report, 12)

Cyjetanovic B. Sanitation versus immunization in control of enteric and diarrhoeal diseases. Prog Water Technol 1979;11(1-2):81-7

Improved sanitation systems and immunization are often introduced as control methods in places endemic with diarrheal diseases and other similar illnesses. They have certain advantages and drawbacks in specific epidemiological circumstances. The decision-makers have to adopt an appropriate control strategy for matching prevailing circumstances to obtain the highest health benefits consistent with the efforts and resources invested. presents the relative effectiveness, costs and benefits of sanitation and immunization programs, referring particularly to the epidemiological models. The effect of immunization is temporary, disease specific and limited to enteric fever and cholera. Immunization implies, from an economic point of view, the provision of consumable commodity, while sanitation, such provision of potable water and safe excreta disposal, is capital investment permanent value. Sanitation covers all diseases related to the use of dirty Investment on sanitation has a cumulative effect as hands and poor hygiene. more resources are allocated to it. However, proper and extensive use and good maintenance are necessary to obtain the best possible health benefits from investments in sanitary facilities. This again requires some investment in health education and possibly some other items, such as drainage of used water, provision of soap, etc. The simple methods as well as complex ones, such as mathematical models, are available and should be used for the evaluation of alternative strategies for control of enteric infections based on sanitation or vaccination. Studies carried out in developing countries indicate higher cost-benefit value for sanitation than vaccination in control of enteric infections. Thus, sanitation is to be considered always as a method of choice unless there is evidence that immunization for some reason would prove more effective, e.g. in travelers and other special high risk groups.

D'Antonio AM see Auricchio S

D'Aoust J-Y. Recent developments in <u>Salmonella</u> epidemiology and methodology. Food Lab Newslett 1987 Apr; (9):32-6

Recent developments in <u>Salmonella</u> epidemiology and methodology are reviewed. Recent epidemiological reports indicate continued increases in the incidence of human salmonellosis. Clinical symptoms of salmonellosis in humans may include diarrhea, fever, nausea, vomiting, and prostration. Treatment of acute infection with no evidence of systemic involvement is limited to supportive therapy and fluid replacement. Antibiotic therapy in noncomplicated cases of enteritis is contraindicated as it tends to prolong the carrier state. Chloramphenicol, ampicillin, and cotrimoxazole, and more recently quinolones and cephalosporins have been used successfully for the treatment of systemic infections. Significant advances have been made in the development of prophylactics. Administration of serotypic (somatic) oligosaccharides from

Salmonella typhimurium coupled with a carrier protein was found immunogenic to the same serovar in mice. Application of such technology to human populations shows great promise. Salmonella continues to rank first amony bacterial agents of foodborne disease; poultry and raw meat constitutes the principal vehicles of human infection. Holding of foods at growth-permissive temperatures ranks highest in the list of common faults in food preparation and handling. Detection of Salmonella in foods relies on culture methods that generally include enrichment in a nonselective medium (pre-enrichment), followed by enrichment in a selective broth medium, and isolation on differential agar media. The choice of enrichment media and incubation temperature is critical for the successful recovery of Salmonella from foods. Recently, biotechnology has facilitated the development of novel procedures for the detection of Salmonella. The advent of immunoenzyme assay procedures and deoxyribonucleic acid probe technology are important developments. The search for simple, sensitive, and cost-efficient analytical methods is most challenging and needs to be encouraged.

d'Hauteville H see Sansonetti PJ

D'Souza S see Chowdhury AMR

Dabral M see Saran M

Danhof IE. Pharmacology, toxicology, clinical efficacy, and adverse effects of calcium polycarbophil, and enteral hydrosorptive agent. Pharmacotherapy 1982 Jan-Feb;2(1):18-28

Calcium polycarbophil is the calcium salt of polyacrylic acid cross-linked with divinyl glycol. In dilute alkali, it possesses marked hydrophilic capacity (60 to 100 times its weight), which is the basis for its therapeutic use. In daily dosages of 4 to 5 g in adults, it appears to be quite safe, is nontoxic, does not interfere with digestion of absorption, and does not cause gastrointestinal irritation. It appears to be effective in the treatment of both constipation and diarrhea due to functional or organic causes. Continuous use is necessary before effectiveness becomes apparent. Clinical studies, of which there are relatively few, range from uncontrolled, unblinded evaluations of an almost anecdotal nature to well-controlled, double-blind, cross-over Additional carefully controlled studies on dietary influences, exercise, and patient compliance would be helpful. Adverse effects include epigastric fullness or heaviness, abdominal distention and bloating, and flatulence. with all bulk-forming agents, calcium polycarbophil should not be used by persons who have stenotic lesions of the gastrointestinal tract. (Modified author's abstract)

Darling P see Leung AKC

da Rocha JM. [Disaccharidase disorders]. Bol Inst Puericult 1963 Apr;20(1): 311-22

Literature on disaccharide-hydrolyzing enzyme deficiencies is reviewed. In addition to observations on clinical symptoms - weight standstill, foamy diarrhea, acid stools - disaccharide-loading tests are suggested for diagnosis. For definitive diagnosis and to exclude alimentary allergy, enzymatic activity is searched for in a fragment of intestinal mucosa obtained by oral biopsy. The helminths affect the integrity of intestinal cell wall, localized in the

brush borders of the villus. Thus, all helminthiases accompanied by diarrhea, principally strongyloidiasis, contribute to malabsorption, including disaccharide malabsorption.

Darrow DC, Pratt EL, Flett J, Jr., Gamble AH, Wiess HF. Disturbances of water and electrolytes in infantile diarrhea. Pediatrics 1949 Feb;3(2):129-56

This classic study presents data and concepts derived from metabolic balances on 7 patients with moderate to severe diarrhea. The magnitude of the deficits of H₂O, Cl. Na, K. and P present at the onset of treatment is described. study confirms and amplifies the evidence demonstrated in a previous study that the loss of body K in excess of the loss of body N is a striking feature of infantile diarrhea. Average deficits per kg in severe dehydration due to diarrhea are H₂O, 125 g; Cl, 9.2 mM; Na, 9.5 mM; and K, 10.4 mM. These figures may be about 10% higher for $extsf{H}_2 extsf{U}_2$, $extsf{N}_a$, and $extsf{Cl}$ because of overexpansion of extracellular fluids in some cases. These findings, especially approximately equal retentions of Na and Cl during recovery, indicate: (1) that intracellular Na is slightly high during dehydration in many cases; (2) that NaCl without NaHCO₃ should suffice to restore all deficits of Na and Cl; (3) that acidosis is explained chiefly by deficit of K which leads to transfer extracellular Na into the cells during dehydration; (4) that fluids used to replace deficits of H₂O and electrolyte in diarrhea should contain K as well as Na and CI; (5) that administration of NaHCO $_3$ without potassium salts so as to restore rapidly the concentration of serum HCO $_3$ in acidosis is likely to aggravate the abnormality in cellular composition and may produce alkalosis and low concentration of K in serum. Losses of H₂O as sweat are frequently 50 ml/kg.day; the maximum measured was 100 ml/kg.day. Losses of H_2 0 in stools are frequently 50 ml/kg.day; maximum measured was 85 ml/kg.day. Nitrogen losses are rapidly replaced when milk feedings are instituted even at low-caloric intakes. In the development of dehydration, losses of water in sweat are quantitatively as important as losses in stools. Relation of these findings to treatment of infantile diarrhea is discussed. (Modified authors' abstract)

Darrow DC, Pratt EL. Fluid therapy: relation to tissue composition and the expenditure of water and electrolyte. JAMA 1950 May 27:143(4):365-73

Darrow DC, Pratt EL. Fluid therapy: relation to tissue composition and the expenditure of water and electrolyte. JAMA 1950 Jun 3;143(5):432-9

Datta T. Intestinal amoebiasis and giardiasis in children. Indian J Pediatr $1985\ Mar-Apr; 52(415):184-5$

Giardiasis and amebiasis are widespread in the pediatric community of Calcutta and also in other regions of India. Accurate management of these patients is not yet possible, since several issues are not clear. Presence of one or more cysts in every high power field in microscopical stool examination is associated with symptoms, though a single stool examination offers no conclusive evidence, and at least 3 consecutive examinations are necessary. Other methods of diagnosis are not commonly available. To curb the incidence and spread of parasite-caused illness in the family or in the community, all possible carriers of cysts (Giardia or Entamoeba histolytica), symptomatic or asymptomatic, should be treated with easily available and low-cost drugs. Metronidazole, given at a dose of 20 mg/kg.day for giardiasis and diiodohydroxyquin at 40 mg/kg.day for amebiasis, in 3 divided doses orally for 10 days, are quite useful. Metronidazole-benzoate preparations are not as

effective. A child may be declared free of organisms if 3 serial samples of stool 2 to 3 weeks after the treatment do not show cysts or trophozoites. This is a difficult proposition in practice. If stool examination cannot be done, a second course of treatment may be given after 2 to 3 weeks. In chronic cases, the growth and development of child may be hampered. Some hygienic sense should be evoked in the people of the community by the authorities and community leaders.

Davidson GP. Viral diarrhoea. Clim Gastroenterol 1986 Jan:15(1):39-53

"It is apparent from this review that great progress has been made over the past 10 years in defining the aetiology of viral diarrhoea. Rotavirus is a major cause of gastroenteritis in children, particularly during the winter months. However, if bacteriological and virological data are pooled, our current aetiological knowledge reveals that a pathogen is not detected in 20 to 30% of cases in most perennial investigations. Now that human rotavirus has been cultured, complete characterization may be possible. However, practical methods for cultivating many of the other possible viral pathogens are needed before they can be characterized completely. Meanwhile, electron microscopy, although time-consuming, cumbersome and expensive, is the only method for detecting many of the other potential viral pathogens. We have much still to learn about the epidemiology of these agents, particularly in the developing countries, their importance in causing chronic diarrhoea, how they are transmitted, and the immune responses to infection. The development of a potential rotavirus vaccine is exciting and creates the possibility of control for this devastating disease." (Author's abstract)

Dean JA see Chernoff R

DeGirolami PC, Dunn JC, Federman M. Infections caused by intestinal protozoa. Pathol Annu 1985;20(pt 2):463-505

This chapter updates the information available concerning the epidemiology and pathogenesis of the major intestinal infections and describes the advantages and limitations of common laboratory techniques which have been developed to By far the most significant of the intestinal amoebae, aid diagnosis. Entamoeba histolytica exhibits considerable variability, both morphologic and a pathogenetic point of view. The presence of a specific isoenzyme, phosphoglucomutase, has been considered a marker for pathogenicity of the organism. Many other factors have been reported to affect virulence, Trophozoites namely diet, intestinal bacterial flora, and associated viruses. of E. histolytica are capable of damaging and killing target cells in vitro. An antiamebic vaccine is being developed and has been tested successfully in monkeys. The reported prevalence of amebiasis has remained relatively stable for over 20 years in the USA. The infection is transmitted most often by ingestion of cysts. Infection is usually acquired by the fecal-oral route in the USA, and by ingestion of sewage contaminated water or contaminated food While microscopic examination of stool or material the tropical regions. obtained by sigmoidoscopy is the most cost-effective diagnostic method, is employed occasionally, especially where parasitological examination are not readily available. Luminal active drugs, e.g. diloxanide furoate or diiodohydroxyquin, should be used to treat asymptomatic cyst-passers. Metronidazole, a tissue-active drug, should be employed in cases of acute amebic dysentery or moderately symptomatic nondysenteric colitis and should be followed by a luminal acting drug to ensure

cyst eradication. Giardia lamblia is a widely distributed protozoan, capable of producing various gastrointestinal sighs and symptoms in humans. It is the most frequently identified pathogen in the outbreaks of waterborne diseases in the USA. The parasite is adaptable to sharply different climates. are common carriers of the parasite and are symptomatic more often than adults. Patients with immunodeficiency syndromes have a high incidence of giardiasis associated with malabsorption. Both humoral and cellular immunity are thought to play an important role in the defense against the parasite and in the containment of infection. Clinical presentations range from complete absence of signs and symptoms to acute infection with the sudden onset of explosive foul-smelling watery diarrhea or to subacute and chronic infection. All 3 types of microscopic examination of stools--direct wet smears, wet smears of concentrate, and permanently stained smears—are valuable. Quinacrine, metronidazole, and furazolidone are the drugs most often used to treat giardiasis in the USA. Minor intestinal protozoa, e.g. E. polecki, Dientamoeba fragilis, coccidia, Cryptosporidium, and Blastocystis hominis, are focussed in this work.

de Graaf FK see Mooi FR

Degryse A see Doms L

Dehaen F see Butzler JP

Dekeyser P see Butzler JP

de Louvois J. Necrotising enterocolitis. J Hosp Infect 1986 Jan;7(1):4-12

De Ritis G see Auricchio S

Derryberry M. Health education aspects of sanitation programmes in rural areas and small communities. Bull WHO 1954;10(2):145-54

In large population centers, the sanitarian can effect the environmental changes needed without necessarily gaining the widespread participation or understanding of the people who are to benefit. In villages and rural areas, however, this is not so, since the people themselves will have to perform many of the actions needed to break the chain of transmission of disease. sanitarian, to be successful, must, therefore, apply the sciences of human behavior in any attempt to carry out environmental improvements. Before any educational program for environmental sanitation can be planned, it is necessary to obtain the essential facts about the people of the community. In the actual planning, the sanitarian must consider how to enlist the participation of the people, what decisions can be left to the people themselves, what informational materials are likely to be needed and how they are to be used, and what the criteria of progress are to be. If all these questions are satisfactorily answered, the sanitarian can assist the people to accept responsibility for their own betterment.

Desai AG. Diarrhoeal disorders in childhood. Q Med Rev 1979 Jul;30(3):1-3:

Various aspects of childhood diarrhea are: discussed with particular emphasis on etiology, pathophysiology, laboratory investigation, and management. Acute diarrhea has been classified as: (1) infective (bacterial, viral, parasitic, and fungal); (2) allergic; (3) food intolerance; (4) endocrinal; (5) emotional; and (6) iatrogenic. Chronic diarrheas in children have well-established disease patterns, such as: (1) parasitic (amebiasis and giardiasis); (2) malabsorption syndromes; (3) inflammatory bowel disease, including ulcerative colitis; (4) fibrocystic disease of the pancreas; (5) lactose intolerance; (6) congenital chloridorrhea; (7) milk allergy; (8) abdominal tuberculosis; (9) liver cirrhosis; and (10) immunological deficiency. Pathophysiology, clinical approach, and laboratory investigations, including microscopy and stool culture, are outlined. The use of rehydration fluids, chemotherapeutic agents, and diets and rehabilitation in the management of childhood diarrhea are discussed. The article also presents a brief account of some important newer entities, such as milk allergy, disaccharidase deficiency, congenital chloridorrhea, necrotizing enterocolitis, and pseudomembranous enterocolitis and their treatment.

Desai M see Kinoti SN

Detrain M see Butzler JP

Development of vaccines against shigellosis: Memorandum from a WHO meeting. Bull WHO 1987;65(1):17-25

Endemic shigellosis is a worldwide problem, with a high morbidity rate in most developing countries; substantial mortality may also occur, especially with disease caused by <u>Shigella dysenteriae</u> serotype 1. The limited efficacy of current measures to control this infection makes the development of vaccines for the prevention of shigellosis particularly important. This Memorandum describes the clinical features of and immunity to shigellosis, and summarizes the present status of efforts to develop suitable vaccines for shigellosis. This report also lists the topics that should be given priority in research. It is hoped that these recommendations will serve to stimulate further research which will ultimately lead to the development of efficacious anti-shigellosis vaccines. (Modified abstract)

De Vizia B see Auricchio S

de Zoysa I, Feachem RG. Interventions for the control of diarrhoeal diseases among young children: chemoprophylaxis. Bull WHO 1985;63(2):295-315

Dharmsathaphorn K see Gertler S

Diamond LS. Amebiasis: nutritional implications. Rev Infect Dis 1982 Jul-Aug;4(4):843-50

"Studies on the role of nutrition in amebiasis in humans and experimental animals are meager. Some reports suggest that malnutrition of the host increases the incidence of infection and potentiates the severity of the disease. Others suggest that malnutrition protects the host against invasion. A few reports indicate that dietary regimens can alleviate symptoms and even eradicate the parasite. Others doubt a correlation between diet and rate of infection or disease manifestations. The problem is complex because the ameba is influenced by its own diet, which in turn depends on the host's diet, the bacterial flora of the gut, and coexisting infections. The host is variously altered by dietary depletions and supplementations, which affect susceptibility and resistance, and by the presence of other disease conditions. Carefully designed and executed studies of infections in humans and experimental animals,

combined with studies <u>in vitro</u> of the nutritional requirements and physiology of the parasite, are needed for definition of the influence of host nutrition in amebiasis." (Author's abstract)

Diarrhea and malabsorption associated with the acquired immunodeficiency syndrome (AIDS). Nutr Rev 1985 Aug;43(8):235-7

Diarrhoea. Clin Gastroenterol 1986 Jul; 15(3): 477-744

DiJoseph JF, Taylor JA, Mir GN. Alpha+2 receptors in the gastrointestinal system: a new therapeutic approach. Life Sci 1984 Sep 3;35(10):1031-42

Dionigi R, Cremaschi RE, Jemos V, Dominioni L, Monico R. Nutritional assessment and severity of illness classification systems: a critical review on their clinical relevance. World J Surg 1986 Feb;10(1):2-11

"Several studies have attempted to define nutritional parameters that can be used to select undernourished hospitalized patients and identify those who present higher risk of post-operative septic complications. Nevertheless, the majority of these studies do not take into consideration the severity of infectious episodes. Systems for scoring the severity of illness are of critical importance in hospital practice and clinical research. In fact, they could be used for the following purposes: (a) a more correct stratification of patients in clinical studies; (b) monitoring the evolution of the infectious complication; (c) prediction of survival; (d) optimization of the criteria for admission to and discharge from intensive care units; and (e) evaluation of quality, quantity, and costs of nutritional therapy in critically ill patients. In this article, the authors present a critical review of the clinical relevance of the methods of nutritional assessment more commonly used in clinical practice; and, moreover they discuss the more recently proposed systems for scoring the severity of illness." (Authors' abstract)

Dobbins JW, Binder HJ. Pathophysiology of diarrhoea: alterations in fluid and electrolyte transport. Clin Gastroenterol 1981 Sep;10(3):605-25

This article discusses the alterations of fluid and electrolyte movement during diarrhea in humans and focuses on the mechanisms responsible for changes in solute movement. Increased fecal solute excretion can result from decreased absorption of solute, increased secretion of electrolytes, or a combination of the two. Decreased absorption can result from (a) ingestion of unabsorbable solutes, (b) loss of brush-border engymes or absorptive cells, or inhibition of absorption. The important intracellular regulators of transport are cyclic AMP, cyclic GMP, and calcium. Calcium may be the ultimate regulator, stimulating both absorption and secretion - secretagogues working by increasing cytosolic calcium activity and agents that stimulate absorption (absorptogogues) working decreasing cytosolic by calcium activity. Secretagogues have been divided into 3 broad categories: (1) bacterial enterotoxins, (2) hormones, and (3) detergents. Glucose stimulates fluid and electrolyte absorption. Several other agents have recently been described to stimulate absorption; these include glucocorticoids, adrenaline (epinephrine), somatostatin, enkephalins, and other opiates. Secretagogus, through their intracellular mediators, affect fluid and electrolyte transport by inhibiting Na+-coupled Cl $^-$ absorption and stimulating active Cl $^-$ (and HCO $_3^-$) secretion: absorptagogues may have the opposite effect. An understanding of these events has led to a much greater insight into the mechanisms of diarrhea and the rationale behind current therapy.

Doe WF. Immunodeficiency and the gastrointestinal tract. Clin Gastroenterol 1983 Sep;12(3):839-53

Doe WF, Hapel AJ. Intestinal immunity and malabsorption. Clin Gastroenterol 1983 May;12(2):415-35

Does malnutrition predispose children to diarrhoea? Nutr Rev 1985 May;43(5): 144-5

Dominioni L see Dionigi R

Donohue-Rolfe A see Keusch GT

Donowitz M, Wicks J, Sharp GWG. Drug therapy for diarrheal diseases: a look ahead. Rev Infect Dis 1986 May-Jun;8(suppl 2):5188-201

"The gastrointestinal tract is involved in both absorption and secretion of electrolytes and water, with absorption as the predominant process. In diarrheal diseases, this balance is disturbed, and the result is net secretion. Most of the drugs used for the treatment of diarrhea at least partially act by (1) stimulating absorption only, (2) both stimulating absorption and inhibiting secretion, or (3) inhibiting secretion only. The therapeutic usefulness of an antidiarrheal agent depends on how efficiently it alters secretion and/or absorption and on how few systemic adverse reactions it causes. When more information on the regulation of absorption and secretion has been accumulated, it may be possible to develop new drugs that can be aimed directly at these processes." (Authors' abstract)

Donowitz M, Wicks J, Cusolito S, Sharp GW. Pharmacotherapy of diarrheal diseases: an approach based on physiologic principles. KROC Found Ser 1984; 17:329-59

Donowitz M see Keusch GT

Donrov N. Thymic atrophy and immune deficiency in malnutrition. Curr Top Pathol 1986;75:127-50

Dorner F see Finkelstein RA

Dougan G, Hormaeche CE, Maskell DJ. Live oral <u>Salmonella</u> vaccines: potential use of attenuated strains as carriers of heterologous antigens to the immune system. Parasite Immunol 1987 Mar;9(2):151-60

"Live attenuated strains of <u>Salmonellae</u> are showing promise as live oral vaccines against human typhoid fever and other <u>Salmonella</u> infections of man and animals. Attenuation can be achieved by introducing genetically defined, nonreverting mutations into specific genes on the <u>Salmonella</u> chromosome. Mutations in the gale or aroA genes of <u>Salmonella</u> inhibit the ability of the bacteria to grow in <u>vivo</u>, and strains carrying such lesions are effective vaccines against <u>Salmonellosis</u>. Genetic determinants encoding for the expression of potentially protective antigens from heterologous, non-<u>Salmonella</u> pathogens can be readily introduced into these attenuated <u>Salmonella</u> strains. Expression of the heterologous antigen does not affect the ability of the <u>Salmonella</u> host to be used as a <u>Salmonella</u> vaccine. Mice infected orally with a <u>Salmonella</u> typhimurium aroA vaccine expressing the <u>Escherichia</u> coli

heat-labile toxin B subunit developed both a secretory and serum antibody response to this antigen. These serum antibodies were able to neutralise the activity of \underline{E} , coli heat-labile toxin in tissue culture assays. A humoral and cell-mediated ($\overline{\text{DTH}}$) immune response was detected against beta galactosidase, an intracellular antigen, in mice infected with an $\underline{\text{aroA}}$ vaccine expressing this cloned antigen. The prospects for the development of live $\underline{\text{Salmonella}}$ vaccines as a method for delivering heterologous antigens derived from bacteria, viruses and parasites is discussed." (Authors' abstract)

Drachman RH. Acute infectious gastroenteritis. Pediatr Clin North Am 1974 Aug;21(3):711-37

Drasar B see Feachem R

Drossman DA, Lowman BC. Irritable bowel syndrome: epidemiology, diagnosis and treatment. Clin Gastroenterol 1985 Jul:14(3):559-73

The physiological, epidemiological and clinical aspects of irritable bowel syndrome are examined to provide the physician with a more confident approach for diagnosis and patient care. Irritable bowel syndrome is a disorder of bowel motility, and the major clinical feature is abdominal pain associated with constipation, diarrhea, or alternating constipation and diarrhea. The disorder is extremely common and exacts a considerable economic toll on patients and the health-care system. Patients with irritable bowel syndrome are more often young and are females. The association of stress, emotion, personality with bowel dysfunction is based on clinical observations numerous studies. The physician can make a confident diagnosis primarily from the history, physical examinations, and a few simple diagnostic studies. the diagnosis is fairly well established, treatment should be directed toward amelioration of symptoms, identification, and modification of factors that aggravate the disorder, and helping the patient to adapt to his or her condition. Foods that stimulate bowel action (caffeinated beverages) or produce increased intestinal gas (beans, cabbage) should be omitted from the diet. Patients with lactose deficiency should avoid milk and milk products. Anticholinergic drugs are recommended for patients with predominant pain and Antidepressants may help patients whose symptoms include depression. Baths, hot water bottles, exercise, and periods of rest often benefit an irritable bowel syndrome patient. Recently, relaxation techniques, biofeedback, and behavioral modification have also been used to treat irritable bowel syndrome cases.

Ducrotte P see Weber J

Dunn JC see DeGirolami PC

DuPont HL, Ericsson CD, Johnson PC, Cabada FJ. Antimicrobial agents in the prevention of travelers' diarrhea. Rev Infect Dis 1986 May-Jun;8(suppl 2): S167-71

"Each of 433 adults traveling to Guadalajara, Mexico, from the United States during summer months was enrolled in one of four clinical trials of the protective effect of antimicrobial agents against travelers' diarrhea. Only one (2%) of 57 subjects taking trimethophim-sulfamethoxazole (160 mg/800 \cdot mg daily) experienced diarrhea during a 2-week study, whereas 8 (14%) of 58 subjects taking trimethoprim alone (200 mg daily) and 10 (33%) of 30 taking the

placebo developed illness (p<.05 and p<.0001 respectively). Diarrhea occurred significantly less frequently among subjects receiving trimethoprim than among placebo recipients (p<.05). None of 11 students given bicozamycin (500 mg four times daily) developed diarrhea during a three-week study, whereas 10 (53%) of 19 placebo recipients became ill (p=.003). Four (7%) of 54 subjects receiving norfloxacin (400 mg daily) experienced diarrhea during a two-week study; in contrast, 34 (60%) of 57 placebo recipients developed diarrhea (p<.0001). various antimicrobial agents prevented illness due to enterotoxiqenic Escherichia coli and Shigella as well as that unassociated with a pathogen. well drugs were tolerated. Current evidence suggests trimethoprim-sulfamethoxazole is the optimal antimicrobial agent available for prophylaxis in travelers' diarrhea." (Authors' abstract)

DuPont HL, Ericsson CD, Reves RR, Galindo E. Antimicrobial therapy for travelers' diarrhea. Rev Infect Dis 1986 May-Jun;8(suppl 2):S217-22

Bacterial enteropathogens that have recently been isolated in various regions of the world generally are susceptible to a number of antimicrobial agents, including trimethoprim-sulfamethoxazole, bicozamycin, furazolidone, quinolone carboxylic acid derivatives, such as norfloxacin, ciprofloxacin, enoxacin. The present study provides evidence that 4 of these drugs trimethoprim-sulfamethoxazole. alone, trimethoprim furazolidone. bicozamycin - are effective in the therapy for travelers' diarrhea occurring in one urban area of Mexico. Three clinical trials were carried out in Mexico to examine the efficacy of various antimicrobial agents in the treatment travelers' diarrhea among students from the United States. Thirty-seven subjects received twice daily for 5 days 160 mg of trimethoprim and 800 mg of sulfamethoxazole, 38 received 200 mg of trimethoprim, and 35 received a placebo. Another group of students were given 100 mg of furazolidone (47 students) or ampicillin (47 students) 4 times a day for 5 days. In the third study, 500 mg of bicozamycin (72 students) or a placebo (68 students) was given 4 times a day for 3 days. Most students who received trimethoprimsulfamethoxazole (78%), trimethoprim (84%), or bicozamycin (85%) had recovered by 48 h after initiation of treatment, as compared with 14% and 47% in the corresponding placebo groups and 55% in the furazolidone group. The agents had a positive effect for all etiologic categories, including diarrhea due to enterotoxigenic <u>Escherichia coli</u> and <u>Shigella</u> strains and illness without any established etiologic agents. <u>Treatment</u> failures were unusual with trimethoprim-sulfamethoxazole, trimethoprim, and bicozamycin therapy (5% vs 39% for the placebo-treated students). The drugs were well tolerated. The use of trimethoprim-sulfamethoxazole or trimethoprim alone in the empiric treatment of moderate-to-severe travelers' diarrhea is advocated,

DuPont HL. Diarrheal diseases: an overview. Am J Med 1985 Jun 28;78(suppl 68):63-4

DuPont HL. Nonfluid therapy and selected chemoprophylaxis of acute diarrhea. Am ∂ Med 1985 Jun 28;78(suppl 68):81-90

Various available forms of therapy can decrease morbidity and mortality associated with acute diarrhea. Oral fluids represent the cornerstone of therapy of all cases. A variety of agents acting nonspecifically can decrease diarrhea and improve other worrisome symptoms associated with enteric infection. Kaopectate makes the stool more formed but has little additional effects. Bismuth subsalicylate reduces the number of stools passed by about

50% and improves other associated symptomatology. The drugs that affect motility, such as loperamide and diphenoxylate, are the most active of the be avoided in patients nonspecifically acting drugs. They must significant fever and dysentery. Trimethoprim-sulfamethoxazole is now considered the drug of choice for shigellosis due to the presence of ampicillin-resistant Shigella strains in most regions of the world. Trimethoprim-sulfamethoxazole is also an effective form of therapy enterotoxigenic <u>Escherichia</u> coli (ETEC) infection and for traveler's diarrhea without definable causes. Erythromycin, although not proved to be effective against Campylobacter, probably shortens the disease. Furazolidone, although not dramatically effective, has a spectrum of activity that includes <u>Shigella</u>, ETEC, <u>Campylobacter</u>, and <u>Giardia lamblia</u>. It may not be effective in <u>severely</u> ill (hospitalized) patients with diarrhea. Mildly ill patients (one to 3 unformed stools in 24 h with minimal additional symptoms) probably are best treated with fluids only. Mild-to-moderately ill persons (3 to 6 unformed stools in 24 h) can be treated with a drug that acts nonspecifically, such as bismuth subsalicylate or loperamide. Those with severe diseases (6 or more unformed stools with moderate-to-severe-associated symptoms), particularly when associated with fever and the passage of bloody mucoid stools, may be given an The antimicrobial drug given will be determined antimicrobial agent. ancillary laboratory tests (dark-field examination or examination of wet-mount preparation for motile Campylobacter or stool culture for Snigella, Campylobacter, or Salmonella) or may be administered on an empiric basis. Traveler's diarrhea can be eliminated in selected persons by the administration of a pharmacologic agent. Liquid bismuth subsalicylate is effective in large doses, which may be impractical. Studies with the tablet formulation suggest that it is partially effective in preventing the illness. Doxycycline and trimethoprim-sulfamethoxazole are more effective. (Modified author's abstract)

DuPont HL, Steele JH. Use of antimicrobial agents in animal feeds: implications for human health. Rev Infect Dis 1987 May-Jun;9(3):447-60

DuPont HL see Ericsson CD

DuPont HL see Johnson PC

DuPont HL see Sattherwhite TK

DuPont HL see Steffen R

Echeverria P, Seriwatana J, Sethabutr D, Taylor DN. DNA hybridization in the diagnosis of bacterial diarrhea. Clin Lab Med 1985 Sep;5(3):447-62

Deoxyribonucleic acid (DNA) hybridization with either cloned genes for enteropathogenic determinants or DNA segments that are species-specific is a valuable tool to identify certain bacterial enteric pathogens. Thus far, only Escherichia coli and Vibrio cholerae enterotoxin gene probes have been used to identify enterotoxigenic E. coli and V. cholerae in clinical specimens. DNA probes developed for Salmonella, Shigella, Campylobacter, and enteroinvasive and enteropathogenic E. coli need to be evaluated with clinical specimens. The major contribution of this system so far has been to examine large numbers of specimens in epidemiologic studies. Once nonradioactive-DNA probes are developed, this system will have potential application in clinical laboratories and in research laboratories in the developing world where diarrheal disease causes its greatest impact. (Modified authors, abstract)

Echeverria P see Taylor DN

Edelman R. Prevention and treatment of infectious diarrhea: speculations on the next 10 years. Am J Med 1985 Jun 28;78(suppl 68):99-106

"The next decade should explode with exciting schemes and novel agents for the prevention and treatment of infectious diarrhea. The development of oral, nonabsorbed antibiotics will continue, but new antidiarrheal drugs, such as gastrointestinal hormone analogues and alpha-adrenergic agonists, will be added to the therapeutic armamentarium. Improved oral rehydration solutions, such as glycine in electrolyte solution, promise to revolutionize the management of diarrhea by diminishing diarrheal stool volume to the point where losses are too small to be clinically relevant. Infant formulas and adult oral solutions fortified with antibodies raised against selected enteropathogens may provide a way to prevent infectious diarrheas in infants and travelers. Advances in genetic engineering will usher in a new era of experimental and licensed enteric vaccines, including those against cholera, Escherichia coli, Shigella, typhoid fever, and rotavirus." (Author's abstract)

Edelman R see Levine MM

Edelson PJ see Smith SM

Eichenwald HF, McCracken GH, Jr. Acute diarrheal disease. Med Clin North Am 1970 Mar;54(2):443-53

Eidels L. Proia RL, Hart DA. Membrane receptors for bacterial toxins. Microbiol Rev 1983 Dec:47(4):596-20

Ellencweig AY, Slater PE. Demographic and socio-economic patterns of hospitalization for infectious diseases in Israel. Eur J Epidemiol 1986 Jun;2(2):83-9

"Hospitalization rates in Israel for five common enteric communicable diseases were computed according to age, sex, religion, origin and place of residence. Higher rates were found for non-Jews of both sexes and males of all origins. Higher rates were also found for settlements inhabited by new immigrants of low socio-economic classes. These findings suggest that more emphasis should be placed upon sanitary improvements and education for better health, rather than on merely improving the health delivery system." (Authors' abstract)

Elliott EJ, Walker-Smith JA, Farthing MJG. The role of bicarbonate and base precursors in treatment of acute gastroenteritis. Arch Dis Child 1987 Jan;62 (1):91-5

The inclusion of bicarbonate or a base precursor (citrate, acetate, or lactate) in oral rehydration solutions (ORS) is generally assumed to be necessary, both for promotion of water and sodium absorption and correction of acidosis. This article reviews the available evidence on the role of bicarbonate and base precursors from animal and human studies. It has been shown that bicarbonate, acetate, and citrate are unable (unlike glucose) to reverse water and sodium secretion induced by cholera toxin. These findings suggest that bicarbonate may not be beneficial with respect to the promotion of water absorption in the enterotoxin-mediated diarrheas. It has also been found that provided perfusion is maintained, renal compensation ensures that acid base state will normalize

with rehydration without additional base in the vast majority of cases of acute gastroenteritis. In severely dehydrated or acidotic patients (pH<7.2), in those with persistent high-volume diarrhea, and in those with renal function and worsening metabolic acidosis, the administration of intravenous base, such as sodium bicarbonate, is indicated and may be life-saving. Also, there is little clinical evidence to support the inclusion of base in the treatment of acidosis and dehydration in acute diarrhea. When considering ORS, particularly for use in developing countries, the prime concern must be simplicity, effectiveness, and economy. Bicarbonate and base precursors pose problems with respect to availability, cost, packaging, and stability. Exclusion of these substances would decrease the production cost of pre-packaged ORS, make preparation in the home easier, and possibly result in early initiation of oral rehydration therapy, leading ultimately to the prevention of serious dehydration.

Elson CO, Kagnoff MF, Fiocchi C, Befus AD, Targan S. Intestinal immunity and inflammation: recent progress. Gastroenterology 1986 Sep;91(3):746-68

Elzouki AY, Vesikari T. First international conference on infections in children in Arab countries. Pediatr Infect Dis 1985 Sep-Oct;4(5):527-31

Ene L see Alexandrescu M

Eneroth P see Ljungh A

Ericsson CD, DuPont HL, Johnson PC. Nonantibiotic therapy for travelers' diarrhea. Rev Infect Dis 1986 May-Jun;8(suppl 2):S202-6

A number of studies of the efficacy of nonantibiotic agents for the treatment of diarrhea have been conducted, but relatively few studies have examined the usefulness of these agents in the treatment of acute travelers' diarrhea. When compared with placebo (p<.025), bismuth subsalicylate (pepto-bismol) taken orally at a dosage of as low as 30 ml every half hour for 8 doses is shown to be effective in reducing the frequency of episodes of diarrhea. Preliminary results indicate that loperamide (two 2-mg capsules followed by one capsule after each loose bowel movement) is more effective (p<.025) than bismuth subsalicylate in relieving diarrhea. Likewise, preliminary results also show that BW942c, an unlicensed endorphin-like pentapeptide, offers more relief of diarrhea in the first 12 h of therapy than did trimethoprim— sulfamethoxazole (p=.02) or placebo (p=.0007). Use of a nonantibiotic drug for empiric treatment of travelers' diarrhea appears to be a reasonable approach, especially for patients with mild-to-moderate disease and with no evidence of high fever or dysentery. (Modified authors' abstract)

Ericsson CD see DuPont HL

Ericsson CD see Johnson PC

Esrey SA, Habicht J-P. Epidemiologic evidence for health benefits from improved water and sanitation in developing countries. Epidemiol Rev 1986;8: 117-28

This report describes the epidemiologic evidence of health benefits from improved water and sanitation in developing countries. In developing countries, an improvement in the quality of drinking water, an increase in the

quantity of water provided and used, and the provision of sanitation facilities for safe disposal of human excreta could indeed benefit child health. review attempts to evaluate the suitability of methodologies, and to assess the conclusions of studies, since 1950, that describe the effectiveness of water and sanitation interventions on the incidence of diarrheal disease morbidity. nutritional status, and early childhood mortality. Epidemiologic evidence from selected studies on child health and the health impact of improved water and Twenty-six studies examined the health impact of sanitation are cited. improved sanitation. These studies reported an association between sanitation and improved health. The findings suggest that sanitation was a more important determinant of child health than was water. A flush toilet produced a larger health impact than did pit latrines. The majority of studies compared the health status between groups with different types of water supplies. provision of new or improved water supplies did not correspond to a concomitant increase in the use of water. Water-related improvements encompass an increase in the availability of water as well as better water quality. Piped-water supplies and modern sanitation has reduced the morbidity rates, and has contained the spread of infections. Health impact due to water quantity and quality is discussed in detail. Twelve studies examined the health impact of pure versus contaminated water supplies. It is concluded that water and sanitation interventions can markedly improve the health of children by significantly reducing transmission possibilities.

Esrey SA, Feachem RG, Hughes JM. Interventions for the control of diarrhoeal diseases among young children: improving water supplies and excreta disposal facilities. Bull WHO 1985:63(4):757-72

This paper is the ninth in a series of reviews of potential antidiarrhea interventions. This review analyses the effectiveness of water supply and excreta disposal improvements for reducing diarrhea rates in young children in their also examine authors countries. The diarrhea-related infections, nutritional status, and mortality. A theoretical model is proposed that relates the level of ingestion of diarrhea-causing pathogens to the frequency of diarrhea in the community. The implications of this model are that, in poor communities with inadequate water supply and excreta disposal, reducing the level of enteric pathogen ingestion by a given amount will have a greater impact on diarrheal mortality rates than on morbidity rates, a greater impact on the incidence rate of severe diarrhea than on that of mild diarrhea, and a greater impact on diarrhea caused by pathogens setting off the illness at high-infectious doses than on diarrhea caused by pathogens of a low-infectious dose. The impact of water supply and sanitation on diarrhea-related infections, nutritional status, and mortality is analysed by reviewing 67 studies from 28 countries. The median reduction in diarrhea morbidity rates is 22% from all studies and 27% from a few better-designed studies. All studies of the impact on total mortality rates show a median reduction of 21%, while the few better-designed studies give a median reduction of 30%. Improvements in water quality have less of an impact than improvements in water availability or excreta disposal. (Modified authors' abstract)

Evans DG see Evans DJ, Jr.

Evans DJ, Jr., Evans DG. Classification of pathogenic <u>Escherichia coli</u> according to serotype and the production of virulence factors, with special reference to colonization-factor antigens. Rev Infect Dis 1983 Sep-Oct;5 (suppl 4):S692-701

"Multiple criteria are required to classify Escherichia coli isolates according to pathogenic grouping. There are three groups associated with intestinal illness. Acute watery diarrhea is the hallmark of enterotoxigenic E. coli, the majority of which belong to a small set of specific serotypes harboring plasmids encoding for the production of heat-stable enterotoxin and/or heat-labile enterotoxin and also for a fimbrial colonization-factor antigen. Bacterial dysentery is caused by specific, nonmotile Shigella-like E. coli serogroups identifiable by the property of tissue invasiveness. Specific virulence factors have not been defined for the traditional enteropathogenic E. coli serogroups, although the ability to colonize and overgrow the intestine appears to be of primary importance in this group. E. coli associated with extraintestinal infections generally belong to serogroups and serotypes other than those noted above; the virulence factors of these E. coli include polysaccharide capsular antigens, hemolysin, and fimbrial colonization factors that are antigenically complex and different from those of enterotoxigenic E. coli." (Authors' abstract)

Evans N. Pathogenic mechanisms in bacterial diarrhoea. Clin Gastroenterol 1979 Sep;8(3):599-623

This chapter reviews the etiology and pathogenesis of acute bacterial diarrhea. The incidence with which specific bacteria are implicated in diarrhea will depend on the age group being studied, the locality, the time of year, and the diagnostic abilities of the local laboratory. Escherichia coli has long been established as an important pathogen. Depending on their pathogenesis, they are divided into different types, such as enteropathogenic, enterotoxigenic, enteroinvasive, and cytotoxic. Enterotoxigenic <u>E. coli</u> may produce heat-labile toxin, heat-stable toxin, or both. Species of <u>Salmonella</u> and <u>Shigella</u> are important pathogens throughout the world in all age groups. In <u>Shigella</u> infections, the main impact is on the colon with inflammation and penetration of the mucosa, associated with fever, cramping abdominal pain, and the passage of profuse stools, containing blood, pus, and mucus. In Salmonella infections, the organisms penetrate the small and large intestine. The lymphoid follide of the bowel may ulcerate. The organisms multiply intracellularly, and there may be systemic upset. Vibrio cholerae colonize the small intestine and elaborate an enterotoxin (choleragen), which elicits a chemical diarrhea without tissue damage. <u>Campylobacter</u> <u>fetus</u> ssp. <u>jejuni</u>, a microaerophilic <u>Vibrio</u>, has recently emerged as a major intestinal pathogen, but the pathogenic mechanism However, tissue invasion or a cytotoxin may be involved. Salmonella food poisoning, the organisms are ingested through contaminated food and multiply in the body, causing diarrhea, vomiting, and fever. poisoning bacteria also include Clostridium welchii (type A), C. botulinum, staphylococcal and streptococcal strains, <u>Bacillus cereus</u>, <u>Vibrio</u> <u>parahaemolyticus</u> and noncholera vibrios. Some strains of clostridia and other bacteria are associated with enterocolitis. The pathogenic potential of certain enteric bacteria may be invested in a plasmid, the extrachromosomal genetic element composed of deoxyribonucleic acid. There are 3 mechanisms leading to diarrhea; (1) colonizing bacteria adhere to the small intestine and produce an enterotoxin that promotes a net secretion of water and electrolytes; (2) a cytotoxin elicits tissue damage in the distal small bowel and large intestine; and (3) bacteria invade and damage the colon producing a dysenteric stool, with blood, pus, and mucus. The host and environmental factors and implications for treatment and prevention are discussed.

т

Everson RJ <u>see</u> Riviere JE

Ewing WH, Hugh R, Johnson JG. Studies on the Aeromonas group. Atlanta, Ga.: U S Department of Health and Human Services, 1981. 37 p.

Fainstein V see Rolston KV

Falchuk ZM. Gluten-sensitive enteropathy. Clin Gastroenterol 1983 May;12(2): 475-94

"Gluten-sensitive enteropathy is a disease in which the small intestinal mucosa of susceptible persons is damaged after eating gluten-containing foods. damaged intestinal mucosa is incapable of normal function, and the affected patients have malabsorption of one or more dietary components. The childhood and adult forms of the disease are identical. The small intestinal lesion is characterized by villus flattening, cuboidal epithelial cells, and infiltration of the lamina propria with lymphocytes and plasma cells. The diagnosis in all cases must be confirmed by intestinal biopsy before and after treatment with a gluten-free diet, since other conditions may produce a similar lesion. The post-treatment biopsy should disclose reversion toward normal. Treatment with a gluten-free diet is lifelong. Various theories have been proposed to account for the pathogenesis of the gluten-induced damage. These include the presence of an enzyme deficiency which allows toxic degradation products of gluten to accumulate and kill the epithelial cell, and the presence of surface receptors which allow binding of gluten to the cell surface, with cell death as the result. A third theory states that immune factors are important since anti-gluten antibodies are made in the mucosa, and contisone inhibits the lesion in vivo and in vitro. Genetic studies show a familial pattern of the disease and a preponderance of histocompatibility antigens, HLA-B8 and HLA-DW3. The pathogenesis may be related to cell surface receptors, allowing for immunological cytopathic factors to be generated. An $\underline{\text{in}}$ $\underline{\text{vitro}}$ technique of culturing biopsy specimens may be useful in making a diagnosis." (Author's abstract)

Fanaroff AA see Kliegman RM

Farmer JJ, 3d. see Holmberg SB

Farmer RG. Nonspecific ulcerative proctitis. Gastroenterol Clin North Am 1987 Mar; 16(1):157-74

Farthing MJG see Elliot EJ

Fattal B see Shuval HI

Fayer R, Ungar BLP. <u>Cryptosporidium</u> spp. and cryptosporidiosis. Microbiol Rev 1986 Dec;50(4):458-83

This review summarizes publications that have increased the knowledge about <u>Cryptosporidium</u>, its biology and its veterinary and medical importance. The organism's history, classification, host specificity, life cycle and morphology have been described. An account of the hosts has been presented. This includes infection in fish and snakes, birds, small mammals, large mammals, nonhuman primates and humans. The surveys on human infection suggest that <u>Cryptosporidium</u> spp. is associated with diarrhea in all areas of the world. It

is most prevalent in the less-developed regions. <u>Cryptosporidium</u> infections are more common in the warmer or more humid months of the year. Children may be the most susceptible and those aged under 2 may have the greatest prevalence. Characteristically, the diarrhea is profuse and watery, and as many as 71 stools passed per day has been reported. Diarrhea appears to be more copious in immunocompromised patients. Significant weight loss may result. Pests and farm animals may be important sources of infection; laboratory and research animals have also been implicated in several instances. Contact with other infected persons seems to play a role. Diagnosis of human infection was initially based on identification of developmental stages in biopsy sections, usually from the small intestine and occasionally from the rectum. Supportive care with oral or intravenous hydration is the primary therapeutic intervention available for humans with cryptosporidiosis, Controlling the spread of Cryptosporidium requires reduction or elimination of oocysts from the environment.

Feachem R, Miller C, Drasar B. Environmental aspects of cholera epidemiology. II. Occurrence and survival of <u>Vibrio cholerae</u> in the environment. Trop Dis Bull 1981 Oct;78(1D):865-80

The occurrence and survival of Vibrio cholerae in the environment is discussed on the basis of a review of available literature. Some traditionally held beliefs about cholera epidemiology may be challenged in light of recent discoveries. There is now strong evidence for an aquatic reservoir of V. cholerae. Some V. cholerae strains have been isolated from noncontaminated aquatic environments, and may be part of the permanent microflora. Moreover, V. cholerae appear to have prolonged existence in some environments, i.e. sewage. Although the evidence is limited, foods also act as one of the vehicles for spreading cholera. Possible interbiotypic and intrabiotypic variability in environmental persistence of the El Tor and classical cholera biotypes remains to be documented.

Feachem R. Priorities for diarrhoeal disease control: water, excreta, behaviour and diarrhoea. Diarrhoea Dialoque 1981;(4):4-5

Feachem R, McGarry M, Mara D, eds. Water, wastes and health in hot climates. London: Wiley, 1977. xvi, 399 p.

Feachem RG, Hogan RC, Merson MH. Diarrhoeal disease control: reviews of potential interventions. Bull WHO 1983;61(4):637-40

"Diarrhoeal diseases are a major cause of sickness and death among young children in most developing countries. Since effective interventions to control these diseases are available, they are a priority target for the primary health care programs being planned or implemented in many countries. Governments and international agencies, including the World Health Organization, have emphasized oral rehydration as an effective intervention for reducing diarrhoeal disease mortality. Other interventions are, however, needed to reduce morbidity, to reduce mortality not averted by oral rehydration, and to develop a multifaceted approach in which oral rehydration is one of several anti-diarrhoea measures being implemented simultaneously with mutually reinforcing and complementary impacts. This paper presents a classification of potential interventions for the control of diarrhoeal disease morbidity and/or mortality among children aged under 5 and introduces a series of reviews of these interventions." (Authors' abstract)

Feachem RG. Environmental aspects of cholera epidemiology. I. A review of selected reports of endemic and epidemic situations during 1961-1980. Trop Dis Bull 1981 Aug;78(8):675-98

This paper is the first of a series of 3 papers which focus on certain modern issues and debates relating to the epidemiology of cholera. Particular attention has been given to the relative importance of non-waterborne and waterborne transmission of cholera, to the role of bathing water in cholera transmission, to cholera seasonality, to the survival of Vibrio cholerae in the extraintestinal environment, to the possible maintenance of marine or estuarine reservoirs of V. cholerae, and to cholera control. The review covers over 60 reports on cholera (1961-1980) in 20 countries: Australia, Bahrain, Bangladesh, Gilbert Islands (now Kiribati), Guam, Hong Kong, India, Israel, Italy, Japan, Malaysia, Philippines, Portugal, South Africa, Sri Lanka, Taiwan, the USA, West Africa. The selection is based partly on the need for wide geographical coverage and partly on seeking studies which bear upon the epidemiological and environmental issues mentioned above. Outbreaks of cholera in which waterborne transmission was impossible or unlikely were found to occur in several countries. With a few exceptions, these outbreaks were all in nonendemic areas where living standards are relatively high. The only studies reviewed here in which waterborne transmission was demonstrated or strongly implicated were those on bottled mineral water in Portugal, the Queensland cases, and the transmission investigations in Bangladesh. The consumption of raw or partially cooked seafood was implicated in several outbreaks of cholera. The maintenance of an aquatic reservoir of V. cholerae Ol, and an association between this reservoir and human cholera cases were suspected in Queensland, Sardinia, Louisiana. V. cholerae multiplication and survival in sweat was thought to contribute to person-to-person transmission in the Sahel and in the South African gold mines.

Feachem RG. Environmental aspects of cholera epidemiology. III. Transmission and control. Trop Dis Bull 1982 Jan;79(1):1-47

This paper, the last in a series of 3 studies on environmental aspects of cholera epidemiology, draws on some of the themes of the other two, and presents a detailed discussion on transmission and control of cholera. A review of the historical perspectives show that many modern cholera epidemiologists have come extremely close to the exclusive waterborne theory of transmission. One of the purposes of this paper is to refute the hypothesis that cholera is exclusively waterborne and to show that, even in Bangladesh, other epidemiological factors are likely to be responsible in transmission. Data on the epidemiologic features of cholera, infectious dose and transmission, and transmission studies in Bangladesh are given. Water supply, sanitation and cholera, waterborne and non-waterborne, person-to-person transmission and the epidemiological role of aquatic reservoirs are discussed. Some of the salient points are summarized, and future research priorities are outlined.

Feachem RG, Guy MW, Harrison S, Iwugo KO, Marshall T, Mbere N, Muller R, Wright AM. Excreta disposal facilities and intestinal parasitism in urban Africa: preliminary studies in Botswana, Ghana and Zambia. Trans R Soc Trop Med Hyg 1983;77(4):515-21

The relationship between intestinal parasitism and a variety of excrete disposal systems were investigated in different urban environments of Africa.

The cities selected were Gaborone (Botswana). Ndola (Zambia) and Kumasi (Ghana). Parasitic prevalence and intensity rates amongst groups of urban residents having similar socioeconomic status and housing but different excreta disposal technologies, were compared. Protozoal prevalence rates in Gaborone were considerably lower than that in other cities with the exception of infections with Giardia and Entamoeba coli. Ndola had the highest prevalence of all protozoal infections (71%) and had a considerably higher E. histolytica infection prevalence (26%) than the other cities. Gaborone had a low prevalence of all helminths except Hymenolepis nama (3%), while the communities surveyed in Ndola and Kumasi were commonly infected with hookworm (24% and 16%) and roundworm (27% and 33%). In Gaborone, there was no difference in intestinal parasitism between those using aqua privies and having access to public taps and those in identical houses enjoying flush toilets. inhouse water connections and showers. In Ndola, the group with sewered agua privies had larger houses, cleaner toilets, safer water supplies, longer residence and more people in well-paid employment than those using pit latrines or communal flush toilets. The sewered aqua privy users were not different from the other groups with regard to hookworm and protozoal infections but showed higher Ascaris infection rates. In Kumasi, despite the differences in toilet type - from squalid communal aqua privies through often fouled bucket latrines to well-maintained flush toilet systems - and despite also the differences in water provision, no evidence was obtained of any differences, in intestinal parasitism between the groups studied. The results suggest that the provision of superior water and sanitation facilities to a small cluster of houses, or to houses scattered throughout an area may not protect those families from infection if the overall level of fecal contamination of the environment is high. Since the sample sizes and response rates achieved this study were low, follow-up studies with larger samples have recommended.

Feachem RG. Infections related to water and excreta: the health dimension of the decade. In: Water supply and sanitation in developing countries. London: Institute of Water Engineers and Scientists, 1983:25-46

Feachem RG. Infectious disease related to water supply and excreta disposal facilities. AMBIO 1977:6(1):55-8

Feachem RG, Koblinsky MA. Interventions for the control of diarrhoeal diseases among young children: measles immunization. Bull WHO 1983;61(4): 641-52

"The effects of measles immunization on diarrhoea morbidity and mortality are reviewed, using data from field studies and theoretical calculations. types of measles-associated diarrhoea are distinguished: pre-rash-onset and diarrhoea, which starts between 1 week post-rash-onset, and post-measles diarrhoea, which starts weeks post-rash-onset. The etiology of these measles-associated diarrhoeas is unknown but some evidence points towards a frequently severe and dysenteric form of disease, with $\frac{\text{Shigella}}{\text{immunization}}$ playing a major role. Theoretical calculations indicate that measles $\frac{\text{immunization}}{\text{immunization}}$, at the age of 9-11 months, with coverage of between 45% and 90% can avert 44-64% of measles cases, 0.6-3.8% of diarrhoea episodes, and 6-26% of diarrhoea deaths among children under 5 years of age. The cost of measles immunization is in the range of US\$ 2-15 (1982 prices) per child vaccinated. The impact of measles immunization on diarrhoea mortality may be partly additional to the impact of oral rehydration, because it averts deaths that are not prevented by oral rehydration. Community research is

urgently needed to confirm or reject these theoretical suppositions, to clarify the etiology of measles-associated diarrhoea, and to determine the cost-effectiveness of measles immunization as an intervention to reduce diarrhoea mortality." (Authors' abstract)

Feachem RG, Koblinsky MA. Interventions for the control of diarrhoeal diseases among young children; promotion of breast-feeding. Bull WHO 1984;62 (2):271-91

The literature on the relative risks of diarrheal morbidity for infants on several methodological different feeding modes suffers from Thirty-five studies from 14 countries were reviewed; 83% of studies found that exclusive breast feeding was protective compared to partial breast feeding, 88% that exclusive breast feeding was protective compared to no breast feeding, and 76% that partial breast feeding was protective compared to no breast feeding. When infants receiving no breastmilk are contrasted with infants on exclusive or partial breast feeding, the median relative risks are 3.0 for those aged 0-2 months, 2.4 for those aged 3-5 months, and 1.3-1.5 for those aged 6-11 months. Above 1 year of age no protective effect of breast feeding on diarrheal morbidity is evident. When infants receiving no breastmilk are contrasted with those on exclusive breast feeding, median relative risks are 3.5-4.9 in the The literature does not suggest that the relative first 6 months of life. risks of diarrheal morbidity for bottlefed infants are higher in poor families than in more wealthy families. The protective effects of breast feeding do not appear to continue after the cessation of breast feeding. There is evidence of considerably increased severity of diarrhea among bottlefed infants. There is limited literature on the relative risks of diarrheal mortality for infants on different feeding modes. Nine studies from 5 countries were reviewed, most of which showed that breast feeding protects substantially against death from diarrhea. When infants receiving no breastmilk are contrasted with those on exclusive breast feeding, the median relative risk of death from diarrhea during the first 6 months of life is 25. When partially and exclusively breastfed infants are contrasted, the median relative risk of death from diarrhea is 8.6. Breast feeding can be promoted by changes in hospital routine and by giving information and support to mothers. A review of 21 studies from 8 countries shows that, by such promotion, the most likely reductions in the prevalence of nonbreastfed infants are 40% among infants aged 0-2 months, 30%among those aged 3-5 months, and 10% among those between 6 months and 1 year Theoretical calculations based on these data show that such promotion can reduce diarrheal morbidity rates by 8-20% and diarrheal mortality rates by 24-27% in the first 6 months of life. For children aged 0-59 months, diarrheal morbidity rates would be reduced by 1-4% and mortality rates by 8-9%. A recent study in Costa Rica has documented a substantial impact of breast feeding promotion on neonatal diarrheal morbidity and mortality, and on diarrheal morbidity in infants aged 0-5 months. The Costa Rican data show good agreement with the theoretical computations presented in this paper. Several important aspects of breast feeding and diarrhea remain to be clarified by research. However, the need for this research should not delay action designed to promote breast feeding and to monitor its effects upon feeding practice and upon diarrhea. (Modified authors' abstract)

Feachem RG. Interventions for the control of diarrhoeal diseases among young children: promotion of personal and domestic hygiene. Bull WHO 1984;62(3): 467-76

Intervention for the control of diarrheal diseases among young children, and aspects concerning the promotion of personal and domestic hygiene are

described. Three studies from Bangladesh, the USA, and Guatemala on the impact of hydiene education programs on diarrhea are reviewed. In Bangladesh and the USA, this education focused exclusively on handwashing, while in Guatemala, the program sought to improve several aspects of personal and domestic hygiene. Four groups, one given soap and water, a soap-only group, a water-only group and a control group, were chosen for the study in Bangladesh, and the secondary case rate of shigellosis was studied. It was 2.2% for the soap and water group, while it was 14.2% for the control group who used nothing. It is concluded that the intervention had lowered the secondary case rate by 84%. Attack rates of non-Shigella diarrhea were 37% lower in the soap and water group than in the control group. In The USA, the impact of handwashing on the incidence of diarrhea in 4 day-care centers was studied. Two groups of children, aged 6-17 months and the other, aged 18-29 months, were studied. The incidence of diarrhea among the children, aged 6-29 months, was reduced by 48% in the handwashing day-care centers. The hygiene education program of Guatemala was carried out in 1979-1980, in which 106 mothers with children, aged under 6 were included, while 32 mothers acted as controls. The impact on the proportion of days with diarrhea was higher than the impact on the incidence. Diarrhea peaked during March-June rather than being endemic throughout the year. reduction of incidence rate of diarrhea due to hydiene education ranged between These 3 studies suggest that hygiene education, especially handwashing, has a marked impact on diarrheal morbidity rates.

Feachem RG. Preventing diarrhoea: what are the policy options? Health Policy Plann 1986;1(2):109-17

"Oral rehydration therapy (ORT) has become the cornerstone of most programs of diarrhoea control, and is highly effective in preventing death dehydration, caused by acute watery episodes of diarrhoea. The effectiveness of ORT in reducing mortality from chronic or dysenteric diarrhoeas is believed to be low, and, in addition, ORT can be expected to have little or no impact on diarrhoea morbidity rates. In view of this, ORT should be complemented by other interventions designed to avert mortality not averted by ORT, and reduce morbidity rates. The Diarrhoeal Diseases Control Programme of WHO has initiated a systematic study of the effectiveness and cost of interventions that are potentially useful in diarrhoea control. This paper summarizes findings of this study, concentrating on those interventions for which the evidence for high effectiveness and feasibility is strong. These are: promotion of breast feeding, weaning education, measles immunization, improving water supply and sanitation, promotion of hygiene, and, when the new vaccines are available, rotavirus, and possibly cholera, immunization. Estimates are presented of the cost-effectiveness of these interventions in reducing diarrhoea morbidity and mortality rates among children aged under 5 years of age." (Author's abstract)

Feachem RG. The role of water supply and sanitation in reducing mortality in China, Costa Rica, Kerala State (India) and Sri Lanka. <u>In</u>: Halstead SB, Walsh JA, Warren KS, eds. Good health at low cost; proceedings of a conference, held at the Bellagio Conference Center, Italy, 29 Apr-3 May 1985:191-8

Feachem RG. Rural water and sanitation; community participation in appropriate water supply and sanitation technologies: the mythology for the decade. Proc R Soc Lond (B) 1980 Jul;209(1174):15-29

"The International Drinking Water Supply and Sanitation Decade (1981-90) is almost upon us. During this period, massive international efforts will be made

to accelerate the provision of domestic water supply and sanitation facilities for the rural population of developing countries. Certain concepts and approaches are being developed and promoted as guiding themes that will help to steer and coordinate the activities of the Decade. Among them are 'appropriate technology' 'community participation' and a 'village level' or 'user-choice' approach. The validity and importance of these concepts are discussed. It is concluded that these concepts are often applied in an over-simplified manner and that they divert attention away from the fundamental political and administrative realities that primarily determine the success or failure of rural water and sanitation programmes." (Author's abstract)

Feachem RG, Bradley DJ, Garelick H, Mara DD. Sanitation and disease: health aspects of excreta and wastewater management. New York: Wiley, 1983. 501 p. [World Bank studies in water supply and sanitation, 3]

Feachem RG. Vitamin A deficiency and diarrhoea: a review of interrelationships and their implications for the control of xerophthalmia and diarrhoea. Trop Dis Bull 1987:84(3):R1-16

This review explores the relationships between vitamin A deficiency and diarrhea among young children. The major focus is on the implications of these relationships for policies towards the control of childhood diarrhea and xerophthalmia. The possible benefits of giving vitamin A to children with diarrhea are also considered. Prevalence rates for mild xerophthalmia in pre-school children are seldom over 10%, and the range of 5-10% represents the worst end of the spectrum of frequency. Typically, prevalence rates of active corneal xerophthalmia are well below 0.5% among pre-school children. surveys report that mild xerophthalmia is significantly more common in boys than in girls. Limited evidence suggests that vitamin A deficiency predisposes to increased risk of diarrhea, and to an increased risk of death among pre-school children. It is possible that the prevalence rate of vitamin A deficiency that confers these increased risks is higher than the prevalence physiologically rate of xerophthalmia, because there are children with important vitamin A deficiency but without eye signs. It is plausible that, in some settings, diarrhea may precipitate xerophthalmia, although the evidence for this is weak. The evidence suggests that, despite lowered absorption, clinically significant amounts of vitamin A can be absorbed when administered to patients during diarrhea. There are 4 possible reasons for administering vitamin A to children with diarrhea: (1) to contribute to the community-wide control of vitamin A deficiency; (2) to avert subsequent xerophthalmia; (3) to influence the course of the current diarrhea episode; or (4) to reduce the risk of subsequent diarrhea. The 3 methods for preventing vitamin A deficiency that are normally advocated are the periodic distribution of massive oral doses of vitamin A (200,000 IU every 4-6 months), the fortification of staple foods with vitamin A, and nutrition education to encourage adequate consumption of locally available foods which are rich in vitamin A. If, for any or all of the reasons mentioned earlier, it is decided to administer vitamin A to children with diarrhea, the method of administration must carefully be researched. various options have different implications for targeting as well as for minimizing the risk of administering over-doses or under-doses. Further, if fortification of oral rehydration solution is selected as the method of administration, specific practical issues, such as stability, cost, etc., are Research needs in the field of diarrhea and vitamin A to be resolved. deficiency have been identified.

Feachem RG, Burns E, Cairnoross AM, Cronin A, Cross R, Curtis D, Khan MK, Lamb

D, Southal H. Water, health and development: an interdisciplinary evaluation. London: Tri-Med Books, 1978. 267 p.

Feachem RG see Ashworth A

Feachem RG see Briscoe J

Feachem RG see de Zovsa I

Feachem RG see Esrey SA

Federman M see DeGirolami PC

Fedorak RN, Field M. Antidiarrheal therapy: prospects for new agents. Dig Dis Sci 1987 Feb;32(2):195-205

"Successful treatment of severe diarrhea has traditionally relied upon opiates or opiate derivatives. Recent advances in the understanding of intestinal fluid and electrolyte absorption have provided the opportunity to develop therapeutic agents specific for various points in the secretory and absorptive process. Present and proposed antidiarrheal agents, in addition to antimotility activity, will be capable of stimulating intestinal fluid absorption, inhibiting intestinal fluid secretion, or both. The mechanism(s) of action and clinical implications for proposed antidiarrheal agents are reviewed." (Authors' abstract)

Fedorak RN see Chang EB

Feeding during diarrhea. Nutr Rev 1986 Mar:44(3):102

Fekety R. Recent advances in management of bacterial diarrhea. Rev Infect Dis 1983 Mar-Apr;5(2):246-57

This paper reviews some of the bacteria recognized in the past few years as important causes of diarrhea. The number of recognized infectious causes of diarrhea potentially treatable with specific antibiotics has markedly increased in the past 10 years. Laboratories are developing and expanding their abilities to deal with these new pathogens. Neither prophylaxis nor specific treatment of diarrhea in travelers is simple, practical, and safe. enterotoxigenic Escherichia coli is the most important cause of diarrhea in U.S. travelers to tropical areas, <u>Campylobacter jejuni</u> causes acute diarrhea in persons in the USA about as often as do <u>Salmonella</u> and <u>Shigella</u>. <u>Vibrio</u> parahaemolyticus is an important cause of outbreak of gastroenteritis following ingestion of improperly cooked shellfish; Bacillus cereus is important in outbreaks of diarrhea after ingestion of improperly cooked and stored rice in Chinese restaurants. Although Yersinia enterocolitica is probably an important cause of severe enteritis in the USA, imperfect techniques for its isolation and lack of good serologic tests have hampered recognition of its importance. Practical means for diagnosing antibiotic-associated colitis are now available. and the role of <u>Clostridium difficile</u> toxins in this disease has been established. Vancomycin, metronidazole, bacitracin, and cholestyramine are useful in treatment of antibiotic-associated colitis. (Modified author's abstract)

Fekety R see Silva J, Jr.

Feld LG, Kaskel FJ, Schoeneman MJ. The approach to fluid and electrolyte therapy in pediatrics. Adv Pediatr 1988;35:497-536

Feldman M, Schiller LR. Disorders of gastrointestinal motility associated with diabetes mellitus. Ann Intern Med 1983 Mar;98(3):378-84

"Gastrointestinal symptoms such as vomiting, constipation, diarrhea, and fecal incontinence occur frequently in patients with diabetes mellitus. In a survey of 136 diabetic outpatients, 76% had one or more gastrointestinal symptoms, the commonest symptom being constipation (found in 60%). In many cases, these symptoms are thought to be due to abnormal gastrointestinal motility that, in turn, may be a manifestation of diabetic autonomic neuropathy involving the gastrointestinal tract. The pathophysiology of these gastrointestinal symptoms, clarified in recent studies, and the clinical features and treatment of these problems in diabetic patients are reviewed." (Authors' abstract)

Feldman M. Southwestern internal medicine conference: traveler's diarrhea. Am J Med Sci 1984 Oct;288(3):136-48

"Three hundred million people, mostly tourists, participate in international travel each year. Development of an acute diarrheal syndrome abroad, while returning home, or shortly after arriving home is referred to as traveler's diarrhea (TD). TD is not a specific diagnosis but, rather, a clinical syndrome with multiple etiologies. In this article, clinical and epidemiological features of TD, specific etiologies and their pathogenesis, as well as current means of diagnosis, treatment, and prevention will be reviewed." (Author's abstract)

Feldman RA see Blaser MJ

Feldon K see Jelliffe EFP

Fettman MJ, Rollin RE. Antimicrobial alternatives for calf diarrhea: iron chelators or competitors. J Am Vet Med Assoc 1985 Oct 1;187(7):746-8

Field M see Chang EB

Field M see Fedorak RN

Fingl E, Freston JW. Antidiarrhoeal agents and laxatives: changing concepts. Clin Gastroenterol 1979 Jan;8(1):161-85

Finkelstein RA, Dorner F. Cholera enterotoxin (choleragen). Pharmacol Ther 1985;27(1):37-47

The history, purification, structure, and mode of action of cholera enterotoxin (choleragen) have been reviewed, with particular emphasis on the immunological aspects. Today, the pathogenesis of cholera is better understood than many other infectious diseases. During the past decade and a half, the toxin has been purified, its subunit structure defined, its cell membrane receptor identified, and its effect on cell function explained in considerable detail. Concepts and methods elaborated for cholera toxin have proven to be very useful for research with other enterotoxins. While cholera toxin normally has direct access in nature only to human intestinal epithelial cells, the ubiquity of Gm₂ ganglioside on the surface of other mammalian cells has enabled the toxin to

serve as an excellent experimental tool for studying cyclic adenosine monophosphate-mediated reactions in a wide variety of cell systems. Although more specific and immediate remedies or antidotes remain to be developed and applied, the understanding of the pathophysiology of cholera has led to an unexpected reward of incredible value -- oral rehydration therapy -- practical treatment for diarrheal disease which will save millions of lives, particularly of children, annually. Unfortunately, only rudimentary knowledge has yet been obtained of effective prevention of cholera, though current studies offer promise for the development of useful and practical methods for the prevention of cholera and related diarrheal disease. The best protection yet obtained has been that provided by the living cholera vibrios growing in vivo. Recent evidence indicates that Vibrio cholerae, grown in vivo, express novel outer-membrane proteins which could contribute to immunity.

Finkelstein RA, Sciortino CV, McIntosh MA. Role of iron in microbe-host interactions. Rev Infect Dis 1983 Sep-Oct:5((suppl 4):S759-77

Fiocchi C see Elson CO

Fitzgerald JF, Clark JH. Chronic diarrhea. Pediatr Clin North Am 1982 Feb;29 (1):221-31

This paper discusses the pathogenic mechanisms in diarrhea, including osmotic and secretory diarrheas, bacterial overgrowth, defective anion exchange systems, mucosal damage, and dysmotility. A useful clinical classification of chronic diarrhea is based on character of stool - watery, fatty, or bloody. It is emphasized that a complete history and a thorough physical examination are essential for the evaluation and diagnosis, while hospitalization is often necessary for a complete evaluation. A detailed diagnostic approach to the problem of chronic diarrhea is outlined. A list of the causes of chronic diarrhea, classified according to the stool characteristics, is also provided.

Fitzgerald JF. Management of the infant with persistent diarrhea. Pediatr Infect Dis 1985 Jan-Feb;4(1):6-9

Fitzgerald JF see Arasu TS

Flett J. Jr. see Darrow DC

Flewett TH, Woode GN. The rotaviruses: brief review. Arch Virol 1978;57(1): 1-23

Aspects relating to rotaviruses which cause acute gastroenteritis in young animals of various species are discussed along with a historical background of epizootiologic diarrhea of infant mice. The pathogenicity of diarrheal viruses and their mode of action in humans and animals are reviewed. In humans, it has been shown that rotaviruses are responsible for 25% of cases of diarrhea among those aged under 1, 60% between the ages of 1 and 3, 20% between ages 4 and 6 and occasionally in older children. The clinical features of rotavirus infection in children are highlighted. Similar viruses in other species were studied and salient features of their pathogenic potential have been compared with that of rotaviruses. The relationship between viruses of different species and the methods of detecting rotavirus in feces are explained. Little 1s known of the histopathology of human rotavirus infections; the possible mechanisms of the causation of diarrhea are outlined. IgM antibodies appear

very rapidly, but IgG soon replaces the IgM in humans. Over 90% of children have acquired antibodies by the age of 6. The pattern of immunity in animals has been analyzed. Rotaviruses in pigs and calves isolated in the UK were virulent to gnotobiotic piglets resulting in death when fed within a day of birth. The morphology and characteristics of rotaviruses have been studied. Many workers have reported that a complete virion is between 65 and 70-75 nm. The density of the human virus is between 1.36 and 1.38. The presence of the outer-capsid layer is necessary for infectivity. Results of studies on rotavirus RNA are reported along with details of common banding patterns. Cross hybridization studies are required to determine how the RNAs of different rotaviruses are related. Studies done by others compared polypeptides of human and calf rotaviruses purified from feces. For studies on animal rotaviruses constructing a cryptogram is useful. Other members may fit into the same cryptograms for further study. Crucial aspects of immunity in humans and animals against rotaviruses infection are stated. Finally, the taxonomy and antigenic relationship of rotavirus is analyzed. Rotavirus infections are of great economic importance to farmers and stock breeders. Comments on prospects of the recently developed vaccine against rotavirus are given, with optimistic notes on its possible uses.

Flewett TH see Horzinek MC

Flores J see Kapikian AZ

Folio D see Auricchio S

Fondacaro JD. Intestinal ion transport and diarrheal disease. Am J Physiol 1986 Jan; 250(1, pt 1):G1-8

"The physiology of intestinal electrolyte transport is currently an area of intense research interest. Also, reports regularly appear that define possible roles of various endocrine, paracrine, and neurohumoral substances in regulating intestinal ion and water flux. A vast body of knowledge has appeared recently that focuses on the action of specific intracellular mediators or second messengers and certain biochemical events that are thought to be involved in this transport process. This area of research has drawn the attention of the clinical investigator as well as the basic scientist because of the implications of these findings to the understanding of secretory disorders of the gastrointestinal tract, in particular diarrheal disease. The purpose of this review is to focus on recent findings reported in three major areas: the physiology of intestinal electrolyte transport and its regulation; the pathophysiology of secretory diarrhea; and current thoughts and practices in the therapeutic approach to the disease." (Author's abstract)

Formal SB, Hale TL, Sansonetti PJ. Invasive enteric pathogens. Rev Infect Dis 1983 Sep-Oct;5(suppl 4):S702-7

"Invasive enteric pathogens of the <u>Salmonella</u> or <u>Shigella</u> genera initiate infections by invading the intestinal epithelium. Depending on the species, salmonellae either translocate across the mucosa of the small intestine and cause a systemic febrile disease or they evoke a localized inflammatory response in discreet areas of the infected mucosa. The latter type of infection is chracterized by gastroenteritis, and a choleragen-like enterotoxin may contribute to the symptomatology. <u>Shigellae</u> can also evoke diarrheal episodes; however, classic shigellosis is <u>Characterized</u> by localized invasion

of the colonic epithelium, with inflammation and ulceration of the mucosa. Derangement of the colonic mucosa is manifested in the bloody, mucoid stool characteristic of bacillary dysentery. Genetic analysis of invasive enteric pathogens has shown that extrachromosomal elements (plasmids) are required for full expression of virulence in Salmonella typhimurium, Yersinia enterocolitica, Shigella sonnei, and Shigella flexneri. In the latter species, at least three chromosomal regions are also necessary for virulence." (Authors' abstráct)

Formal SB see Hale TL

Formal SB see Sansonetti PJ

Fortson WC, Tedesco FJ. Drug-induced colitis: a review. Am J Gastroenterol 1984 Nov;79(11):878-83

Aspects relating to drug-induced colitis are reported. various drugs and toxins are being recognized with increasing frequency to cause colonic damage and should be included in the differential diagnosis of colitis. gold has been implicated as the toxigenic agent. The clinical gold has been implicated as the toxigenic agent. The clinical course of gold-induced colitis is stormy and prolonged, frequently complicated by hypoproteinemia and bacterial sepsis. Therapy is directed toward nutritional, antibiotic and fluid support. The development of colitis in patients, undergoing therapy for malignancies, is not uncommon; most patients receive combination regimens as well as antibiotics and other drugs. The clinical courses are frequently complicated by agranulocytosis, sepsis, or hypotension. These patients are susceptible to neutropenic colitis, an ileocecal syndrome, the clinical features of which include fever, diarrhea, and ileus. many investigators suggest antineoplastic drugs play an integral part in this syndrome, other etiological factors, such as malignant infiltration, bacterial infections, ischemia, antibiotic therapy, are other potential Antibiotic-associated pseudomembranous colitis is discussed. Clostridium difficile is known to cause pseudomembranous colitis. Oral vancomycin, bacitracin, and metronidazole have been used as treatment, but relapses occur in up to 20% of patients with antibiotic-associated pseudomembranous colitis. Acute hemorrhagic colitis is described in association with oral ampicillin or Colitis trigerred by use of various drugs, such as amoxicillin therapy. vasopressin, penicillamine, ergot, methyldopa, etc., are described. Action of other agents in causing colitis are reported. Drugs which are given in enema form are described in relation to their action in this disease condition. Resultant colitis, ranging from mild inflammation to peritonitis, stricture formation, perforation, and death, are described.

Fox JG. Campylobacteriosis - a "new" disease in laboratory animals. Lab Anim Sci 1982 Dec;32(6):625-37

Though <u>Campylobacter</u> (Vibrio <u>fetus</u>) has been known as a pathogenic and commensal bacterium in domestic animals for many years, the bacterium only recently became suspect as a common cause of diarrheal disease in humans and laboratory animals. Because of the growing awareness of <u>Campylobacter fetus</u> ssp. <u>jejuni</u> (<u>C. jejuni</u>) as a pathogen in humans, researchers are seeking experimental animal models to explore the pathogenic mechanism exerted by this organism. Campylobacteriosis is also poorly understood, and the role of laboratory animals as a source of infection for humans and other animals has not been fully explored. With the development of proper culture techniques, C.

jejuni has been isolated from humans with diarrhea as frequently as Salmonella or Shigella species. Laboratory animal models are being developed to study the pathogenesis of Campylobacter-induced diarrhea; also, identification of C. jejuni is occurring more frequently in a variety of laboratory animals, whether asymptomatic carriers or those with clinical diarrhea. Animals shedding C. jejuni have been incriminated as a source of Campylobacter diarrhea in humans. This review article presents existing information on the prevalence and incidence of C. jejuni, as a purported cause of enteritis in laboratory animals, and on zoonotic implications and the role of animal models in Campylobacter research. Similarities of Campylobacter infections in humans and laboratory animals are also emphasized. (Modified author's abstract)

Freeman HJ, Sleisenger MH, Kim YS. Human protein digestion and absorption: normal mechanisms and protein-energy malnutrition. Clin Gastroenterol 1983 May:12(2):357-78

Freier S, Kuperman O. Immune regulation in the gastrointestinal tract. J Pediatr Gastroenterol Nutr 1983;2(suppl 1):S310-4

Freston JW see Fingl E

Freter R, Jones GW. Models for studying the role of bacterial attachment in virulence and pathogenesis. Rev Infect Dis 1983 Sep-Oct:5(suppl 4):S647-58

"Simple in vitro tests for bacterial adhesion can indeed identify the various immunologic, physicochemical, adhesive mechanisms of bacteria on an biochemical, and genetic basis. Difficulties in interpretation arise, however, when attempts are made to relate the presence of a given adhesion to the colonizing ability or virulence of a bacterium. The reasons for this confusion are threefold: (1) there is more than one basic mechanism by which bacteria may associate with mucosae; (2) numerous intervening reactions in the mucosal microenvironment modify the various steps leading to association; and (3) mucosal association may sometimes be detrimental to a bacterium. association with the mucosa, therefore, is determined by the final equilibrium established as a consequence of various synergistic and antagonistic reactions. An understanding of such a complex, interdependent system of reactions cannot be gained solely by studying each of its component parts in isolation. complex models, such as those developed in experimental animals, are therefore required, and the relationship between adhesion and colonization must be explored within the conceptual framework employed by ecologists." (Authors' abstract)

Fromm H, Malavolti M. Bile acid-induced diarrhoea. Clin Gastroenterol 1986 Jul;15(3):567-82

"Three types of bile acid-induced diarrhoea can be distinguished. The best documented and most common entity is represented by type I bile acid malabsorption, which occurs as the result of a pathologically, anatomically defined ileopathy. Type 11 bile acid malabsorption is found in the setting of a morphologically completely normal ileum. This primary disorder of bile acid transport, which has been described in only a few paediatric and adult patients, appears to be rare. The third variety of bile acid malabsorption is characterized by the history of a previous cholecystectomy and/or by the gastroenterological conditions. acid Severe bile other presence of Even in the malabsorption is relatively uncommon in the type III syndrome.

presence of severe bile acid malabsorption, patients with this condition are rarely found to have secretory concentrations of faecal bile acids, and/or rarely respond satisfactorily to cholestyramine. Present data suggest that bile acids play no significant role in the pathogenesis of idiopathic diarrhoea. A careful history, the measurement of stool weight and pH, a therapeutic trial of cholestyramine and the performance of a bile acid test, such as a bile acid breath test, can be used to establish the diagnosis of bile acid diarrhoea. Cholestyramine is the treatment of choice and is virtually always effective in this syndrome." (Authors' abstract)

Fry RD see Kodner IJ

Fusco P see Levine MM

Galindo E see DuPont HL

Gall DG, Hamilton JR. Chronic diarrhea in childhood: a new look at an old problem. Pediatr Clin North Am 1974 Nov;21(4):1001-17

Gamble AH see Darrow DC

Garagusi VF see Chretien JH

Gardner FH. Nutritional management of chronic diarrhea in adults. JAMA 1962 Apr 14;180(2):147-52

Chronic diarrhea in the adult may be associated with a variety of nutritional deficiencies. These deficiencies are often found with abnormal anatomic changes or mucosal alteration of the small bowel. This paper discusses the impairment of electrolyte, vitamin and mineral absorption and general nutritional depletion, e.g. steatorrhea and azotorrhea. These deficiencies can be produced by several mechanisms. Bacterial reactions that initiate chronic diarrhea are related mostly to anatomical lesions that allow areas of stasis for an excessive proliferation of the normal bowel bacterial flora. Such cul-de-sacs may impair nutrition by (1) bacterial competition for nutrients, or by (2) producing toxic elements that irritate the bowel mucosa and decrease absorption of all foodstuffs. Antibiotic therapy is, therefore, important to improve nutritional status of patients with anatomical defects. Impaired fat absorption may be associated with pancreatic insufficiency. In such cases, enzyme replacement with pancreatic extracts is a well-known practice. Available diagnostic procedures permit the physician to classify bowel disorders that may be treated by surgical correction, antibiotics or specific dietary programs.

Gardner JD. Plasma VIP in patients with watery diarrhea syndrome. Am J Dig Dis 1978 Apr;23(4):370-6

Garelick H see Feachem RG

Gasbarrini G see Corazza GR

Gaskin KJ see Soutter VL

Gateere R see Kinoti SN

Geboes K see Vantrappen G

George WL. Antimicrobial agent-associated colitis and diarrhea: historical background and clinical aspects. Rev Infect Dis 1984 Mar-Apr;6(suppl 1): \$208-13

In the late 1970s, Clostridium difficile was found to cause a lethal, clindamycin-induced ileocolitis in the Syrian hamster; this animal model has been an invaluable aid to the understanding of antimicrobial agent-induced diarrhea in humans. C. difficile is involved in almost all cases of pseudomembranous colitis and in approximately one-fourth of cases of antimicrobial agent-associated diarrhea in humans in which pseudomembranes are not detected. The presenting signs and symptoms of C. difficile-induced diarrhea are quite variable. Mild diarrhea may be the only finding in the least severe form of the disease, whereas patients with severe disease may have high fever, leukocytosis, severe abdominal cramping, marked abdominal tenderness, and profuse diarrhea. Occasionally, symptoms may be so marked as to stimulate an acute intra-abdominal catastrophe. Diagnosis of C. difficile-induced disease usually is made by detecting C. difficile cytotoxin in the feces of a patient. Cytotoxin in feces of infants is not a sign reliable, however, because of the high incidence of an asymptomatic carrier state in this age group. Appropriate therapy includes discontinuation of the offending antimicrobial agent and administration of oral vancomycin when specific antibacterial treatment is indicated. (Modified author's abstract)

Georgescu MB. [Colonization factors in enterotoxigenic <u>Escherichia coli</u>]. Rev Ig [Bacteriol] 1985 Apr-Jun;30(2):115-30

Germanier R. Oral vaccination against enteric bacterial infections: an overview. Infection 1984 Mar-Apr;12(2):138-42

"The present situation and the future prospects of the use of oral vaccines against the major enteric diseases, typhoid fever, shigellosis and cholera are discussed in this paper. No significant protection could be demonstrated for oral inactivated whole-cell vaccines. In contrast, an oral live vaccine based on the attenuated Salmonella typhi strain Ty 21a was highly efficacious in volunteer challenge studies and in a controlled field trial. Two attenuated strains are presently being tested in volunteer studies as candidate vaccines against shigellosis; one uses S. typhi Ty 21a and the other Escherichia coli K-12 as the carrier for shigella antigens. Experimental challenge studies in volunteers showed that recovery from clinical cholera confers long-lasting protection. The goal of present research is to develop a vaccine that mimics the events of clinical cholera without causing disease." (Author's abstract)

Gertler S, Pressman J, Cartwright C, Dharmsathaphorn K. Management of acute diarrhea. J Clin Gastroenterol 1983 Dec;5(6):523-34

Ghandur-Mnaymneh L see Rams H

Ghatikar KN. Oral rehydration therapy--an overview. Q Med Rev 1980 Oct;31 (4):1-12

Ghoot RB see Wagh MG

Giannella RA. Chronic diarrhea in travelers: diagnostic and therapeutic considerations. Rev Infect Dis 1986 May-Jun;8(suppl 2):5223-6

"Although the epidemiology, etiology, and pathophysiology of travelers'

diarrhea are well understood, the long-term consequences of this syndrome poorly appreciated. Many people struck with travelers' diarrhea do not completely recover but rather develop one of several chronic diarrheal syndromes. In some patients an episode of travelers' diarrhea seems to unmask a preexistent, underlying gastrointestinal disorder. The frequency of these complications and the magnitude of this problem are unknown. The differential diagnosis, diagnostic workup, and treatment of such patients have been outlined; unfortunately, many patients with chronic diarrhea elude diagnosis. and treatment is problematic. Some of the questions that must be answered include the following: (1) What is the magnitude of the problem of chronic diarrhea following travelers' diarrhea? (2) What is the natural history of patients with this syndrome? (3) Which individuals are most likely to develop a chronic diarrheal syndrome, and can they be identified in advance? (4) Are new etiologic agents involved in these chronic diarrheal syndromes? Answers to these questions will require well-organized, multicenter studies of a large number of travelers to various locales." (Author's abstract)

Gibbs KR. There is no safe water in rural Bangladesh: so what about the kids? Shishu Diganta (Dhaka) 1980 Dec;(9):25-7

In Bangladesh, one operational tubewell is available for use of every 160 persons. Spot checks carried out so far indicate that the tubewell gives totally uncontaminated water in virtually every case, while the pump does not. The reasons are: contaminated water is used for priming, and villagers (particularly children) will touch the spout, often after defecation, thereby contaminating it and even assisting the spread of diarrheal disease during epidemics. Frequently, containers add to the contamination of the water being carried. It is observed, however, that handpumps can and do give better health to children. There is a need to organize how they are to be used by informing everyone - men, women, and children - that: (i) small families are healthier; (ii) high water users from tubewells are healthier; (iii) women's privacy at the pump is healthier; (iv) washing clothes at the pump is healthier; (v) playing at the pump is healthier. There is a need to inform people why this is so.

Gilles HM. Progress in the pathogenesis and therapy of parasitic diseases. Scand J Infect Dis 1982;(suppl 36):12-4

Gilman RH. Bacillary dysentery. Compr Ther 1984 Oct;10(10):14-9

Giorgi PL, Catassi C, Coppa GV, Valentini V, Sbarbati A. [New protagonists of infectious diarrhea in childhood. Pathogenetic and clinical aspects]. Minerva Pediatr 1985 Jan 31;37(1-2):29-48

Gitler C, Calef E, Rosenberg I. Cytopathogenicity of Entamoeba histolytica. Trans R Soc Lond [Biol] 1984 Nov 13;30(1131):73-85

Glass RI see Kapikian AZ

Goldman H. Acute versus chronic colitis: how and when to distinguish by biopsy [editorial]. Gastroenterology 1984 Jan;86(1):199-201

This editorial highlights the findings of Surawicz and Belic (1984) which suggests that rectal mucosal biopsy can be used to distinguish acute and chronic colitis in a high proportion of cases. Seven histologic features noted

in the rectal mucosal biopsy specimens proved to be highly discriminant. features are indicative of chronic colitis either of the ulcerative or Crohn's type. Granulomas and basally located giant cells, although more common in chronic colitis and in Crohn's disease, can be seen in acute disorders, such as Basal-lymphoid aggregates have been noted in chronic serious infections. colitis. One or more of these discriminant features are present in 79% of the cases of chronic irritable bowel syndrome. Superficial acute inflammation or giant cells in the face of normal crypt architecture is highly suggestive, but not diagnostic, of acute colitis. Biopsy can be done as early as 3 or 4 weeks after the onset of symptoms. If the biopsy specimen is completely normal, e.g. no crypt distortion or atrophy, no epithelial regeneration or Paneth cell metaplasia, the patient has self-limited colitis. The patient with irritable bowel syndrome would show crypt irregularity or evidence of persistent inflammation. Signs of continuous inflammation with a longer duration indiate chronic ulcerative colitis. In Crohn's disease, the disorder tends to be more often focal. In any clinical situation, one must collect all available information to establish an exact etiology of the disease. It is concluded that the technique used by Surawicz and Belic can serve as a standard for future structural studies.

Goldschmidt B. Microscopic stool-gazing, a guide to the cause and cure of chronic and recurrent diarrhoea in children. S Afr Med J 1966 Feb 26;40(9): 191-5

Microscopic examination of the fresh, warm stool is a helpful procedure in defining the cause of chronic and recurrent diarrhea in children. It has been suggested that the examination should be performed by the clinician himself, which will enable him to make an immediate diagnosis and consequently institute an appropriate treatment at an early stage. This report also outlines and illustrates the common microscopic findings. It is indicated that infection often initiates malnutrition and may also be responsible for its continuation. Severe cases of kwashiorkor and marasmus can be remedied after treatment is initiated with suggestive guidelines made available from an early microscopic examination of the patient's stools.

Gopalan C. Oral rehydration therapy - the need for a proper perspective. Food Nutr Bull 1986 Sep;8(3):69-70

The promotion of oral rehydration therapy (ORT) has checked undesirable trends and has been quite effective in controlling dehydration timely. importance is being attached to the composition of the oral electrolyte solution or "adaptations" of the formula to suit home conditions and to methods of ensuring supplies and deliveries of oral electrolyte packets to rural homes. These measures have to be seen in the proper perspective, which is mostly Oral rehydration addresses itself to the problem of lacking at present. dehydration, but can not eradicate the basic factors responsible for diarrhea. Developing countries have attempted to contain diarrheal diseases through distribution of oral electrolyte solution packets. The root causes of the problem should be corrected by (a) providing safe water and basic facilities for excreta disposal, (b) improving environmental sanitation, (c) giving health education to promote personal and environmental hygiene and safer procedures of food handling and child feeding, and (d) improving the nutritional status to levels which will endow with an adequate immune competence. Adequate energy and protein intake should be enhanced even during the acute phase of diarrhea to compensate for the loss of nutrients, to catch up on growth and restore

immunocompetence. Health agencies should educate workers on dietary management and on the use of ORT. Providing safe water and excreta disposal facilities alone will not help unless personal hygiene is emphasized for preventing intrahousehold contamination of foods. The prevention and control of diarrheal diseases in children of developing countries can only be achieved if the basic factors involved are adequately addressed.

Gorbach SL see Joiner KA

Gorbach St. see Simon GL

Goriup U, Shmerling DH. [Dietary therapy of intestinal diseases in childhood]. Ther Umsch 1978;35(8):673-8

The dietary management of intestinal enzyme deficiencies, such as lactase and sucrase-isomaltase deficiency and that of the major malabsorptive states, such as celiac disease and cow's milk protein intolerance, consists in the exclusion of the offending nutrient from the diet, which should otherwise remain nutritionally as adequate as possible. The practical aspects of these diets are described in detail. General nutritional support is indicated for catabolic patients with chronic inflammatory bowel diseases (ulcerative colitis and Crohn's disease) and with exocrine pancreatic insufficiency. This is best achieved with elemental diets or parenteral nutrition. (Modified authors' abstract)

Gorziglia M see Kapikian AZ

Goscienski PJ. Zoonoses. Pediatr Infect Dis 1983 Jan:2(1):69-80

Gothefors L see Svennerholm A-M

Gottlieb RP. Dehydration and fluid therapy. Emerg Med Clin North Am 1983 Apr; 1(1):113-23

Gotz VP, Rand KH. Medical management of antimicrobial-associated diarrhea and colitis. Pharmacotherapy 1982 Mar-Apr;2(2):100-9

Gastrointestinal complications, including diarrhea, may occur with virtually all antimicrobial agents. Such diarrhea may represent either a common. nonspecific adverse effect, or it may be one of the manifestations of antimicrobial-associated colitis. Clostridium difficile and a neutralized by C. sordellii antitoxin have been isolated from the stools of nearly all patients with antibiotic-associated pseudomembranous colitis, many patients with antimicrobial-associated colitis, and approximately 20% of those with antimicrobial-induced diarrhea. Demonstration that C. difficile is responsible for cytotoxin production has allowed for specific therapy for these disorders. General treatment measures include discontinuation of the causative antimicrobial agent(s), bowel rest, and supportive care with fluids, electrolytes, and colloids, if necessary. Antiperistaltic agents corticosteroids are not recommended. Various antimicrobials potential efficacy in treating antimicrobial-associated colitis in humans. Oral vancomycin is currently the treatment of choice and is very active against C. difficile in doses of 125-500 mg by mouth taken every 6 h. potentially useful but inadequately tested antimicrobials include metronidazole (500 mg by mouth every 8 h) and bacitracin (25,000 units by mouth every 6 h).

Tetracvcline has been employed with some success in antiblotic-associated diarrhea, although it is as yet untested in humans with antimicrobial-associated colitis and may induce diarrhea itself. miconazole and rifampin are highly effective against C. difficile in vitro. but have not been evaluated in antimicrobial-associated colitis. Anion-exchange resins bind the cytotoxin in antimicrobial-associated colitis. Cholestyramine has been used with variable response in oral doses of 4 g every 6 to 8 h. Since these resins may also bind vancomycin, resulting in lowered vancomycin concentrations in the stool, a combination therapy should be used cautiously. With specific therapy directed against the toxin and aggressive supportive therapy, surgical intervention is rarely necessary. More investigations have been directed at using bacterial preparations to suppress C. difficile by restoring the normal flora. The development of immunological agents (i.e. vaccines, toxoids, antitoxins) for the prevention or treatment of antimicrobial-associated colitis would be a significant advance in therapy. (Modified authors' abstract)

Gracey M. Antibiotic and antiparasitic therapy in chronic diarrhea. <u>In:</u> Lebenthal E, ed. Chronic diarrhea in children. New York: Raven Press, 1984:469-76

Gracey M. Bacterial diarrhoea. Clin Gastroenterol 1986 Jan; 15(1):21-37

"Bacterial infections are important causes of diarrhoea in infants and children, particularly in developing countries and in other settings where standards of personal and community hygiene are low. Knowledge of bacterial diarrhoeas has been significantly expanded in recent years by the finding that many episodes of acute diarrhoea are due to infections with bacteria which produce enterotoxins that interfere with intestinal fluid and electrolyte transport (the 'enterotoxigenic' diarrhoeas). Several "newer' bacterial agents have also been identified which would not have been detected in earlier studies of the epidemiology of infective diarrheas; these include Aeromonas, Campylobacter, Clostridium difficile and Yersinia. Another important advance has been new knowledge about mechanisms by which bacteria cause diarrhea; this has led to the widespread successful application of oral rehydration therapy in treatment of acute watery diarrheas." (Author's abstract)

Gracey M, Burke V. Characteristics of <u>Aeromonas</u> species and their association with human diarrhoeal disease. J Diarrhoeal Dis Res 1986 Jun;4(2):70-3

The characteristics of Aeromonas species and their association with human diarrheal disease are examined. Evidence implicating Aeromonas species as human intestinal pathogens is outlined. Aeromonas species were first considered to be a cause of gastroenteritis in 1961 in a nursery in Colombia. Subsequently, Aeromonas-associated diarrhea was reported from North America, Europe, India, Africa, Australia, and Indonesia. Their role as enteric pathogens is still considered by some to be "not proven'. Reported rates of isolation of fecal Aeromonas species vary a great deal. Variation in laboratory methodology, geographical differences in Aeromonas-related infection and age exert significant influences on isolation rates. Some studies have compared isolation rates of Aeromonas in patients with and without diarrhea in Australia, Italy and the United Kingdom. These studies provide strong supportive evidence for Aeromonas species having a pathogenic role in human diarrheal disease. Classification and identification methods for Aeromonas species are reported, although methods for isolation and identification of the

organisms are not uniform. Layered blood agar plates with 10 ug/ml of ampicillin, a level below the usual inhibitory concentration for most species, can grow greyish, rounded colonies of Aeromonas. The strains are classified as A. sobria, A. hydrophila, and A. caviae. The patterns of clinical illness due to these organisms are highlighted. Most studies have found fecal isolates of Aeromonas species from stools of young children and in older adults (60 years and over). Many reports reveal that Aeromonas have been isolated from stools obtained from healthy persons as well. Aeromonas species are widely distributed in animal and environmental sources, especially in aquatic environments. Virulence factors and biotypes are discussed. Further studies of the epidemiology of Aeromonas-associated diarrhea are needed, particularly in developing countries, where waterborne infections are common. These should be combined with studies of the virulence factors produced by Aeromonas species and the immune responses in affected patients.

Gracey M. Chronic diarrhoea in protein-energy malnutrition. Paediatr Indones 1981 Nov-Dec;21(11-12):235-9

This review describes chronic diarrhea in protein-energy malnutrition (PEM). Studies have shown that bacterial contamination of upper-intestinal secretions is common in malnourished children. Bacterial overgrowth in the proximal gut can produce a wide range of clinical effects, including steatorrhea, carbohydrate malabsorption, hypoproteinemia, vitamin B12 deficiency, and can also be associated with macrocytic anemia and iron deficiency. malnutrition, these problems may operate with recurrent and chronic gastrointestinal infections damaging the intestinal mucosa. Escherichia coli present in the gastrointestinal tract in malnourished children produce toxins which interfere with the intestinal absorption of fluids and electrolytes. There are significant differences between the appearance of gastrointestinal mucosa in tropical and nontropical regions. The changes the gastrointestinal tract due to diarrhea associated with malnutrition include thinning of the gut wall, marked flattening and broadening of the intestinal villi, inflammatory infiltration of the lamina propria and alteration of the enterocytes from columnar to cuboidal or squamous, loss of secretion of hydrochloric acid in the upper intestine. These could contribute to the heavy bacterial contamination of the upper gut of malnourished children. The possible contributions of impaired digestion and absorption, increased mucosal and trans-mucosal losses and the role of intraluminal factors are uncertain. Disaccharide deficiency, rickets, and vitamin E deficiency are also associated with PEM as seen in studies done in Ethiopia, Thailand, and in the Middle East. Anemia in 10% of the malnourished children has been reported. Further studies on the effects of PEM on the gastrointestinal mucosa should be carried out.

Gracey M. The intestinal microflora in malnutrition and protracted diarrhea in infancy. <u>In</u>: Lebenthal E, ed. Chronic diarrhea in children. New York: Raven Press, 1984:223-36

Gracey M, Burke V. Sugar-induced diarrhoea in children. Arch Dis Child 1973 May;48(261):331-6

This paper reviews the knowledge of disorders of carbohydrate digestion and absorption in children. Practical rather than theoretical aspects are emphasized. Types of carbohydrate malabsorption, symptoms, and diagnosis of sugar intolerance and treatment of individual types of malabsorption are discussed. Application of this knowledge promises to be beneficial to children

throughout the world, because these disorders are common and potentially lethal, but eminently treatable.

Gracey MS. Nutrition, bacteria and the gut. Br Med Bull 1981 Jan:37(1):71-5

In a variety of clinical situations, the relative sterility of the small intestine is disturbed, with bacterial contamination of the upper-intestinal secretions, and this has numerous harmful effects on the human host. include malabsorption of nutrients, vitamins, and minerals. Much of this is due to the combined effects of poor nutrition and unsatisfactory living conditions. Profuse bacterial overgrowth of the proximal gut can produce a steatorrhea. of clinical consequences, including malabsorption, hypoproteinemia, vitamin B12 deficiency and associated macrocytic anemia, and iron deficiency. Repeated and chronic gastrointestinal infections can damage the normal digestive and absorptive functions of the intestine. In malnourished children, the histological abnormalities which occur in the proximal small intestinal epithelium are quite severe. These include thinning of the gut wall, marked flattening and broadening of the villi, extensive inflammatory infiltration of the lamina propria transformation of the shape of the enterocytes from columnar to cuboidal or squamous. Recent studies have shown that the gastric mucosa is also damaged. Sometimes life-threatening dehydration and metabolic acidosis have been related to bacterial overgrowth of the upper-intestinal contents. histological damage is also important from a functional point of view, particularly in children. Secondary depression of disaccharidase activity is a common feature of the clinical spectrum of diarrhea in malnutrition, and lactose intolerance occurs in about a quarter of patients. The underlying reasons for diarrhea in any malnourished individual might be diverse, and several factors may operate simultaneously.

Grand RJ see Motil KJ

Green EC. Traditional healers, mothers and childhood diarrheal disease in Swaziland: the interface of anthropology and health education. Soc Sci Med 1985;20(3):277-85

A study of beliefs and practices relating to childhood diarrhea, relying primarily on traditional healers as informants and survey respondents, revealed an indigenous classification of childhood diarrhea into 3 main types. Enemas are used as a treatment in two types of more serious diarrhea regarded as due to unnatural causes. Most children with diarrhea are taken to clinics only after home treatments and those prescribed by traditional healers have failed, by which time a child may be severely dehydrated. The role of oral rehydration and strategies for health education are discussed in the context of Swazi culture. Swazis believe that fluids, especially breast milk, are necessary to maintain a child's strength. It is suggested that this belief and the resulting practice should be reinforced; nutrition and dehydration education could be presented in the context of keeping up a baby's strength. Encouragement of 'home remedies', such as use of sorghum or maize porridge, may prove to be effective in preventing dehydration in children.

Greene HL. A pathophysiologic approach to dietary management in patients with protracted diarrhea and malnutrition. <u>In:</u> Winter RW, Greene HL, eds. Nutritional support of the seriously ill patient. New York: Academic Press, 1983:181-94

Greenough WB, III, Rabbani GH. Antisecretory and antimicrobial drugs for treating diarrhoea. In: Holmgren J, Lindberg A, Mollby R, eds. Development of vaccines and drugs against diarrhea; proceedings of the 11th Nobel Conference, Stockholm, 1985. Lund: Studentilitteratur, 1986:270-7

In the past 3 years, a substantial number of drugs which have been shown experimentarily to enhance intestinal absorption, reduce secretion or, both. have been brought to clinical trial on the most severe testing ground, clinical cholera. However, most of them have failed to show a significant clinical effect, some have had a detectable but modest inhibitory influence on fluid loss in patients with severe cholera. Until now, chlorpromazine and infcotinic acid have had the most influence. Some of them have side-effects and are very costly. Thus, as of the present time, no truly practical low-cost agents can be recommended for general use in the treatment of severe acute watery diarrhea. The case of antimicrobial drugs is also challenging. Over the past 3 years, drug resistance of a high degree has rapidly arisen in virulent enteric pathogens which are currently responsible for high morbidity and mortality related to diarrheal diseases. Newer antibiotics have not shown great promise in overcoming this problem. A major area which requires careful study, however, is the diarrhea lasting for more than 5-7 days which often results in prolonged intestinal malabsorption of the limited food materials available in developing countries. Careful controlled trials are needed to assess the impact of antibiotic treatment on the long-term nutritional status of children. (Modified authors' abstract)

Greenough WB, III. Bacterial diarrhoeal diseases: current concepts on etiology and pathogenesis. Southeast Asian J Trop Med Public Health 1982 Sep; 13(3):319-24

This overview touches on some of the diarrheal disease dogmas that relatively recent research has proven untrue - such as the widespread beliefs that cholera and cholera-like diseases were due to destruction of the gut epithelial lining, with consequent plasma loss; that Gram-negative bacteria do not produce disease-causing exotoxins; and that watery diarrheas undiagnosable 20 years ago were due to helminths and parasites. Now, knowledge of bacteria's role in diarrheal diseases has changed markedly - the key having been determination of the true cholera pathogenesis mechanism, thereby confirming John Snow's 19th century hypothesis. Mentioned are such historic turning points as purification of cholera toxin, discovery that the cholera toxin/adenylate cyclase/cAMP mechanism results in chloride secretion and interference with sodium chloride secretion and interference with absorption, and pinpointing of the toxin/receptor mechanism. Mentioned too are the discoveries that other bacteria, such as \underline{E} , \underline{coli} and other Gram-negative $\underline{Enterobacteriaceae}$ (including organisms, such as vibrios not agglutinated by the classical Vibrio cholerae antisera), cause diarrheal disease. The paper also touches on the invasive diarrheas, noting that not as much is known about how the causative organisms adhere to and invade the intestinal lining, as is known about the cholera mechanism. Little is known yet about the humoral control mechanism of the process of digestion, absorption and secretion in diarrheas, although new intestinally related hormones have been identified in the past decade. The paper concludes with a look at the knowledge areas most permit diarrheal diseases prevention and treatment: adherence/receptor process that might allow development of blocking drugs: post-adhesion process, so that the toxin's action could be averted; and the specific components that mediate the disease, so that effective vaccines could be developed.

Greenough WB, III. Principles and prospects in the treatment of cholera and related dehydrating diarrheas. <u>In:</u> Ouchterlony O, Holmgren J, eds. Cholera and related diarrheas: molecular aspects of a global health problem. Basel: Karger, 1980:211-8 (43rd Nobel Symposium)

This paper reviews diarrhea treatment practices. Presently the therapeutic challenge is to teach families to use an appropriate oral rehydration solution. In the treatment of cholera and the related diseases, the rate of fluid loss needs to be diminished by an agent which is inexpensive, readily available, and nontoxic. The ability to rehydrate cholera patients by mouth decreases very Intravenous fluid rapidly as the rate of purging exceeds 100 ml/kg.24 h. therapy would be needed in such cases. An ideal oral solution should contain sodium and glucose. Sucrose is an acceptable substitute where glucose is expensive and not available. Clinical trials should be carried out to examine whether rice or potato can be used as proteins and peptides. In the case of cane sugar, the advantage lies in the presence of potassium in it. A simple oral fluid replacement contains table salt and sugar in water. Recently in Dhaka, Bangladesh, it was shown that oral rehydration replacement successful, though in some cases, acidosis was associated. Basic studies to test the hypothesis that a receptor blockade would indeed prevent fluid loss are needed. To accomplish this goal, more information about the pathogenicity of Vibrio cholerae and their genetic loci is necessary. The inhibition of adenylate cyclase stimulation and interventions in ion transport are discussed. A fundamental enquiry into the existing health beliefs and practices in the matrix of specific geocultural settings is essential to any application of current knowledge in the prevention or treatment of diarrhea.

Greenough WB, III. Protozoal, bacterial and viral diarrhoea: a common mechanism [editorial]. J Diarrhoeal Dis Res 1984 Jun;2(2):68

Greenough WB, III. "Super ORT" (editorial). J Diarrhoeal Dis Res 1983 Jun;1 (2):74-5

Considering the setting where the full oral rehydration solution (ORS) formulation with sterile drinking water is not available, 3 essential ORS components are identified: water, salt (NaCl) and a carrier substance (glucose). A simple sea salt/crude cane sugar ORS, used regularly in children, was found to result in significantly improved nutrition. It is anticipated that nutrition can be enhanced even further, by using cereals and proteins as components of ORS. Thus, both fluid-electrolyte malnutrition and protein-energy malnutrition can be treated or prevented, by using super ORS solutions.

Greenough WB see Chandra RK

Greenough WB, III see Molla AM

Grigoryev AV <u>see</u> Zhalko-Titarenko VP

Groschel DHM see Guerrant RL

Gross MH. Management of antibiotic-associated pseudomembranous colitis. Clin Pharm 1985 May-Jun;4(3):304-10

Gross RJ. Escherichia coli. J Infect 1983 Nov;7(3):177-92

Gross RJ, Rowe B. <u>Escherichia coli</u> diarrhoea. J Hyg (Lond) 1985 Dec;95(3): 531-50

Although Escherichia coli was first isolated in 1885, most of the work done during the next 50 years was aimed at distinguishing E, coli and related organisms from known pathogens, such as Salmonella and Shigella. In 1933, serotyping was first used to show an association between a particular E. coli strain and infantile enteritis. Thereafter, epidemiological studies and outbreak investigations using similar methods continued for almost 4 decades. Later (in 1961), it was reported that 17 "0" serogroups of \underline{E} , \underline{coli} had been implicated as possible causes of epidemic infantile enteritis. These infantile enteropathogenic \underline{E} , \underline{coli} (EPEC), having been discovered by epidemiological studies using serotyping, belonged by definition to a restricted range of serogroups. In the early 1970s, the development of the suckling-mouse test and tissue-culture tests for enterotoxins greatly stimulated the studies of the pathogenic mechanisms of \underline{E} . \underline{coli} diarrhea. During the later part of the decade, \underline{E} . \underline{coli} strains that cause diarrhea were considered as belonging to 3 groups -- $\overline{\text{EPEC}}$, enterotoxigenic $\underline{\text{E. coli}}$ (ETEC), and enteroinvasive $\underline{\text{E. coli}}$ (EIEC). In environments where good hygiene and nutrition are prevalent, $\overline{\text{ETEC}}$ are uncommon, although they occasionally cause outbreaks of diarrhea among infants in hospital nurseries and among individuals of all ages in the community. ETEC are one of the most common causes of diarrhea among those traveling from such areas to regions with poor hygiene, especially in the tropics. In the developing countries, ETEC are responsible for a high proportion of the acute diarrheas of childhood and, therefore, contribute significantly to the high level of mortality. During the first half of the 1980s, the understanding of genetics of virulence of ETEC and EIEC has developed greatly. Studies of verotoxin and entero-adhesion have begun to explain at least some of the mysteries of EPEC. Further complexity has, however, been added by the discovery of verotoxin entero-hemorrhagic strains and entero-adherent strains that do not belong to accepted EPEC serogroups. It has been confirmed by oral challenge experiments in human volunteers that the original colonization-factor antigen I (CFA/I), ETEC strain causes diarrhea, while its laboratory derivative without CFA/I does not. The development of suitable vaccines against ETEC may be possible, particularly oral vaccines directed against the fimbrial adhesions. While such vaccines could be widely used for the prevention of traveler's diarrhea, their distribution in the tropics and administration to an infant population present considerable problems.

Gruca MA see Soutter VL

Guerrant RL, Lohr JA, Williams EK. Acute infectious diarrhea. I. Epidemiology, etiology and pathogenesis. Pediatr Infect Dis 1986 May-Jun; 5(3):353-9

Guerrant RL, Shields DS, Thorson SM, Schorling JB, Groschel DHM. Evaluation and diagnosis of acute infectious diarrhea. Am J Med 1985 Jun 28;78(suppl 68):91-8

"The appropriate approach to the diagnosis and management of acute infectious diarrhea is determined by the frequency and setting of the illness, the recognizable causes or syndromes, the cost and yield of available diagnostic tests, and the treatability of the disease. Acute diarrhea affects everyone throughout the world from one to more than six times each year, depending on

age, location, and living conditions. The range of identifiable viral. bacterial, and parasitic etiologies is great, and the cost of indiscriminate use of etiologic studies for diagnosis is prohibitive. Because of its insensitivity for many organisms and poor selection of cases for testing, routine stool culture has been one of the most costly and ineffective microbiologic tests; the cost per positive result has traditionally exceeded \$900 to \$1,000. The appropriate treatment for the vast majority of cases (independent of their cause) is simple and effective: oral glucose- and electrolyte-containing rehydration solution. On the basis of an appropriate history and understanding of pathogenesis, fecal specimens can be selectively obtained and promptly examined for leukocytes and parasites, and the common noninflammatory diarrheas can be separated from the inflammatory infections in order to focus further studies on the latter group. The bacteria for which specific antimicrobial therapy should be considered usually cause inflammatory diarrhea in the United States. Therefore, only when the history or fecal leukocyte findings indicates an inflammatory process is it appropriate to to culture for the routine invasive bacterial pathogens. In sporadic inflammatory diarrhea, culture methods should include those for Campylobacter jejuni as well as <u>Salmonella</u> and <u>Shigella</u>. Several special circumstances may prompt a consideration of parasites (including <u>Giardia</u>, Entamoeba, <u>Strongyloides</u>, Cryptosporidium), Vibrio, Yersinia, Clostridium difficile, enterotoxigenic Escherichia coli, foodborne agents, or sexually transmitted pathogens. The practical value of specific identification of rotaviruses (by enzyme-linked is primarily Rotazyme, or electron microscopy) immunosorbent assay, epidemiologic, particularly in hospitalized infants or young children. such a selective approach to fecal culture will greatly increase its yield and can reduce the cost per positive result from \$1,000 to less than \$150." (Authors' abstract)

Guerrant RL, Sauer KT. Selective use of microbiological procedures for identification of etiologic agents of diarrheal illness. J Food Saf 1981;3: 145-64

A number of recent advances and a recognition of enteric pathogens and their mechanisms of virulence have revolutionized enteric microbiology in the last decade. With the large number of new methods to detect the many currently recognized agents, it is imperative that the clinical and epidemiological setting be carefully weighed to select the appropriate diagnostic tools to identify the incriminated agent. This review outlines a practical, realistic laboratory has of enteric infections. The approach to diagnosis substantially greater contribution towards evolving processes for identifying the exact etiology of diarrheal illness than it did a decade ago. The review also presents a summary of diagnostic studies that are potentially available and relates them to historical information, food or water sources and presumptive diagnoses. There are several clinical, epidemiologic preliminary laboratory tests that provide important clues toward arriving at the appropriate studies and an etiologic diagnosis. However, the challenge is greater than ever to link the microbiology laboratory with the appropriate clinical setting to specifically employ the appropriate diagnostic tests. While infection with some agents, such as Shigella sp., Entamoeba histolytica, Giardia lamblia, Campylobacter fetus ssp. jejuni, and possibly rotaviruses, may be initiated by small inocula and thus have potential for direct person-to-person spread, the majority of enteric pathogens require ingestion of high doses of infective organisms in contaminated food or water. It is virtually impossible to examine every stool specimen for all potential

pathogens. More than ever before, the microbiologist and clinician or epidemiologist must, therefore, work together to decide on the appropriate tests for each individual setting. It is only with concerted, selective application of newly developed techniques for recognition of enteric pathogens that their epidemiology and thus eventual control can be made possible.

Guerrant RL see Chandra RK

Guerrant RL see Pearson RD

Guerrant RL see Ravdin JI

Guerrant RL see Rohde JE

Guerrant RL see Williams EK

Guerry P see Walker RI

Guidolin A, Manning PA. Genetics of <u>Vibrio</u> cholerae and its bacteriophages. Microbiol Rev 1987 Jun;51(2):285-98

<u>Vibrio</u> cholerae, a Gram-negative bacterium, is the causative agent of <u>Cholera</u>. During the past few years, this organism has become more readily amenable to genetic analysis and has been a target of studies involving the use of recombinant deoxyribonucleic acid technology, primarily because of the interest in vaccine development. Thus, most emphasis has been given to cellular components likely to play a role in the pathogenesis of cholera, namely those that are associated with the cell surface or that are excreted extracellularly. <u>Vibrio</u> phages have been studied as tools for genetic analysis and as interesting biological entities. This article reviews the current status of the genetics of <u>V</u>. <u>cholerae</u> and its bacteriophages. (Modified authors' abstract)

Guillozet N. Measles in Africa: a deadly disease. Some personal comments. Clin Pediatr (Phila) 1979 Feb;18(2):95-100

Measles dominate childhood illnesses in tropical Africa with a wide variety of clinical conditions and severity of complications virtually unknown in the West. There is no apparent continental variation in measles virus, but environmental factors vary widely. The frequent association of severe measles with malnutrition and recent studies indicating impaired humoral, cellular, and mucocutaneous immunologic function in the malnourished may explain this perplexing disease. Prevention of early malnutrition combined with immunization with a heat-stable vaccine, currently unavailable, are needed to halt this often fatal African scourge. (Modified author's abstract)

Guy MW see Feachem RG

Gyr K. Infectious diarrhoea and gastrointestinal hormones: potential therapeutic implications. Scand J Gastroenterol 1983;18(suppl 84):135-40

"The gastrointestinal hormones, which are continuously increasing in number, have certain effects which could play a part in the pathogenesis of infectious diarrhoea. This refers especially to VIP, motilin, and entero-glucagon, the plasma concentrations of which are elevated in acute infectious diarrhoea,

cholera, and tropical malabsorption. They may act by stimulating intestinal secretion, inhibiting absorption, and altering intestinal motility. In addition, there are some hormonal effects such as those caused by glucagon on motility, by enkephalins on secretion, and by somatostatin on both, which have a therapeutic potential and deserve further investigation." (Author's abstract)

Gyr K. Toxin receptors and their pathogenetic significance. Acta Histochem 1984;(suppl 29):S95-102

The pathogenetic significance of interaction of toxin-receptors in disease is The worldwide problem of infectious exemplified by infectious diarrhea. epidemiology, etiology. with on diarrhea is presented focus Enterotoxigenic Escherichia coli (ETEC), rotavirus, pathophysiology. Vibrio cnolerae account for the highest number of diarrhea cases attributable to presently identifiable pathogens in the developing countries, while Campylobacter jejuni and Salmonellae are the leading pathogens associated with diarrhea in industrialized countries. Many intestinal pathogens are invasive Some bacterial agents cause and penetrate the intestinal mucosa and beyond. Viral diarrhea without invasion of the mucosa, but by producing enterotoxin. diarrhea is most likely to be related to the virus-induced loss of intestinal-absorptive capacity. V. cholerae and ETEC are the classical agents responsible for enterotoxigenic diarrhea though other bacteria also produce enterotoxins. Enterotoxigenic diarrhea is examined in the light of recent knowledge of toxin-receptor interaction, which has greatly contributed to the development of new vaccines as well as drug development in these disease conditions. The treatment of enterotoxigenic diarrhea is primarily aimed at fluid and electrolyte replacement rather than stopping the diarrhea, since patients die of dehydration and not of the intestinal infection as such. development of oral rehydration therapy, use of antisecretory agents, and the role of cholera vaccines are discussed briefly.

Habicht J-P see Esrey SA

Haider K see Ahmed ZU

Hale TL, Formal SB. Genetics of virulence in <u>Shigella</u>. Microbial Pathogen 1986 Dec;1(6):511-8

An essential step in the pathogenesis of bacillary dysentery is the infection of colonic epithelial cells. When ingested Shigellae invade the colonic mucosa intracellular bacterial growth results in ulceration, inflammation, and the clinical manifestation of dysentery. Discovery of large virulence-associated plasmids in Shigella has fostered renewed interest in the genetics of virulence in this genus. Current research in a number of laboratories is rapidly elucidating the organization and expression of plasmid virulence genes, but the role of various gene products in the process of bacterial invasion remains obscure. Even though at least 3 chromosomal regions have been shown to be necessary for virulence in S. flexneri, conjugal transfer of all 3 regions to an avirulent Escherichia coli recipient does not convert this closely related species into a virulent organism. Observations suggest that extrachromosomal loci are also involved in the virulence of Shigellae. All invasive strains of Shigella apparently express plasmid-coded polypeptides, a through g, and the complementation experiments suggest that at least polypeptides are exposed on

the outer membrane. In addition, polypeptides b and c have a predilection for mammalian cell surface. It is hoped that cloning of virulence genes and production of monoclonal antibody, recognizing the gene products, will aid in the study of pathogenic mechanisms. Using classical genetic techniques, it is now possible to construct invasive <u>E. coli K-12</u> hybrids which express either <u>Shigella sonnei</u>, <u>S. flexneri</u>, or <u>S. dysenteriae</u> type 1 somatic antigens. A <u>cocktail</u> of organisms expressing these serotypes should elicit serotype-specific antibody recognizing the etiological agents of most of the bacillary dysentery encountered in the world today. It remains to be seen if these vaccines successfully balance immunogenicity and nonreactivity and ensure lasting protection.

Hale TL see Formal SB

Hamilton JR. Nutritional therapy of chronic diarrhea. <u>In</u>: Lebenthal E, ed. Chronic diarrhea in children. New York: Raven Press. 1984:535-40

This paper examines some current theoretical considerations in the nutritional management of children with severe chronic diarrhea. The pathophysiological characteristics of chronic diarrhea are discussed. It is found that breast feeding and hygienically prepared foods can reduce the incidence and severity of the illness as seen in many parts of the world. In the active treatment of affected patients, specific foods can seldom be identified as important causative factors, but adequate, balanced nutritional intake is undoubtedly an important component of the treatment of chronic infantile diarrhea. Nutrients, both macro and micro, may favorably affect intestinal renewal and differentiation, thereby hastening intestinal repair. The enteric route should be used for nutrients if at all possible, and a variety of products and techniques are available to optimally utilize the existing absorptive function. Feedings should be adjusted according to the individual child's intestinal status and tolerance. Total parenteral nutrition carries certain risks, but provides a powerful tool for sustaining the severely affected babies.

Hamilton JR. Treatment of acute diarrhea. Pediatr Clin North Am 1985 Apr;32 (2):419-27

This paper outlines the basis and nature of the therapeutic advances. The discussions include: the susceptibility of infants and young children to diarrhea, causes of acute diarrhea, mechanisms of infectious diarrhea, and the prevention, active treatment, fluid-electrolyte therapy and nutritional therapy in diarrhea. It is stressed that the early use of iso-osmolar oral solutions could obviate the need for intravenous therapy. Early aggressive use of balanced nutritional intake is recommended. It seems rational to first try the inexpensive palatable feeding with which the child is familiar; this usually means milk or a milk-based formula. Then, for the less common, milk-intolerant patients, special formula prepared without milk protein or lactose can be tried.

Hamilton JR see Gall DG

Hansluwka H see Ruzicka LT

Hapel AJ see Doe WF

Hargrove MD, Jr. see Netchvolodoff CV

Harries J. Amoebiasis: a review. J R Soc Med 1982 Mar:75(3):190-7

Harries JT. Mechanisms and mediators of intestinal secretion in the small intestine. J Pediatr Gastroenterol Nutr 1982 Dec;1(4):575-82

Current knowledge of the mechanisms and mediators of secretion in the small intestine and their relevance to disease states are reviewed. This review is concerned mainly with secretion induced by secretagogues and/or ionophores acting at (a) mucosal or, (b) serosal loci of the epithelial cell, and (c) with secretion resulting from alterations in the kinetics of epithelial cell turnover. The agents and conditions that result in small intestinal secretion are discussed. Despite the advances that have been made in the last decade in the understanding of the mechanisms and mediators of intestinal secretion (and diarrhea) in the small intestine, there are many important and challenging questions that remain unresolved. Some of these questions will undoubtedly be answered during the next decade, and have further implications for the prevention and treatment of diarrheal states.

Harries JT see Booth IW

Harries JT see Candy DCA

Harrison S see Feachem RG

Hart DA see Eidels L

Harvey RF see Bradshaw MJ

Hautefeuille M see Ramband JC

Haverkos HW. Factors associated with the pathogenesis of AIDS. J Infect Dis 1987 Jul:156(1):251-7

Hawkey CJ, Rampton DS. Prostaglandins and the gastrointestinal mucosa: are they important in its function, disease, or treatment? Gastroenterology 1985 Nov:89(5):1162-88

In 1971, interest in the role of prostaglandins in the gastrointestinal tract was stimulated by the publication of two hypotheses - that aspirin damages the gastric mucosa by inhibiting prostaglandin synthesis and that cholera toxin Subsequent research stimulating it. causes diarrhea Ъy gastrointestinal actions of prostaglandins has been considerable and now impinges on clinical practice. This paper reviews the involvement prostaglandins and related compounds in mucosal protection, in ulcer healing, in diarrhea, and in gastrointestinal inflammation, with particular reference to the growing body of human data. Some prostaglandin analogues are effective ulcer-healing agents and may conceivably find sophisticated applications throughout the gastrointestinal tract. Prostaglandins synthesized endogenously are not always associated with the same actions as when applied exogenously, presumably because their synthesis is accompanied by synthesis of other eicosanoids with conflicting activity. Endogenous prostaglandin synthesis is probably protective for the gastric mucosa, but the extent and precise nature of this protection is unclear. Ulcer patients may have deficiencies of prostaglandin production, but confirmation is needed. The possibility that prostaglandins mediate diarrhea, caused by bacterial infection, some laxatives, Otarrhoeal Diseases 211

irradiation, and in the irritable bowel syndrome, is worth investigating further, but in many diarrheal illnesses, prostaglandin synthesis is probably an epiphenomenon. Gastrointestinal inflammation can occur in the face of inhibition of prostaglandin synthesis. There is little evidence prostaglandin synthesis is harmful in chronic inflammatory lesions of the gastrointestinal tract or that it causes accumulation of inflammatory cells. There is suggestive evidence that prostaglandins may offer some protection to the intestine in humans as in laboratory animals, but this has not been clearly established. The nature of this protection is completely Nonsteroidal anti-inflammatory drugs inhibit gastrointestinal prostaglandin synthesis, but have individual prostaglandin-independent actions. Ιf achievements of more than a decade of research into prostaglandins and the gastrointestinal tract look limited, this may be because in some areas the hopes entertained for them were unrealistic. The authors suggest to. investigate products of the lipoxygenase pathways in some areas. and particularly in the pathophysiology of qastrointestinal inflammation.

Hawkey CJ see Rampton DS

Hawkins P see Cutting WAM

Heim T. Requirements and utilization of macronutrients in enteral and parenteral nutrition in acute and chronic diarrhea. In: Lebenthal E, ed. Chronic diarrhea in children. New York: Raven Press, 1984:541-57

This chapter discusses the theoretical and practical aspects of nutritional support to patients with acute and chronic diarrhea with particular emphasis on protein, energy and macronutrient requirements. It is concluded that oral rehydration and maintenance therapy have beneficial effects in the routine clinical management of infants and children with acute diarrhea. Nutritional adequacy with these therapies can, however, be achieved only in well-nourished infants and children who have enough energy reserves to cover their metabolism during the oral maintenance therapy. If the diarrhea lasts more than 2 weeks, i.e. becomes protracted, nutritional management becomes a principal part of the therapy, and treatment is generally based on dietary manipulation. If oral feeding (with either customary of elemental diet) fails, supplemental parenteral nutrition offers a feasible means to deliver the needed increases in caloric and macronutrient supply.

Hellers G see Persson PG

Herniman R see Litvinov SK

Herrington D see Levine MM

Heubi JE see Balistreri WF

Heusser R see Steffen R

Hewlett EL see Pearson RD

Hinman AR, Bart KJ, Orenstein WA. New vaccines. Int J Epidemiol 1985 Dec;14 (4):502-4

Hinton M. The sub-specific differentiation of Escherichia coll with particular

reference to ecological studies in young animals including man. J Hyg (Lond) 1985 Dec;95(3):595-609

"It is possible to differentiate isolates of <u>Escherichia coli</u> using a number of techniques including the determination of the <u>serotype</u>, biotype and phage type and the profiles for resistance to antibacterial agents and toxic chemicals, intracellular enzymes, outer membrane proteins and plasmids and the production of enterotoxin and colicins. These methods have been used principally for the study of pathogenic <u>E</u>. <u>coli</u> and plasmid-mediated drug resistance. However they can also be used <u>successfully</u> for ecological purposes. The application of several of these techniques for the study of the ecology of <u>E</u>. <u>coli</u> in healthy young animals including man is described." (Author's abstract)

Hirschhorn N. The treatment of acute diarrhea in children: an historical and physiological perspective. Am J Clin Nutr 1980 Mar;33(3):637-63

historical, physiological, clinical, examines the and This review epidemiological evidence to support a method of therapy for children's diarrhea that may be recommended for general acceptance. The understanding and use of fluid and nutritional therapy of acute diarrhea in childhood have progressed over the years to a point where acute mortality can be reduced to nearly zero. At the same time, the ill-effects on electrolyte balance and nutrition may be reduced to a minimum. Through use of an oral glucose-electrolyte solution with a carefully designed composition, physiologically correct treatment may now be so simplified and inexpensive as to be readily available in remote, under-served areas of the world where most of the morbidity prevails, and be useful as well in more sophisticated settings. The treatment recommended in this paper, has several important departures from traditional teaching. It advocates rapid restoration of extracellular fluid with a polyelectrolyte solution, containing sodium, base, and potassium; use of an oral glucose-electrolyte solution for repletion of those not in shock and for maintenance; use of a single oral glucose-electrolyte solution for all groups, regardless of diagnosis; and quite early feeding with tolerated foods. Sodium loads given are generally higher than those advocated by standard pediatric teaching. The origins of that teaching and support for the newer approach accrue from a detailed analysis of current knowledge in the epidemiological clinical and physiological aspects of diarrhea. author's abstract)

Hitzig WH. Protean appearances of immunodeficiencies: syndromes and inborn errors involving other systems which express associated primary immunodeficiency. Birth Defects 1983;19(3):307-12

"Developmental defects, interrupting the normal evolution of immunocytes can explain many of the congenital immunodeficiency syndromes. Observations accumulated during the last decade have, however, shown that this is not the only cause, and that many diseases have signs of immunodeficiency as an accompanying feature. Severe combined immunodeficiency (SCID) is a good example of the multiple etiology of similar clinical features—they are phenocopies of a well—delineated hereditary disease. A number of recently described syndromes are reviewed, albeit an incomplete list. Metabolic disorders due to inactivity of enzymes may present characteristic ID. Some of them are explained by lack or increased need of co-enzymes (like biotin or zinc). In other syndromes, better understanding of the pathogenesis might pin down the primary failure to one single point, as shown in the hyper lgE

syndrome. Other fundamental disturbances are located in the chromosome itself, e.g. decreased repair capacity, deletion, translocation. An attempt is made to propose a general classification accommodating all etiologic factors known to date which lead to immunodeficiency. It is obvious that within this framework the same clinical syndrome may be repeated." (Author's abstract)

'Ho DD, Pomerantz RJ, Kaplan JC. Pathogenesis of infection with human immunodeficiency virus. N Engl J Med 1987 Jul 30:317(5):278-86

Ho TF see Yip WC

Hoch JA see Piggot PJ

Hochter W see Wormann B

Hoerr RA, Young VR. Alterations in nutrient intake and utilization caused by disease. Ann NY Acad Sci 1987:499:124-31

Hogan RC see Feachem RG

Hollis DG see Blake PA

Holmberg C. Congenital chloride diarrhoea. Clin Gastroenterol 1986 Jul;15 (3):583-602

Holmberg SD, Farmer JJ, 3d. Aeromonas hydrophila and Plesiomonas shigelloides as causes of intestinal infections. Rev Infect Dis 1984 Sep-Oct; 6(5):633-9

"Infections due to Aeromonas hydrophila and Plesiomonas shigelloides in immunocompromised hosts have been well documented, but the role of these organisms in causing gastrointestinal disease in healthy persons is controversial. Despite difficulties in characterizing the exotoxins produced by Aeromonas species, there is accumulating evidence that these bacteria are capable of causing usually mild, self-limited diarrheal disease in previously healthy adults. Some pediatric patients may have protracted dysenteric symptoms. P. shigelloides, an organism closely related to Aeromonas species, may also cause diarrhea in the healthy host, but no exotoxins have yet been identified by the assays used to identify other bacterial enterotoxins. Replacement of fluid and electrolytes is the basis for treatment, and antimicrobial agents should be reserved for patients with chronic or serious illness, or for those at particular risk of serious illness (hepatobiliary disease, septicemia, neoplasms)." (Authors' abstract)

Holmes PH. Pathogenesis of trichostrongylosis. Vet Parasitol 1985;18:89-101

Trichostrongylosis is a major cause of impaired productivity in ruminants. The pathogenesis of such infections is principally associated with inappetence and increased losses of proteins into the gastrointestinal tract. Resultant changes in host metabolism account for the poor productivity in infected animals, although the precise mechanisms require further investigation. In recent years, there has been a considerable increase in the understanding of the pathogenesis of such infections, especially in terms of alterations in host function or pathophysiology caused by gastrointestinal trichostrongylosis. Trichostrongylosis is associated with a range of clinical signs, including a failure to gain weight or weight loss, poor bodily condition, inappetence and

usually diarrhea. The failure to gain weight or weight loss is a major aspect of trichostrongylus infections, as is inappetence. Diarrhea is normally associated with trichostrongylus infections of veterinary importance, e.g. Ostertagia ostertagi, D. circumcincta; Trichostrongylus colubriformis, T. axei, Cooperia punctata, and Nematodirus battus, but is less common in infected with Haemonchus contortus, C. oncophora, and T. vitrinus. Studies are investigate in greater detail the interactions required to trichostrongylosis and the nutritional and immunological status of the host. It is concluded that there is now a considerably body of knowledge on the pathogenesis of trichostrongylosis. However, new dimensions to the complexities of such infections continue to be revealed. Several aspects require further investigation. These include a fuller appreciation of the mechanisms underlying alterations in protein and energy metabolism; more detailed examination of the factors causing reduced skeletal growth; interactions between gastrointestinal parasitism and the nutritional immunological status of the host; and the impact of larval challenge on animals under chemoprophylactic cover from slow release formulations of anthelmintics.

Holmes RK see O'Brien AD

Holmgren J. Pathogenesis and prevention of cholera. Scand J Infect Dis 1982; (suppl 36):58-64

Cholera is characterized by severe, watery diarrhea which leads rapidly to dehydration and metabolic acidosis. The causative agent, Vibrio cholerae, colonizes the small intestine and elaborates an exotoxin, cholera toxin, that binds to specific receptors on the mucosal cells and stimulates secretion of electrolytes and water from the cells. This paper summarizes current knowledge of these pathogenic events and discusses how, based on recent findings, it be possible to devise new techniques for cholera treatment and prophylaxis to complement those based on fluid replacement therapy and sanitation. Cholera research has now reached the stage where rational counteractive methods can be formulated based on a molecular knowledge of the pathogenesis and be tested in controlled clinical and field trials. However, none of the new preventive approaches described, represents a final solution ready for large-scale application. Each of the methods have been effective against cholera in animal studies, but only to a limited extent in humans. Yet, much more research is needed to define their place, if any, in future prophylaxis and treatment of cholera. For example, in relation to vaccine development, it remains to be seen whether the promising results, obtained with the combined oral B-subunit whole-cell vaccine with regard to mucosal antibody stimulation, will afford the long-lasting protection sought for. With regard to receptor blockade, it has been shown that pretreatment of the gut of rabbits with B-subunits of either cholera toxin or heat-labile toxin of Escherichia coli can completely protect animals from cholera after challenge with high doses of active cholera toxin or live V. cholerae. Basic studies to test the hypothesis that blocking of toxin receptors would prevent fluid loss also in cholera-infected persons are needed. In patients with mild-to-moderate dehydration, the success rate of oral rehydration therapy is very high, usually more than 90%. In severe cases, however, intravenous (i.v.) replacement of fluid is essential for survival. It is, therefore, important to find agents which could reduce fluid losses to a level where no one would need i.v. treatment. In the case of antisecretory agents, the aim should be to find drugs which effectively depress secretion, but lack the sedative action of chlorpromazine.

Holmyren J, Svennerholm A-M. Pathogenic mechanisms and new perspectives in the

treatment and prevention of enteric infections. Scand J Gastroenterol 1982;17(suppl 77):47-59

"Enteric infections cause more than a billion episodes of diarrhoeal disease in humans each year killing many millions of people, especially young children, in developing countries. Recent progress, reviewed in this article, has enabled that a specific pathogen now can be isolated in the majority of patients with acute diarrhoea, and has also elucidated fundamental pathogenic mechanisms and their pathophysiological effects for several of these agents. Based on this understanding it now seems possible to devise new techniques for the treatment and prevention of diarrhoeal disease to complement those based on fluid replacement therapy and sanitation; prospects for the development of new or improved vaccines, receptor-prophylactic binding agents, and antisecretory drugs are discussed." (Authors' abstract)

Holmgren J see Svennerholm A-M

Holscher MA see Nime FA

Holt SC. Anatomy and chemistry of spirochetes. Microbiol Rev 1978 Mar;42(1):

Holtan NR. Amebiasis: the ancient scourge is still with us. Postgrad Med 1988 Jun; 83(8):65-72

"Amebiasis is usually contracted in geographic areas where sanitation is poor, but outbreaks can still occur anywhere that drinking water becomes contaminated with sewage. In the majority of persons infected with the parasite, colonization of the intestine is asymptomatic. In others, symptoms of gastrointestinal distress can appear within a week. In rare cases, extra-intestinal amebiasis can cause abscesses in the liver or elsewhere. Many questions about the disease course in different patients remain to be answered. Diagnosis can be made through symptom identification; findings of right-upper-quadrant tenderness, leukocytosis, and an elevated level of alkaline phosphatase; and testing the feces for trophozoites or cysts. Clinicians disagree on whether asymptomatic persons need to be treated, but anyone who is capable of transmitting the disease should be advised of how to avoid exposing others to it." (Author's abstract)

Holzmuller W see Muller JM

Hood MA see Rodrick GE

Hargrove MD, Jr. see Netchvolodoff CV

Hormaeche CE see Dougan G

Hornick RB. Selective primary health care: strategies for control of disease in the developing world. XX. Typhoid fever. Rev Infect Dis 1985 Jul-Aug;7(4): 536-46

"The incidence of typhoid fever remains unacceptably high in developing countries. Because <u>Salmonella typhi</u> is disseminated by carriers, there is an urgent need to increase the rate of detection of carriers and to decrease the risk they pose to their communities. In urban areas where sewage disposal is

lacking or inadequate, public water supplies are contaminated and typhoid fever is common. The contamination of food by carriers is the second commonest route of infection. Water purification processes lead to a rapid decline in the incidence of the disease; thus, many developing countries hope to develop pure water supplies for all citizens by the end of this century. Until this important public health goal is achieved, the use of vaccine, especially in children, could cause a significant decrease in the incidence of typhoid fever. A new oral attenuated vaccine promises to be effective and safe." (Author's abstract)

Horzinek MC, Flewett TH, Saif LJ, Spaan WJ, Weiss M, Woode GN. A new family of vertebrate viruses: Toroviridae. Intervirology 1987;27(1):17-24

enveloped. is characterized by proposed family Torovirid<u>ae</u> peplomer-bearing particles containing an elongated tubular nucleocapsid with helical symmetry. The capsid may bend into an open torus, conferring a biconcave disk or kidney-shaped morphology to the virion (largest diameter 120-140 nm) or the capsid may be straight, resulting in a rod-shaped particle (35x170 nm). Morphogenesis is by budding of preformed nucleocapsids through membranes mainly of the Golgi system and of the rough endoplasmic reticulum. Berne virus, which is proposed as the family prototype, contains a single strand of infectious positive-sense RNA. Mr (molecular mass) about $6.5 \times 10(6)$. which is polyadenylated. The RNA is surrounded by the major nucleocapsid phosphoprotein (Mr about 20,000) which, in turn, is enveloped by a membrane containing one major protein (Mr 22,000) and a phosphoprotein (Mr 37,000). The viral peplomers, about 20 nm long, carry determinants for neutralization and hemagglutination; they are formed by a polydisperse N-glycosylated protein (Mr 75,000-100,000). Four major subgenomic polyadenylated RNAs have been identified in infected cells, with Mrs of 3.0, 0.71, 0.46 and 0.26x10(6). 75,000-100,000). Four major Torovirus replication is inhibited by actinomycin D, alpha-amanitin and pre-irradiation of the host cell with UV light. All toroviruses identified so far cause enteric infections and are probably transmitted by the fecal-oral route. Serologic relationships between equine, bovine and human toroviruses have been demonstrated." (Authors' abstract)

Hoshino Y <u>see</u> Kapikian AZ

Hosking DJ see Atkinson M

Hossain B see Khan MU

Hossain M see Molla AM

Howard P. Diarrhoeal diseases--priorities in research and intervention. Papua New Guinea Med J 1986 Jun;29(2):125-9

Hrdy DB. Epidemiology of rotaviral infection in adults. Rev Infect Dis 1987 May-Jun;9(3):461-9

Huffman SL see Chen LC

Hugh R see Ewing WH

Hughes JM <u>see</u> Esrey SA

Hughes S. Acute secretory diarrhoeas; current concepts in pathogenesis and

treatment. Drugs 1983;26:80-90

"Acute secretory diarrhoeas constitute a major source of mortality and morbidity world-wide. Our current understanding of the underlying mechanisms involved is reviewed with particular reference to cholera and enterotoxigenic E. coli infections. From the physiological principles involved, a unified concept for the treatment of acute secretory diarrhoeas is presented. The importance of rehydration is highlighted and practical instructions for the use of oral glucose-electrolyte solutions in the treatment of acute secretory diarrhoeas are given, along with some discussion of the rationale behind their use and optimum composition. The important role of nutritional factors during acute diarrhoea is underlined and the place of various drugs, some established, some experimental, are briefly discussed." (Author's abstract)

Hughes TP see Levine MM

Huminer D see Pitlik S

Hug E see Chen LC

Hug I, Aziz KMS. Changing antibiotic sensitivity pattern of the commonly occurring organism causing dysentery. Bangladesh Med J 1977 Apr:5(4):119-25

About 80-85% of cases of bacillary dysentery in Bangladesh are due to Shigella species. Prior to 1970, its treatment was simple since most of the strains were susceptible to commonly available drugs. such as chloramphenicol, etc. In later years (1971 onwards), treatment difficult because of the gradual acquiring of resistance of the organisms to the commonly administered antibiotics. During 1964-1970, it was observed that a lesser percentage of Shigella isolates were resistant to tetracycline and streptomycin. During 1971-1975, strains of Shigella dysenteriae type 1 that were isolated showed an increased resistance to tetracycline, streptomycin, and chloramphenical. Strains of \underline{S} . $\underline{flexneri}$ also showed an increased resistance to streptomycin and tetracycline. S. dysenteriae type 2, S. dysenteriae type 3-10. S. sonnei, and S. boydii all showed resistance to streptomycin. It is suggested that proper bacteriological diagnosis of the causative pathogen and its antiblotic sensitivity pattern should be checked before prescribing antibiotics. Further, since a majority of the clinical isolates happen to be strains of <u>S. dysenteriae</u> type 1 and <u>S. flexneri</u>, antibacterial drugs, streptomycin and tetracylcine, should not be used for treatment. Chloramphenicol should be avoided due to its side-effects. Only very few ampicillin-resistant strains being isolated, this could be the drug of choice for treatment of bacillary dysentery.

Hug I see Ahmad K

Huq MI, Ahmed QS, Rahaman MM. Changing pattern of antibiotic resistance in Shigella isolated in Bangladesh. <u>In:</u> Rahaman MM, Greenough WB, III, Novak NR, Rahman S, eds. Shigellosis: a continuing global problem; proceedings of an international conference, Cox's Bazaar, 1981. Dhaka: International Centre for Diarrhoeal Disease Research, Bangladesh, 1983:174-81

"Considering the increased evidence of multiple-resistant <u>Shigellae</u> in Bangladesh and the consequent problem of treatment, the changing antibiotic sensitivity pattern of Shigellae isolated in the International Centre for

Diarrhoeal Disease Research, Bangladesh (ICDDR.B) during the years 1976-1981 was monitored. Shigellae were isolated from patients reporting to the hospital and Treatment Centre of the ICDDR,B, Dhaka as well as the camps and Treatment centre of Teknaf Dysentery Project. Rectal swabs taken from patients before any therapy was started, were plated onto MacConkey and SS agar, and suspected colonies were identified biochemically followed by serotyping with Shigella antisera. The antibiotic sensitivity was determined by disc diffusion method using the standard Kirby Bauer technique. The 5 antibiotics tested were: tetracycline, ampicillin, chloramphenicol, streptomycin, and co-trimoxazole. With each set of plates a control Escherichia coli strain (ATCC 25922) was included. The Shigella serotype isolated from hospitalized patients was mostly Shigella flexneri, followed by S. dysenteriae type 1. Although there was no appreciable change in the percentage of resistance of <u>S. flexneri</u> to tetracycline, all the <u>S. dysenteriae</u> type 1 strains became resistant to tetracycline. Ampicillin resistance in <u>Shigella</u> did not show much change. All the different serotypes were commonly resistant to 2 drugs, usually the combination of tetracycline with ampicillin or streptomycin. Only 2 cases each of S. dysenteriae type 1 and S. boydii, showed resistance to all the five antibiotics. Since S. dysenteriae type I and S. flexneri account for 75-80% of the total Shigella isolated at the ICDDR.B. tetracycline should no longer be considered to be the drug of choice in treating shigellosis. The number of ampicillin-resistant strains of $\frac{Shigella}{considered}$ being very small, ampicillin was considered to be the drug of choice. However, it was suggested that antibiotics should be administered after performing proper sensitivity tests." (Authors' abstract)

Hyman PE see Cheromcha DP

Hyypia T, Pettersson U. Spot hybridization for the detection of adenoviruses and enteroviruses. Clin Lab Med 1985 Sep;5(3):491-501

Iaccarino E see Auricchio S

The International Drinking Water Supply and Sanitation Decade. Review of regional and global data (as at 31 December 1983). WHO Offset Publ 1986;(92):1-30

Intestinal protozoan and helminthic infections. WHO Tech Rep Ser 1981;(666): 1-150

Isaacs PET, Kim YS. Blind loop syndrome and small bowel bacterial contamination. Clin Gastroenterol 1983 May:12(2):395-414

Isaacson RE. Pili of enterotoxigenic <u>Escherichia coli</u> from pigs and calves. Adv Exp Med Biol 1985;185:83-99

Islam MR see Samadi AR

Ito T see Sakai S

Iwugo KO see Feachem RG

Jacewicz M see Keusch GT

Jacquenod P see Ramband JC

Jahan K see Ahmad K

Jakubowski W see Akin EW

Janda JM, Powers C, Bryant RG, Abbott SL. Current perspectives on the epidemiology and pathogenesis of clinically significant <u>Vibrio</u> spp. Clin Microbiol Rev 1988 Jul;1(3):245-67

Janda JM, Bryant RG. Pathogenic <u>Vibrio</u> spp: an organism group of increasing medical significance. Clin Microbiol Newslett 1987 Apr 1;9(7):49-56

Janoff EN, Reller LB. <u>Cryptosporidium</u> species, a protean protozoan. J Clin Microbiol 1987 Jun;25(6):967-75

Janssen PAJ, van Den Bossche H. Treatment of helminthiasis. Scand J Infect Dis 1982;(suppl 36):52-7

Janssen PAJ <u>see</u> Awouters F

Jelliffe DB see Jelliffe EFP

Jelliffe EFP, Jelliffe DB, Feldon K, Ngokwey N. Traditional practices concerning feeding during and after diarrhoea (with special reference to acute dehydrating diarrhoea in young children). World Rev Nutr Diet 1987;53:218-95

Jemos V see Dionigi R

Jenkins AL see Jenkins DJA

Jenkins DJA, Jenkins AL, Wolever TMS, Rao AV, Thompson LU. Fiber and starchy foods: gut function and implications in disease. Am J Gastroenterol 1986 0ct;81(10):920-30

"Increased intake of fiber and starchy foods has been recommended in the treatment or prevention of a range of diseases including dumping syndrome, hyperlipidemia, gallstones, diabetes, Crohn's disease, constipation, irritable bowel, diverticular disease, and colonic cancer. The nature and physiological effects of fiber are diverse. However in general, insoluble fibers increase fecal bulk and decrease transit time. On the other hand, soluble fibers have metabolic effects secondary to reducing the rate of small intestinal absorption. In the colon, along with undigested starch, they are largely fermented yielding short-chain fatty acids which may have further metabolic effects. At present although much further work is required, the clinical management of hyperlipidemia, diabetes, constipation, and diverticular disease have already been significantly influenced as a result of the ideas and experimental evidence generated by the fiber hypothesis." (Authors' abstract)

Jertborn M see Svennerholm A-M

Johnson JG see Ewing WH

Johnson PC, DuPont HL, Ericsson CD. Chemoprophylaxis and chemotherapy of travelers' diarrhea in children. Pediatr Infect Dis 1985 Nov-Dec;4(6):620-1

Johnson PC see DuPont HL

Johnson PC see Ericsson CD

Joiner KA, Gorbach St. Antimicrobial therapy of digestive diseases. Clin Gastroenterol 1979 Jan;8(1):3-35

Jones E see Kelts D

Jones GW see Freter R

Joseph SW, Colwell RR, Kaper JB. <u>Vibrio parahaemolyticus</u> and related halophilic vibrios. CRC Crit Rev Microbiol 1982;10(1):77-124

Judson FN. Sexually transmitted viral hepatitis and enteric pathogens. Urol Clin North Am 1984 Feb;11(1):177-85

Juma R see Kinoti SN

Juranek DD see Navin TR

Kagnoff MF see Elson CO

Kagnoff MF see Targan SR

Kahrs RF see Poppensiek GC

Kalina GP. [The genus <u>Pseudomonas</u>: new aspects of an old problem]. Zh Mikrobiol Epidemiol Immunobiol 1985 May; (5):91-8

Kaper JB, Lockman HA, Baldini MM, Levine MM. Development of live oral cholera vaccine candidates through recombinant DNA techniques.. <u>In</u>: Kuwahara S, Pierce NF, eds. Advances in research on cholera and related diarrheas. Tokyo: KTK Scientific Publishers, 1986:181-91

Kaper JB see Joseph SW

Kaper JB see Levine MM

Kapikian AZ, Flores J, Hoshino Y, Glass RI, Midthun K, Gorziglia M, Chanock RM. Rotavirus: the major etiologic agent of severe infantile diarrhea may be controllable by a "Jennerian" approach to vaccination. J Infect Dis 1986 May; 153(5):815-22

Rotaviruses are among the most important agents producing severe diarrhea in infants and young children aged under 2, because these viruses are associated with a disproportionate rate of life-threatening dehydrating episodes. Therefore, determined efforts are being made to develop, safety test, and evaluate rotavirus vaccines for infants and young children in this age group. The use of conventional cell-culture techniques for the growth of animal rotavirus strains holds great promise for the development of an effective rotavirus vaccine for humans. If this "Jennerian" approach, which follows Edward Jenner's concept of using a related virus from a different host as the immunizing antigen, does not induce adequate immunity to rotavirus-associated disease induced by each of the four serotypes, then reassortants with single-gene substitutions for each of the serotypes are available and "waiting

in the wings". Rotavirus strains, obtained from neonates with asymptomatic infections, may be potential vaccine candidates, since they might represent naturally occurring attenuated strains. However, none of these vaccines are likely to prevent illnesses due to the non-group A rotaviruses (also called pararotaviruses) that have recently caused large outbreaks of gastroenteritis in China. This virus type does not share the common group antigen and has been recovered only sporadically outside China. Their prevalence should be determined carefully. Vaccine development, if needed, must await growth of these viruses in tissue culture.

Kaplan JC see Ho DD

Karim A see Svennerholm A-M

Karmali MA. Infection by verocytotoxin-producing Escherichia coli. Clin Microbiol Rev 1989 Jan;2(1):15-38

"Verocytotoxin (VT)-producing Escherichia coli (VTEC) are a newly recognized group of enteric pathogens which are increasingly being recognized as common causes of diarrhea in some geographic settings. Outbreak studies indicate that most patients with VTEC infection develop mild uncomplicated However, a significant risk of two serious and potentially life-threatening complications, hemorrhagic colitis and the hemolytic uremic syndrome, makes VTEC infection a public health problem of serious concern. The main reservoirs of VTEC appear to be the intestinal tracts of animals, and foods of animal (especially bovine) origin are probably the principal sources for human infection. The term VT refers to a family of subunit exotoxins with high biological activity. Individual VTEC strains elaborate one or both of at least two serologically distinct, bacteriophage-mediated VTs (VT1 and VT2) which are closely related to Shiga toxin and are thus also referred to as Shiga-like toxins. The holotoxins bind to cells, via their B subunits, to a specific receptor which is probably the glycolipid, globotriosyl ceramide (Gb_s). Binding is followed by internalization of the A subunit, which, after it is proteolytically nicked and reduced to the Al fragment, inhibits protein synthesis in mammalian cells by inactivating 60S ribosomal subunits through selective structural modification of 28S ribosomal ribonucleic acid. mechanism of VTEC diarrhea is still controversial, and the relative roles of locally acting VT and "attaching and effacing adherence" of VTEC to the mucosa have yet to be resolved. There is increasing evidence that hemolytic uremic syndrome and possibly hemorrhagic colitis result from the systemic action of VT on vascular endothelial cells. The role of antitoxic immunity in preventing the systemic complications of VIEC infection is being explored. Antibiotics appear to be contraindicated in the treatment of VTEC infection. The most common VTEC serotype associated with human disease is 0157:H7, but over 50 different VT-positive O:H serotypes have now been identified. The best strategies for diagnosing human VTEC infection include testing for the presence of free VT in fecal filtrates and examining fecal cultures for VTEC by means of deoxyribonucleic acid probes that specify genes encoding VT1 and VT2. methods are currently confined to specialized laboratories and await commercial development for wider use. In the meantime, most laboratories should continue to screen for the most common human VTEC serotype, 0157:H7, using sorbitol-containing MacConkey medium." (Author's abstract)

Kaskel FJ see Feld LG

Kato T. [Molecular mechanisms of induced mutations]. Radioisotopes 1985

Jun:34(6):334-43

Kaufman D see Ma P

Kawata K. Water and other environmental interventions—the minimum investment concept. Am J Clin Nutr 1978 Nov;31(11):2114-23

This paper describes water and other environmental interventions — in the minimum investment concept. Effective environmental interventions to reduce enteric infections include provision for water of good quality and of sufficient quantity with ready availability, and sanitary disposal of excreta so that the transmission of pathogenic organisms through fluids (water and milk), fingers, flies, food, and fields (soil) does not occur. Studies have shown that in the southern US, a 50%-reduction in acute childhood diarrheal diseases was obtained when water was piped into homes. Epidemiological surveys have revealed that, e.g. in Peru, Chad, and Afghanistan, indiscriminate defecation in and around villages is very common. Several studies have shown that a good secondary sewage treatment plant and a good chlorination process can remove 99% of the coliform organisms. Isolation, inactivation, and dilution of pathogens are used in modern control systems. The role of flies in the transmission of Shigella is discussed. It is felt by scientists that the focus of treatment of diarrheal disease must shift from the host to environment if a permanent reduction in acute diarrheal diseases is to be achieved.

Kay RG see Tasman-Jones C

Kean BH. Travelers' diarrhea: an overview. Rev Infect Dis 1986 May-Jun;8 (suppl 2):S111-6

"The diarrhea of travelers is a syndrome and not a disease; its history is reviewed here. Dysentery is caused by agents that damage the epithelium of the intestinal tract; diarrhea results from the response of intact intestinal cells to toxins that stimulate enzymatic processes releasing liquid and ions. Toxigenic Escherichia coli, the most frequent pathogen in diarrhea, produces a recognizable, benign syndrome; however, other agents of disease, including viruses, bacteria, and protozoa, are responsible for a significant proportion of cases of diarrhea. Hygienic precautions are generally advised, despite the lack of evidence supporting their efficacy. Antibiotic prophylaxis effective in reducing the incidence of diarrhea caused by \underline{E} , \underline{coli} , but may be desirable because of toxicity and the possibility of $\underline{complicating}$ diagnostic process. Diagnosis requires better laboratory methods than are usually available but can be made with limited accuracy by a clinician acquainted with the various manifestations of potential causes. after diarrhea has started should be limited to fluid and ion replacement, with the possible addition of drugs that reduce intestinal motility, although some advocate the use of antibiotics at this time. The number of people traveling, especially to developing countries, continues to increase. This trend would expand enormously if the fear of diarrhea were removed." (Author's abstract)

Keat A. Reiter's syndrome and reactive arthritis in perspective. N Engl J Med 1983 Dec 29;309(26):1606-15

Keller HW see Muller JM

Kelly MT <u>see</u> Tison DL

Kelts D, Jones E. Selected topics in therapeutic nutrition. Curr Probl Pediatr 1983 Mar:13(5):1-62

Keusch GT, Solomons NW. Microorganisms, malabsorption, diarrhea and dysnutrition. J Environ Pathol Toxicol Oncol 1985 Jul;5(6):165-209

Keusch GT, Donowitz M. Pathophysiological mechanisms of diarrhoeal diseases: diverse aetiologies and common mechanisms. Scand J Gastroenterol 1983;18 (suppl 84):33-43

"A detailed review of the pathophysiology of diarrhoeal diseases is presented. Recent developments in the understanding of the mechanisms by which water absorption occurs via electrolyte transport are detailed. The secretion of Cl across the apical membrane in vivo is accompanied by a flow of Na as a counter ion, probably via the paracellular pathway. Normal regulation of active intestinal ion transport depends on cyclic AMP and cyclic GMP as well as intracellular calcium. In diarrhoeal diseases, some portion of the gut is usually found to be in a secretory rather than absorptive condition. This may be due to changes in active ion transport (decrease in Na absorption or increase in Cl secretion), alterations in intestinal motility and/or luminal osmolarity or to an increase in tissue hydrostatic pressure." (Authors' abstract)

Keusch GT, Scrimshaw NS. Selective primary health care: strategies for control of disease in the developing world. XXIII. Control of infection to reduce the prevalence of infantile and childhood malnutrition. Rev Infect Dis 1986 Mar-Apr;8(2):273-87

"Malnutrition is due to many complex and interacting factors, both biologic and social. This may be why so little has been accomplished in the global efforts to reduce its prevalence and impact. The greatest burden of malnutrition falls on the youngest members of society, and in these infants and children it is closely associated with infectious diseases. Because of multiple effects on host nutrition and metabolism, infections result in nutritional deterioration that must be corrected during convalescence. When this is precluded by limitations in the adequacy and availability of food, and infections are frequent, progressive deterioration in nutritional status occurs, with high Measures that reduce the prevalence and morbidity and mortality rates. nutritional consequences of infection are the most feasible and cost-effective interventions to improve nutritional status of young children at the present time. These measures include immunization, oral rehydration programs for diarrheal disease, promotion of breast feeding, continued feeding during infection, development of adequate weaning foods from mixtures of available local commodities, specific nutrient fortification, growth monitoring, improved environmental sanitation and water supplies, and education. The first seven measures can be introduced immediately in all societies and are basic elements of effective primary health care. The last two, which promise the greatest return in benefits, are the most costly and most difficult to implement. Malnutrition and infection are inseparable and the measures to deal with the former must effect a reduction in the later if they are to succeed." (Authors' abstract)

Keusch GT. Shigella infections. Clin Gastroenterol 1979 Sep;8(3):645-62

Keusch GT, Donohue-Rolfe A, Jacewicz M. Shigella toxin and the pathogenesis of

shigellosis. Ciba Found Symp 1985;112:193-214

Shigella dysenteriae 1 produces a periplasmic protein with multiple toxic effects in vivo and in vitro. These include neurotoxicity, cytotoxicity and enterotoxicity as well as the ability to inhibit cell-free protein synthesis. The purified toxin is a protein of relative molecular mass (Mr) 64,000. It is composed of one catalytically active A subunit (Mr=32,000) that inhibits protein synthesis, and a complex of five B monomers (Mr approximately 6,500 each). Studies using subunit-specific antibodies demonstrate that the B subunit mediates the binding of toxin to toxin receptors in the cell membrane. In a model system in HeLa cell culture, the surface membrane receptor has been shown to be a glycoprotein, most probably asparagine-linked, and to contain oligomeric beta 1-4 linked N-acetyl-D-glucosamine. Studies with metabolic inhibitors and agents that disrupt the cytoskeleton, and/or alter the pH and function of acidic cytoplasmic vesicles, provide indirect evidence that toxin is transported from the cell surface to the cell interior. This process is probably receptor-mediated endocytosis, since it is also inhibited by amines that prevent receptor-mediated uptake of other ligands in well-characterized systems. The toxic action in the HeLa cell is due to the subsequent inhibition of protein synthesis which results from catalytic inactivation of the 60S ribosomal subunit and the cessation of polypeptide chain elongation. Inhibition of protein synthesis by toxin produced subsequent to bacterial invasion of colonic epithelial cells could explain the destructive lesions found in shigellosis. Although toxin can induce jejunal secretion in animal models, there is at present no clear explanation for the secretory response of the gut mucosa in Shigella infection. (Authors' abstract)

Keusch GT, Donohue-Rolfe A, Jacewicz M. Shigella toxin(s); description and role in diarrhea and dysentery. Pharmacol Ther 1982;15(3):403-38

The interest in soluble toxins from Shigellae has risen and fallen more than once in the 80 years, since <u>Shigella dysenteriae</u> 1 was reported to produce a toxin ("Shiga toxin"). Debate has continued to center on whether or not the toxic activity might be involved in pathogenesis of any part of the disease syndrome produced by the microorganism itself. This review summarizes nearly 8 decades of work on Shigella toxins, but also concentrates on the phenomenal productivity of the last 10 years. The genus Shigella produces a protein toxin with neurotoxic, cytotoxic and enterotoxic properties. Toxin production is regulated in part by iron concentration in the medium and is produced under aerobic conditions. The toxin appears to be a periplasmic protein, but its role in the life of the organism is unknown. Under appropriate conditions, small quantities of toxin are made. The holotoxin has a molecular weight of approximately 72,000 daltons and contains a 30,000-dalton subunit. estimates of molecular weight and the presence of smaller 4-7000 11,000-dalton fragments or subunits have also been reported. The precise size, structure and composition of toxin are not yet certain. Toxin antigen is made in vivo during shigellosis in humans, and serum neutralizing antibodies may Shigellosis involves two distinct regions of readily be detected. intestine, the proximal small bowel and the colon, resulting in two distinct intestinal disease syndromes, watery diarrhea and dysentery. The authors believe toxin plays a crucial role in pathogenesis of both jejunal (watery) and colonic (dysenteric) phases of the illness. The mechanism by which it causes intestinal secretion or cell death is under active investigation.

Khan MR see Khan MU

Khan MU, Khan MR, Hossain B, Ahmed QS. Alum potash in water to prevent cholera [letter]. Lancet 1984 Nov 3;2(8410):1032

Since cholera is primarily transmitted by water, this ICODR, B study, carried out in Bangladesh, examines the efficacy of a traditional water purification practice used in the Indian subcontinent – namely, mixing a pinch of aluminium potassium sulphate (alum potash) into each pitcher of household water. Half the families of index cholera patients were randomized to an alum potash group to whom alum was supplied, teaching them how to use it (500 mg/L). Significantly fewer (p<0.05) family contacts using alum became infected (23/238) than the controls (47/265). Alum potash flocculates suspended materials, but the primary mechanism of alum's bactericidal activity appears to be acidification. In vitro experiments showed that alum treatment of pond water lowers its pH from 7.5 to 4.1 and kills all Vibrio cholerae 01 within 3 h. Killing of Shigella spp. and Escherichia coli takes longer. The study demonstrates that alum potash can significantly decrease secondary infection rates during cholera outbreaks. In Bangladesh, alum is cheap (1 US cent for 20 L water) and widely available. Decontamination of domestic water with alum during cholera epidemic is recommended.

Khan MU. Interruption of transmission of diarrhoeal agents. <u>In:</u> Programme, papers and abstracts of the Third Asian Conference on Diarrhoeal Diseases, Bangkok, 10-14 Jun 1985:173-80

Khan MU see Mosley WH

Khatoon M see Molla A

Khin-Maung U. Recent research on acute diarrhoea in Burma. DMR Bull 1987 $\operatorname{Jan}(4):1-19$

This paper summarizes the findings of recent research on acute diarrhea carried out by scientists from the Department of Medical Research, Department of Health, and Department of Medical Education working in hospitals, teaching institutions and various health services departments in Burma. The research findings summarized and reviewed here are presented under various subject headings, such as (1) etiology and epidemiology; (2) pathophysiology; (3) gut function in diarrhea; (4) nondehydration deaths from acute diarrhea; (5) case management of acute diarrhea; and (6) intervention. Major pathogens causing diarrhea were bacterial pathogens, such studied. These include enterotoxigenic Escherichia coli (producing heat-labile toxin, heat-stable toxin, or both), <u>Salmonella</u>, <u>Shigella</u>, <u>Vibrio cholerae</u>, <u>Vibrio parahaemolyticus</u>, enteropathogenic <u>E. coli, Campylobacter jejuni</u>, and <u>Yersinia</u> enterocolitica, and viral agents, such as rotavirus, astrovirus, calicivirus, corona-like viruses, small-round viruses, etc. The role of various formulations of oral rehydration solutions, dietary management, antisecretory agents, and antibiotics are considered. Intervention through early institution of home oral rehydration therapy, handwashing, and health education is discussed and reviewed.

Kim YS see Freeman HJ

Kim YS see Isaacs PET

Kimmey M. Infectious diarrhea. Emerg Med Clin North Am 1985 Feb;3(1):127-42

King EQ. Human infections with <u>Vibrio</u> <u>fetus</u> and a closely related <u>Vibrio</u>. C Infect Dis 1957 Sep-Oct;101:119-28

Vibriosis in cattle was recognized first in 1909, and in 1918, Smith isolated and studied the causative organism, naming it Vibrio fetus. For years, natural infections with the organism were thought to occur only in domestic animals, but several instances of human infection have been reported as well. This study describes the cultural and biochemical properties and serological relationship of 10 animal and 7 human strains of V. fetus, 3 animal strains of V. bubulus, and 4 human strains of a "related Vibrio". Symptomatology of the 15 known human infections with V. fetus and the 4 infections with the "related Vibrio" is discussed. The epidemiology of the disease in animals is discussed in view of the possibility of a similar means of transmission in humans. The possible epidemiology of the disease in humans is also discussed in the light of what is known concerning the disease in animals.

Kinoti SN, Wasunna A, Turkish J, Gateere R, Desai M, Agwanda R, Juma R. A comparison of the efficacy of maize-based ORS and standard W.H.O. ORS in the treatment of acute childhood diarrhea at Kenyatta National Hospital, Nairobi, Kenya: results of a pilot study. East Afr Med J 1986 Mar;63(3):168-74

Kivilaakso E. Antacids and bile salts. Scand J Gastroenterol 1982;17(suppl 75):16-9

"Antacids, especially aluminium hydroxide, adsorb bile salts and lysolecithin with an affinity and capacity comparable to that of cholestyramine, a property which is likely to contribute to the beneficial effect of antacids in peptic ulceration. Dihydroxy bile salts are bound more strongly than trihydroxy bile salts and glycine conjugates more strongly than taurine conjugates, but ambient pH does not seem to have any effect on the binding. The bile salt-binding property of aluminium hydroxide may be of therapeutic value in the management of bile salt-induced diarrhoea, but its usefulness in the management of bile-reflux gastritis still remains questionable." (Author's abstract)

Kleinman RE see Lake AM

Klemm P. Fimbrial adhesions of <u>Escherichia coli</u>. Rev Infect Dis 1985 May-Jun;7(3):321-40

Kliegman RM, Fanaroff AA. Necrotizing enterocolitis. N Engl J Med 1984 Apr 26:310(17):1093-1103

Necrotizing enterocolitis is the most common acquired gastrointestinal emergency in the neonatal intensive care unit. This condition is suspected when gastrointestinal signs and symptoms predominate. Neonatal necrotizing enterocolitis appears to be a single pathologic response whereby the immature intestine reacts to injury. The mucosal pathology may be initiated by multiple factors or microbiologic agents acting either alone or in concert. The incidence increases markedly at lower gestational ages. The onset of necrotizing enterocolitis is usually between the 3rd and 10th day of life. The typical patient has abdominal distension, ileus with delayed gastric emptying, diarrhea, and abdominal tenderness. The diagnosis is confirmed only by demonstration through an abdominal roentgenogram of abnormal intestinal

bacterial gas formation as pneumatosis intestinalis or intrahepatic venous gas or both. To permit more consistent evaluation of patients, an important system of staging has been proposed. Milk feeding is a nearly universal observation among patients. Intestinal mucosal injury may be induced by excessive osmolarity of formula. The absence of immunoprotective factors may contribute to the development of necrotizing enterocolitis. However, the disease may also develop in infants who have never been fed enterally, and may be present without abnormal intestinal gas formation. Intestinal and, more specifically, mucosal ischemia has been considered as a risk factor. Epidemiologic observations support a direct role for microbial organisms or their toxin in the development of necrotizing enterocolitis. Alternatively, necrotizing enterocolitis may be a host response to multiple adverse intestinal conditions. The disease may be classified according to clinical presentation. Each of the subgroups has a characteristic clinical course and may have Because there is evidence suggesting that necrotizing initiating factors. enterocolitis is infectious in nature, both preventive and therapeutic regimens are directed toward the control of microbiologic agents. Mortality is directly related to the presence of bacteremia, disseminated intravascular coagulation, ascites, and very low-birth weight. The disease has an overall mortality of 20-40%

Kluge RM. Infectious diarrhea: an update. Compr Ther 1983 Nov;9(11):26-30

Knight R. Hepatic amebiasis. Semin Liver Dis 1984 Nov:4(4):277-92

Kobari K. Recent trends of cholera. Jpn J Trop Med Hyg 1985 Mar: 13(1):53

Recently it has been noted that cases of cholera due to vibrios of classical biotype have reappeared in Bangladesh. Since September 1982, these cases have remarkably increased. It has been observed that these strains are able to survive in mixed culture with cholera <u>Vibrio</u> of the El Tor type, unlike the original classical type. The progress of research on cholera toxin has been noteworthy, particularly the success of Finkelstein in purifying cholera toxin. Successful development of animal models reproducing cholera-like symptoms has also been achieved. (Modified author's abstract)

Koblinsky MA see Feachem RG

Kodner IJ, Fry RD. Inflammatory bowel disease. Clin Symp 1982;34(1): 3-32

Konno T. [Viral gastroenteritis: serological characteristics and molecular epidemiology of human rotavirus]. Rinsho Byori 1985 Feb;33(2):129-35

Kopecko DJ, Baron LS, Buysse J. Genetic determinants of virulence in <u>Shigella</u> and dysenteric strains of <u>Escherichia</u> <u>coli</u>: their involvement in the pathogenesis of dysentery. Curr Top Microbiol Immunol 1985;118:71-95

Korman SH, Berant M, Alon U. Review: prostaglandins in diarrheal states. Isr J Med Sci 1981 Dec:17(12):1109-13

Kosloske AM. Pathogenesis and prevention of necrotizing enterocolitis: a hypothesis based on personal observation and a review of the literature. Pediatrics 1984 Dec;74(6):1086-92

It is thought that necrotizing enterocolitis of the neonate occurs by the coincidence of two of three pathologic events: (1) intestinal ischemia, (2)

colonisation by pathogenic bacteria, and (3) excess protein substrate in the intestinal lumen. The disease is more likely to appear following quantitative extremes, i.e. severe ischemia, highly pathogenic flora, or marked excess of substrate. Necrotizing enterocolitis develops only if a threshold of injury, sufficient to initiate intestinal necrosis, is exceeded. The hypothesis is derived from theories by Santulli, which implicated all three events, and by lawrence in which a single event, abnormal bacterial colonisation, was considered sufficient to induce necrotizing enterocolitis. This hypothesis may explain both typical occurrences of necrotizing enterocolitis among high-risk premature infants in neonatal intensive care units, and atypical occurrences among infants considered at low-risk. e.g. previously healthy term infants. infants fed breast milk exclusively, and infants never fed. It may further explain why necrotizing enterocolitis fails to develop in most high-risk infants in neonatal intensive care units. Preventive measures include: pharmacologic stabilization of intestinal perfusion, (2) modification of the intestinal flora, or (3) feeding colostrum or other protective substances. Each theoretical benefit is accompanied by potential risks. (Modified author's abstract)

Krag E. Irritable bowel syndrome: current concepts and future trends. Scand J Gastroenterol 1985;20(suppl 109):107-15

"About five percent of the adult population each year see their doctor with complaints that are finally characterised as irritable bowel syndrome (IBS). The complaints are constipation (perhaps alternating with diarrhoea), abdominal abdominal rumbling pain (dull or colicky), abdominal distension, flatulence. The diagnosis of IBS implies that a relevant examination has precluded any organic disease. The etiology is unknown and the syndrome probably does not represent a disease entity. It is therefore difficult, if not impossible, to produce a definite rationale of treatment. However, several aspects of the pathogenesis of the individual symptoms of IBS are well known: (1) chronic constipation is most likely due to fiber-depleted anal fissures. psychological factors. local organic disorders (e.g. hemorrhoids, diverticulosis) and disturbance of the body fluid balance (e.g. high consumption of diuretic compounds such as coffee and tea); (2) pain is related to spasms and motility disturbances causing increased intraluminal pressure; (3) meteorism is not due to an increased amount of intestinal gas, but "air traps" and segmental accumulation of gas seem to occur. Furthermore. psychopathological factors and perhaps also food intolerance may play an At present the rationale of treatment in IBS is: (1) etiological role. management of constipation, (2) ease of spasms, (3) reduction of surface tension of intestinal contents. (4) ease of mental stress." (Author's abstract)

Krejs GJ. VIPoma syndrome. Am J Med 1987 May 29;82(suppl 58):37-48

"Since the description of the watery diarrhea syndrome by Verner and Morrison 29 years ago, clinical and experimental observations have elucidated the pathophysiology of this disease. Vasoactive intestinal polypeptide (VIP) is produced and released by a tumor of the pancreatic islets or by a tumor of neural crest origin such as a ganglioneuroma. Under normal conditions, current evidence suggests that VIP is a neurotransmitter in the central and peripheral nervous systems and particularly in the peptidergic nervous system. The low-VIP plasma concentration observed in healthy subjects is viewed as a neuronal overflow since it has been impossible to ascertain any endocrine role

for circulating VIP. Markedly elevated VIP plasma levels in the VIPoma syndrome lead to intestinal secretion with severe secretory diarrhea, resulting in hypovolemia, hypokalemia, and acidosis. These symptoms subside after successful tumor removal. Approximately, 50 percent of patients have metastatic spread at the time of diagnosis. For these patients, a new and promising therapeutic modality is available in the form of a subcutaneously administered somatostatin analogue that relieves symptoms through potent inhibition of VIP release from tumor tissue." (Author's abstract)

Kristidis P see Soutter VL

«Kronmall R see Cvjetanovic B

Kubba R. Gastrointestinal manifestations of skin diseases: a review. Trop Gastroenterol 1983 Apr-Jun;4(2):67-78

Kuo C. Measures to control diarrhoeal diseases -- environmental sanitation. Regional Meeting on Cholera and Diarrhoeal Diseases, Alexandria, 1-5 Jun 1978. Alexandria: Regional Office for the Eastern Mediterranean, World Health Organization, 1978. 6 p.

The various implications and issues in the implementation of the different measures to control diarrheal diseases are described. Environmental sanitation is crucial in the control of diarrheal diseases, including cholera. The main emphasis in this approach is to ensure safe water at the source. water sources must not be used by people, and replacement of those with improved piped supplies is needed. Sewerage systems with treatment facilities provide for safe evacuation and disposal of human wastes. Food sanitation is needed to prevent the disease. Education of the people for a better understanding of the mode of transmission of these diseases and for improvement of their personal hygiene is emphasized. Flies have been identified as an important insect vector in the spread of diarrhea. Proper disposal of solid wastes is the permanent solution to fly control. Chemical control has been found to provide quick results. The United Nations Water Conference, held in Argentina in March 1977, recommended the adoption of targets for safe water supply and sanitation for all by 1990 and the designation of 1981-1990 as the International Drinking Water Supply and Sanitation Decade. authorities have been urged to respond to the call of the United Nations and to speed up their environmental sanitation programs.

Kupchinsky LG see Zhalko-Titarenko VP

Kuperman O see Freier S

Kurstak C see Kurstak E

Kurstak E, Kurstak C, van Den Hurk J, Morisset R. Animal rótaviruses. In: Comparative diagnosis of viral diseases, v. 4. New York: Academic Press, 1981:105-48

Kusmiesz H see Nelson JD

La Brooy J, Rowley D. Cholera vaccine - recent progress. In: Easmon CSF, Jeljasewicz, eds. Medical microbiology, v. 2. London: Academic Press, 1983:157-76

La Brooy J see Rowley D

Lake AM, Kleinman RE, Walker WA. Enteric alimentation in specialized gastrointestinal problems: an alternative to total parenteral nutrition. Adv Pediatr 1981;28:319-39

Lake AM see Cohen S

Lamb D see Feachem RG

Lambert HP. Antimicrobial agents in diarrhoeal disease. Clin Gastroenterol 1979 Sep;8(3):827-33

This review assesses the role of antimicrobial agents in acute diarrheal diseases in adults and children and provides a rational basis for their use. In gastroenteritis due to <u>Salmonella</u>, the antimicrobial agents have no effect on the duration of illness and prolong the average duration of positive-stool cultures. The use of antimicrobials in Salmonella gastroenteritis has the disadvantage of favoring the selection of drug-resistant flora. Antimicrobials are not indicated for mild-transient illnesses, but are considered essential in severe bacillary dysentery. A number of agents which inhibit growth of Vibrio cholerae in vitro was shown to be effective in clinical trials, these include tetracycline and doxycycline among others. Infection due to Yersinia enterocolitica is discussed with reference to the use of drugs. Some studies have demonstrated the usefulness of chloramphenical in treating $\underline{\text{Yersinia}}$ septicemia, although others have reported that human isolates of $\underline{\underline{\text{Y}}}$. enterocolitica were susceptible to cotrimoxazole. In infection due to Escherichia coli, it is concluded that antimicrobials should not be prescribed for routine use. Antimicrobials are often used to control the spread of \underline{E} . coli gastroenteritis. Antimicrobial drugs and their application in diarrheal diseases of other etiologies are also highlighted. Since research reports on this subject are still few and because the clinical patterns of diarrheal diseases vary so much from country to country, a decline in antibiotic use, as desired by physicians and researchers in unspecified diarrheal diseases, has not come about.

Lamont JT see Trnka YM

Lang W. Progress in the pathogenesis and therapy of viral and bacterial diseases. Scand J Infect Dis 1982; (suppl 36):7-11

Laroche Y see Cornelis G

Lebenthal E, Rossi TM. Intractable diarrhea of infancy: an alternative treatment strategy. Postgrad Med 1983 Aug;74(2):153-9

Prolonged diarrhea in an adult is a serious problem and in an infant it can be life-threatening. This paper reviews current concepts in pathophysiology in relation to conventional treatment and management aspects in diarrheal illness. Intractable diarrhea of infancy occurs most often in young infants aged under 3 months, but can also occur in older infants. Conventional therapy for intractable diarrhea of infancy, which involves use of clear liquids and gradual reintroduction of cow's milk or formula, is adequate in most situations. However, in some infants this type of management exacerbates diarrhea. Regardless of cause, intractable diarrhea of infancy is usually

accompanied by small intestinal mucosal injury. The recommended treatment alternative is aimed at correcting fluid, electrolyte, and nutritional deficiencies. Elemental diets are helpful, because minimal mucosal absorption is required. If used early in the course of the syndrome, they may prevent multiple formula changes and the gradual deterioration in nutritional status that occurs with reintroduction of a more complex formula. When a trial of an elemental diet fails, total parenteral nutrition is required. The alternative therapy would lessen the likelihood of diarrhea exacerbation and decrease morbidity and mortality.

Lebenthal E see Rossi TM

Lee EC see Walker RI

Lee EW. Safe water supply and sanitation in diarrhoeal diseases control. Regional Planning Meeting on Diarrhoeal Diseases Control, Manila, 5-7 Jun 1979. Manila: Regional Office of the Western Pacific, World Health Organization, 1979. 5 p. (WPR/BVD/DDC/79.3)

The provision of safe drinking water and modern sanitation systems play a preventive role and can have a long-term impact on the health of people in any community. The latest World Health Organization (WHO) survey shows that 1,230 million people are without adequate water supply and another 1,350 million without proper sanitation in developing countries (except China). Only 38% of the Third World population had access to safe drinking water, 80% of all diseases in the world being associated with water use. A large population of the world's people suffers from waterborne diseases: 400 million from gastroenteritis, 160 million from malaria, 30 million from river-blindness, and 200 million from schistosomiasis. The global problems of water-related diseases led to the creation of an International Drinking Water Supply and Sanitation Decade (1981-1990), the goals of which are the provision of safe drinking water and adequate sanitation for all by 1990. Successful program implementation will require a national commitment, a reorientation of policies, mobilization of resources, and use of appropriate technology and appropriate administrative developments.

Lee SP see Tasman-Jones C

Leggiadro RJ see Yolken RH

Lennard-Jones JE. Medical treatment of ulcerative colitis. Postgrad Med J 1984 Nov;60(709):797-802

Leung AKC. Chronic nonspecific diarrhea of childhood (irritable colon syndrome). Contemp Pediatr 1987 Mar-Apr:10-3

Leung AKC, Darling P, Auclair C. Oral rehydration therapy: a review. J R Soc Health 1987;107(2):64-7

Rehydration and maintenance of adequate fluid and electrolyte balance is the key to the management of the child with acute diarrheal disease. Introduction of oral carbohydrate-electrolyte solutions for the treatment of dehydration due to acute diarrhea has been one of the major therapeutic advances of this century. Oral rehydration treatment has been shown to be simple, practical, inexpensive, highly effective and safe for developing as well as for developed

countries. The efficacy and safety of oral rehydration therapy (ORT) is well established. A decline in the diarrheal mortality rate has been a consistent finding when it has been accessible to the community and properly used. A better understanding of the physiological mechanisms implicated in diarrheal illness as well as extensive clinical testing of oral rehydration solutions have led to the improvement of the composition of electrolyte, carbohydrate and base constituents. The widespread use of ORT may result in a decreased need for hospitalization and less discomfort and complications which are associated with intravenous rehydration therapy. Although minor refinements in its composition may still be possible, its role in the treatment of diarrheal dehydration is unquestionable.

Leung TSM see Candy DCA

Levine MM. Antimicrobial therapy for infectious diarrhea. Rev Infect Dis 1986 May-Jun:8(suppl 2):S207-16

"Acute diarrheal disease may be due to viral, bacterial, or enteropathogens. In our current state of knowledge and medical specific antiviral agents are not used in the treatment of known or presumed In contrast, for a number of the bacterial and protozoal viral diarrhea. with certain antimicrobial diarrheal infections, therapy significantly ameliorate the severity and duration of illness and curtail the excretion of the pathogen. A recurring theme encountered in reviewing information on the therapy for diarrheal infections is that demonstration of the susceptibility in vitro of a bacterial pathogen to a particular antibiotic by no means assures clinical success. Many antibiotics that show potent activity in vitro have little or no efficacy in vivo. Controlled clinical trials are necessary to assess the clinical and bacteriologic efficacy of an antibiotic in diarrheal infections." (Author's abstract)

Levine MM. Bacillary dysentery: mechanisms and treatment. Med Clin North Am 1982 May:66(3):623-38

The term, bacillary dysentery, connotes shigellosis in any of its clinical presentations. This article discusses the clinical patterns, mechanisms and treatment modes of bacillary dysentery. The spectrum of Shigella infections includes asymptomatic intestinal infection, watery diarrhea with or without fever, and severe dysentery manifested by high fever, toxemia, chills, convulsions, abdominal cramps, tenesmus, and frequent bloody mucoid stools. To cause disease, Shigella organisms must (1) possess smooth lipopolysaccharide O antigen, (2) have gene encoding for the ability to invade epithelial cells and to proliferate therein, and (3) elaborate a toxin following cell invasion. These virulence properties have been described, and the role of bacteria-host interaction in pathogenesis has been discussed. The therapy of acute bacillary dysentery has been divided into 4 components: (1) emergency treatment of life-endangering complications; (2) supportive measures using rehydration regimens or antimotility agents; (3) specific antibacterial drugs, such as ampicillin, cotrimoxazole, or nalidixic acid; and (4) health education.

Levine MM, Edelman R. Enteropathogenic <u>Escherichia coli</u> of classic serotypes associated with infant diarrhea: epidemiology and pathogenesis. Epidemiol Rev 1984;6:31-51

Currently, there are 4 recognized classes of Escherichia coli that cause diarrheal disease in humans; enteropathogenic \underline{E} . \underline{coli} (EPEC), enterotoxigenic

E. coli, enteroinvasive E. coli, and the recently recognized enterohemorrhagic \overline{E} . \overline{coll} . Each of these classes of \underline{E} . \underline{coli} manifests distinct features in pathogenesis, clinical syndrome, and epidemiology, and each falls within a different set of 0 antigen serogroups. A series of 0:H serotypes of \underline{E} . \underline{coli} were incriminated by epidemiologic studies during 1945-1960 as a cause of epidemic diarrhea in infant nurseries as well as a major cause of sporadic infant diarrhea in the community. The term EPEC was coined to refer to these infant diarrhea-associated serotypes. In the early 1970s, with the advent of laboratory tests to assess heat-labile and heat-stable enterotoxin production and enteroinvasiveness of \underline{E} , \underline{coli} , the classic EPEC strains were found to lack those particular properties. These observations led some to question their pathogenicity. Since 1977, there has been an explosion of new information on classic serotype EPEC, including volunteer studies with carefully characterized strains. They yielded identification of novel virulence properties and partial elucidation of pathogenesis, recognition of a pathognomonic histopathologic lesion in the affected intestine, and new epidemiologic insight. information is summarized in this review and put into context with earlier information on EPEC. Since 1978, EPEC have come to be appreciated anew as a separate class of diarrheagenic \underline{E} . \underline{coli} that cause diarrhea by distinct The pathogenesis of these strains, which have been pathogenic mechanisms. diarrhea in volunteers, appears to involve both shown to cause enteroadhesiveness step and production of a toxin identical to Shigella toxin. A 55- to 65-megadalton plasmid is involved in the attachment of EPEC to intestinal mucosa which results in a pathognomonic histopathologic lesion visualized by electron microscopy. The lesion involves dissolution of enterocyte microvilli by the bacteria, effacement of the enterocyte outer membrane, and formation of a pedestal around the bacterium at point of contact with the outer membrane of the enterocyte. Case-control epidemiologic studies, carried out since 1975, document that EPEC remain an important cause of sporadic infant diarrhea in the community with up to 30% of cases of diarrhea in young infants in Brazil and South Africa being attributed to these pathogens. Although nursery epidemics of EPEC-associated diarrhea virtually disappeared from industrialized countries, sporadic EPEC-associated diarrhea in infants in the community continues to occur. The relative importance of EPEC as a cause of sporadic diarrhea in both industrialized and developing countries needs to be reassessed. The clearcut demonstration of the pathogenicity of EPEC strains and recognition of their virulence properties warrants further studies of the epidemiology of EPEC infection. To facilitate such studies, a diagnostic tool other than serotyping is desirable to screen for such strains. Another major consequence of elucidation of the pathogenesis of EPEC has been to focus research on vaccine development. New areas of investigation include: (1) a search for the bacterial products responsible for enteroadhesiveness of EPEC to allow purification as potential oral immunogens, and (2) attempts to prepare attenuated strains for use as live oral vaccines.

Levine MM. <u>Escherichia coli</u> that cause diarrhea: enterotoxigenic, enteropathogenic, enteroinvasive, enterohemorrhagic, and enteroadherent. J Infect Dis 1987 Mar;155(3):377-89

"There are four major categories of diarrheagenic <u>Escherichia</u> <u>coli</u>: enterotoxigenic (a major cause of travelers' diarrhea and <u>infant diarrhea</u> in less-developed countries), enteroinvasive (a cause of dysentery), enteropathogenic (an important cause of infant diarrhea), and enterohemorrhagic (a cause of hemorrhagic colitis and hemolytic uremic syndrome). Besides manifesting distinct clinical patterns, these categories of E. coli differ in

their epidemiology and pathogenesis and in their 0:H serotypes. Common features (albeit distinct for each category) include plasmid-encoded virulence properties, characteristic interactions with intestinal mucosa, and elaboration of various types of enterotoxins or cytotoxins. A less well-defined fifth category of diarrheagenic \underline{E} , \underline{coli} is that of enteroadherent \underline{E} , \underline{coli} , so far identifiable only by their pattern of adherence to $\underline{Hep-2}$ cells in tissue culture." (Author's abstract)

Levine MM, Kaper JB, Black RE, Clements ML. New knowledge on pathogenesis of bacterial enteric infections as applied to vaccine development. Microbiol Rev 1983 Dec;47(4):510-50

Enteric bacterial infections, causing diarrhea, dysentery, and enteric fevers, are important health problems throughout the world. Among the most important enteric bacterial pathogens recognized are the following: (i) enterotoxigenic Escherichia coli (ETEC), (ii) Vibrio cholerae 01, (iii) Campylobacter jejuni, (iv) Shigella spp., (v) Salmonella typhi, and (vi) nontyphoidal Salmonella spp. For many years, attempts have been made to prepare immunizing agents against some of these infections, with varying results. More recently, there have occurred great advances in the knowledge of the pathogenesis of infections due to these enteropathogens. The new information has been applied toward vaccine development, resulting in fresh evaluations of older vaccines as well as This review attempts to bring together new innovative new approaches. knowledge on the pathogenesis of bacterial enteric infections and relates this information to vaccine development. Immune responses to various antigens are reviewed: such information is crucial for vaccine development to determine what antigens are particularly desirable for inclusion as immunizing agents. Current approaches to immunoprophylaxis of ETEC infection involve vaccines that stimulate antitoxic or antiadhesion immunity or both by means of killed antigens or attenuated strains. Preliminary results in animal models suggest that the outer-membrane proteins of pathogenic $\underline{\sf Shigella}$ strain may indeed be important immunogens. Several varieties of killed whole-cell parenteral S. typhi vaccine have been subjected to rigorous field trials to evaluate safety and efficacy in humans. The major thrust now is on identifying immunizing agents at least equal in efficacy to parenteral acetone-killed vaccine (70-90%) but which cause no adverse reactions.

Levine MM, Clements ML, Black RE, Hughes TP, Tome FC. Oral rehydration with simple sugar/salt solutions as an alternative in rural areas when glucose/electrolyte solutions are unavailable. In: Holme T, Holmgren J, Merson MH, Mollby R, eds. Acute enteric infections in children: new prospects for treatment and prevention. Amsterdam: Elsevier, 1981:325-31

Carefully prepared simple sugar/salt solutions accompanied by adequate potassium supplementation, may be used as an alternative to the preferred glucose/electrolyte oral rehydration solution (ORS) formula when the latter is unavailable. Many persons, particularly in developing countries' rural areas, do not have ready access to health facilities. Mothers can use ingredients readily available at the village level (crude salt and sugar) mixed with reasonable accuracy to produce a safe, effective ORS. Improper mixing could result in hyperosmolar solutions, which could induce in infants hypernatremia accompanied by convulsions, intracerebral hemorrhage and high case fatality. This chapter provides data on (i) the variability of 3 methods of preparing a simple sugar/salt ORS; (ii) developing a practical and reliable method; and (iii) the simple solution's efficacy in oral rehydration. There must be at

least one person in a village who has been formally taught how to properly prepare the simple ORS, and who must be responsible for choosing correctly sized utensils and for teaching/supervising ORS preparation by mothers.

Levine MM, Losonsky G, Herrington D, Kaper JB, Tacket C, Rennels MB, Morris ${\it J}$ G. Pediatric diarrhea: the challenge of prevention. Pediatr Infect Dis 1986 Jan;5(suppl 1):S29-43

Approximately two-thirds of the world's population live in less-developed areas, characterized by a lack of potable water, inadequate means for disposal of human fecal waste, intense crowding in rudimentary housing, lack of refrigeration and primitive standards of personal hygiene. Under conditions, the various pathogens that cause diarrheal disease/dysentery and enteric fever are readily transmitted to young children, resulting in an enormous burden of disease. In recent years, there has occurred an impressive increase in knowledge of the agents that cause diarrheal diseases, their epidemiology and pathogenesis; as a consequence, it has led in recent years to multiple vaccine candidates against the most important causative agents. vaccines are completing large-scale field trials of efficacy, while others are Governments of most developing countries are strongly entering this stage. committed to improving primary health care, with a special emphasis on immunization of infants. It is, therefore, anticipated that the most promising new vaccines against enteric infections will be incorporated into the Expanded Programme on Immunizations. (Modified authors' abstract)

Levine MM, Black RE, Brinton CC, Jr., Clements ML, Fusco P, Hughes TP, O'Donnell S, Robins-Browne R, Wood S, Young CR. Reactogenicity, immunogenicity, and efficacy studies of Escherichia coli type 1 somatic piliparenteral vaccine in man. Scand J Infect Dis 1982; (suppl 33):83-95

Levine MM. Travellers' diarrhoea: prospects for successful immunoprophylaxis. Scand J Gastroenterol 1983;18(suppl 84):121-34

"Enterotoxigenic Escherichia coli (ETEC) are the major aetiological agent of travellers' diarrhoea, usually accounting for 30-60% of cases. Thus, a safe and effective vaccine against ETEC could play an important role in prevention of this infection. A successful vaccine must somehow protect against a heterogenous array of ETEC pathogens comprising many 0:H serotypes and three enterotoxin phenotypes (LT $^+$ /ST $^-$, LT $^-$ /ST $^+$, and LT $^+$ /ST $^+$). Three major approaches to vaccine development are currently in progress including: $(\bar{1})$ LT and ST toxoids that stimulate antitoxic immunity; (2) purified pili vaccines that induce anti-adhesion immunity; (3) attenuated strains (prepared by recombinant DNA techniques) that mimic natural infection by stimulating antibacterial and anti-adhesive immunity as well as antitoxic immunity. Since Shigella represents the second most common cause of travellers' diarrhoea. immunoprophylaxis against this pathogen is also desirable. A recently described attenuated Salmonella typhi strain (Ty21a) expressing Shigella sonnei O antigen on its surface represents an attractive Shigella vaccine prototype. (Author's abstract)

Levine MM see Booth IW

Levine MM <u>see</u> Kaper JB

Levine MM see Rennels MB

Levison DA see Blackshaw AJ

Linde K. Stable, highly immunogenic mutants of <u>"Salmonella"</u> with two independent, attenuating markers as potential live vaccine and their validity for "Shigella" and other bacteria. Dev Biol Stand 1983;53:15-28

"This paper presents for Salmonella sp. an easily realizable principle to develop high-immunogenic. stable live vaccines with two independent attenuating mutations, which is valid for <u>Shigella</u> sp. and Past<u>eurella</u> sp. too. To avoid overattenuation by the stepwise introduction of two attenuating markers, only those mutations are suitable which do not suppress pathogenic structures or essential metabolic functions, but only transduce them into a leaky function, as e.g.: mutagen induced auxotrophic phenotypes with diminished virulence by commutation; purine dependent strains, which hitherto erroneously were regarded as non immunogenic and particularly distinct chromosomal resistant genotypes, in which the conformational change of the drugtarget simultaneously causes resistance and changes in virulence behaviour (pathwaydrift-mutants). Moreover, in Salmonella, an additional high sensitivity marker against tensides and drugs possessing a permeation barrier in the outer diminishes--without influence on virulence and immunogenicity--surviving in the intestine (bile) and (detergent contaminated) environment. Therefore this hst-marker confers antiepidemic quality to vaccine strains and increases safety. To meet security demands for such vaccines with laboratory methods some easy and practicable tests for standardization are suggested, which include estimation of reversion frequency of pur- and hst-marker and the proof of immunogenicity for mice. The safety and efficacy of such vaccine strains are pointed out by veterinarians in live-stock." (Author's abstract)

Linggood MA see Porter P

Lishnevshil MS see Litvinov SK

Litvinov SK, Merson MH, Oblapenko GP, Herniman R, Lishnevshil MS. [The WHO program for controlling diarrheic diseases: its status and development outlook - the organizational and operative components of the program]. Zh Mikrobiol Epidemiol Immunobiol 1985 Jun;(6):93-8

Ljungh A, Wadstrom T. Aeromonas and Plesiomonas as possible causes of diarrhoea. Infection 1985 Jul-Aug;13(4):169-73

"The number of reports on the isolation of Aeromonas from patients with diarrhoeal disease is now large and suggests an etiological role of the bacterium. It is well established that strains of Aeromonas produce an enterotoxin. This enterotoxin is cytotonic, i.e. it does not damage the membrane, and it does not cross-react immunologically with cholera toxin and Escherichia coli heat-labile (LT) toxin. Most enterotoxigenic strains also produce a cytotoxic protein (hemolysin), the role of which is probably limited in diarrhoea but potentially toxic in humans in other kinds of infections. Strains of Plesiomonas shigelloides also seem to be able to cause diarrhoea in some cases. The pathogenesis of Plesiomonas-induced diarrhea remains to be elucidated, but a heat-stable enterotoxin may be involved." (Authors' abstract)

Ljungh A, Wadstrom T. Aeromonas toxins. Pharmacol Ther 1982;15(3):339-54

This paper discusses toxins of the 2 closely related species, Aeromonas

hydrophila and A. sobria. A. hydrophila produces 2 hemolysins: (i) α -hemolysin, and (ii) cytotoxic protein or β -hemolysin. The yield of α -hemolysin in various complex media is higher at 22°C and is repressed at 37°C. Crude hemolysin is stable between pH 3.5 and 9.5 at room temperature. Hemolysin is destroyed by proteolytic enzymes and is inactivated by DTT and zinc ions. The isoelectric points are 5.5 and 4.3. Purified α -hemolysin has phospholipase C activity. In the logarithmic phase, the growth of β -hemolysin is stimulated in the presence of ribonucleic acid in a complex medium. The molecular weight is estimated to be 49,000-53,000. The isoelectric points are 4.8-5.2 and 3.5 (+01). Partially purified β -hemolysin is stable at room temperature between pH 4 and 9. The hemolytic activity of β -hemolysin decreases significantly when incubated with gangliosides. α - and β -hemolysins have several characteristics in common. Aeromonas enterotoxin induces fluid accumulation in the rabbit, rat and mouse intestinal loops. Their isoelectric point is 4.0-5.7 and molecular weight 15,000-20,000. The toxin is stable between pH 4.5 and 10. It is destroyed by papain. Enterotoxin remains stable when heated at 56°C. These findings support the hypothesis that enterotoxin production in Aeromonas is under chromosomal control as in Vibrio cholerae. The pathogenesis of intestinal A. hydrophila infection may be associated with still other virulence factors.

Ljungh A, Eneroth P, Wadstrom T. Cytotonic enterotoxin from <u>Aeromonas</u> <u>hydrophila</u>. Toxicon 1982;20(4):787-94

"Aeromonas hydrophila produces two hemolysins and an enterotoxin during growth. Enterotoxin, separated from the hemolysins, gave positive reactions in the rabbit intestinal loop test, the rabbit skin test, and the adrenal Y_1 cell test. Neutralization experiments in the rabbit loop, rabbit skin and Y_1 cell tests failed to demonstrate any immunological relationship between Aeromonas enterotoxin and cholera toxin or Escherichia coli heat-labile enterotoxin. Prior incubation of Aeromonas enterotoxin with gangliosides did not inhibit the positive test results in these systems. A coagglutination test with antiserum to purified cholera toxin was negative for Aeromonas enterotoxin, which, therefore, seems to be immunologically distinct from cholera toxin. The Aeromonas enterotoxin induced steroid secretion in adrenal Y_1 cells and increased the intracellular cyclic adenosine 3',5'-monophosphate (cAMP) content of Y_1 cells as well as of rabbit intestinal epithelial cells. It thus seems to act via the adenylate cyclase-cAMP pathway and should be classified as a cytotonic enterotoxin." (Authors' abstract)

Lo CW, Walker WA. Chronic protracted diarrhea of infancy: a nutritional disease. Pediatrics 1983 Dec;72(6):786-800

Chronic protracted diarrhea of infancy, a nutritional disease, is well known. It can lead to a cycle of malabsorption, malnutrition, and failure to thrive. This paper is a review on the recent scientific findings on the etiology, pathogenesis, diagnosis and laboratory investigation of chronic protracted diarrhea in infancy. Prevention and management are also discussed. A number of causes of chronic diarrhea in infancy have been identified and are discussed, including post-infectious enteritis, celiac disease, cow's milk allergy, and parasitic infection. Celiac disease is related to wheat in the diet. Cow's milk allergy may be due to early exposure to cow's milk protein at a time when there is excessive intestinal uptake of antigenic macromolecules. Giardia and Entamoeba histolytica can also cause chronic dysentery or diarrhea. In this work, the case-fatality rate was 27%. Metronidazole is suggested for

Shigella, Salmonella, Escherichia coli, Yersinia dysentery and amebiasis. enterocolitica, and Campylobacter fetus are the most common causative agents of infectious diarrhea in children. Viral gastroenteritis causes structural abnormalities similar to those found with celiac disease: villus atrophy, crypt The resulting diarrhea may involve hypertrophy, and inflammatory infiltrate. several mechanisms, including osmotic and secretory diarrhea, bacterial overgrowth, and disordered motility. Diagnosis of chronic diarrheal disease should include a careful dietary history and attention to signs of malnutrition and tests for specific malabsorption. Management is directed at providing adequate nutrition through oral elemental diets or total parenteral nutrition. Therapeutic efforts should concentrate on nutritional rehabilitation through appropriate oral elemental formulas or total parenteral nutrition. of the original causes can most effectively be implemented by encouraging breast feeding.

Lockman HA see Kaper JB

Loeb H, Mozin MJ. Prevention of chronic diarrhea: nutritional implications. J Pediatr Gastroenterol Nutr 1983;2(suppl 1):S328-34

Lohr JA see Guerrant RL

Lohr JA see Williams EK

Loizeau E see Berrut C

Lolekha S. Consequences of treatment of gastrointestinal infections. Scand J Infect Dis 1986;(suppl 49):154-9

"Diarrhoeal disease is a common problem in developing countries. As a result of recent advances in diagnostic methodology, the causative agents can now be identified in most cases of acute diarrhoeal diseases. Enteric bacterial pathogens are the common cause of gastroenteritis in developing countries. Appropriate uses of antibiotics in selected cases of diarrhoea will decrease symptoms or reduce faecal shedding of the organism and prevent spread of infection. Antimicrobial agents improve the diarrhoea associated with cholera, shigellosis, enteric fever, enterotoxigenic Escherichia coli, giardiasis, amoebiasis, and probably Vibrio parahaemolyticus, and enteropathogenic E. coli. Antibiotics have no role in the treatment of viral diarrhoea or uncomplicated Salmonella gastroenteritis. Most of the diarrhoeal diseases are self-limited, and the wrong choice of antimicrobial agents will worsen the symptoms. Treatment of gastrointestinal infections with antimicrobials will change intestinal microflora, promote the emergence of resistant strains and overgrowth of potential pathogenic bacteria and fungi. Risks and benefits should be considered before prescribing antimicrobial agents." (Author's abstract)

Losonsky G <u>see</u> Levine MM

Love AHG. Metabolic response to malnutrition: its relevance to enteral feeding. Gut 1986 Nov;27(suppl 1):9-13

"Malnutrition results in a wide variety of metabolic responses, depending on circumstances, from reactions to pure deprivation of nutrients to include the added stress of injury and sepsis. Important differences of responses exist

between adults and children. Weight loss with changes in carbohydrate, fat, and protein metabolism are well documented. Disturbances of fluid and electrolyte balance are newer areas of interest as are changes in requirements for micronutrients, such as trace metals. Many of these metabolic changes are under hormonal control. The intestinal tract shares in the response to malnutrition, and the consequent changes in mucosal function determine the ability of the intestine to handle enteral feeds. Such a route for nutritional support is important in protecting intestinal function not only in absorption but also in hormone production. Enteral feeding is increasingly having an important role in the interactions between acute diarrhoeal disease and malnutrition." (Author's abstract)

Lowman BC see Orossman DA

Ma P, Kaufman D. <u>Isospora belli</u> diarrheal infection in homosexual men. AIDS Res 1984;1(5):327-38

"Unsporulated <u>Isospora belli</u> oocysts were detected in the stool specimens of three homosexual men. The oocysts were ellipsoidal measuring 23-33 x 12-15 µm. It is acid fast with modified cold kinyoun stain (MCK) and reveals orange fluorescence with the Truant's stain. Sheather's sucrose flotation method is effective in concentrating <u>Isospora</u> oocyst as with other coccidial oocysts. One specimen showed many Charcot-Leyden crystals. All three patients responded to treatment. Isosporiosis is rare in this country and is endemic in the tropics and subtropics. Although one case acquired the infection after returning from an endemic area, the other two cases had no travel history. This raised the suspicion that this, like other parasitic infections (amebiasis, giardiasis, cryptosporidiosis) may be sexually transmitted. exotic parasitic infections in homosexual men during the outbreak of the acquired immunodeficiency syndrome adds another unusual infectious agent to the differential diagnosis of diarrheal disease in this high risk group of population." (Authors' abstract)

Mabilangan LM, Tiangco-Torres N. Focus on Salmonella enteritis. Asean J Clin Sci 1983 Dec;4(4):392-4

This paper discusses the clinical features, transmission, occurrence, pathogenesis, diagnosis and therapy of Salmonella enteritis. The 4 main clinical presentations are: enteric typhoid and paratyphoid fever, septicemia with or without localized infection, inapparent infection and carrier state, and acute gastroenteritis or food poisoning. The infection is transmitted from man to man by the fecal-oral route via contaminated food, water and fomites. Although Salmonella enteritis may attack any age group, the highest incidence has been observed in children aged under 9 with a peak rate among the 6-month to 4-year-olds. Stools collected early in the course of enteric disease prior to antibiotic coverage would be ideal for diagnostic examinations. The treatment of Salmonella gastroenteritis consists mainly of the correction of electrolyte and water disturbances, while antimicrobial therapy may be necessary in chronic salmonellosis. Since therapeutic control is not quite effective, Salmonella enteritis should be prevented through measures that involve public health education and the institution of a hygienic environment.

McCallum RW see Minami H

McClain CJ. Trace metal abnormalities in adults during hyperalimentation.

JPEN 1981 Sep-Oct:5(5):424-9

Trace metal deficiencies are now a well-documented complication of total parenteral nutrition. Zinc deficiency may present in a variety of ways, including acrodermatitis, impaired immunity, poor growth or impaired wound healing, and mental disturbances. Copper deficiency presents a more uniform picture of hematologic abnormalities, usually anemia with leukopenia and neutropenia. Chromium and selenium deficiencies occur much less frequently, but well-documented cases have been reported. Regular monitoring and supplementation of these 4 trace metals during the administration of total parenteral nutrition are currently recommended. This article describes the clinical abnormalities that may develop when deficiencies of trace metals occur during the administration of total parenteral nutrition, and recommendations for trace metal supplementation during the administration of total parenteral nutrition are put forward. (Modified author's abstract)

McClain CJ. Zinc metabolism in malabsorption syndromes. J Am Coll Nutr 1985; 4(1):49-64

Research during the past two decades has produced a wealth of information generated great interest in the vital role of zinc for normal metabolic function. This paper deals with zinc metabolism in malabsorptive states due to intestinal or pancreatic diseases. Initial information concerning zinc metabolism in man came from the early studies of investigators, such as Prasad in underdeveloped countries. Further knowledge and Sandstead working concerning the metabolic role of zinc was derived from manifestations of zinc deficiency in either zinc-deficient animals or in patients with acrodermatitis enteropathica. This paper discusses the clinical importance of zinc deficiency in intestinal and pancreatic malabsorptive states, possible mechanisms causing zinc deficiency in these diseases, and examples from some researches concerning how these aforementioned manifestations of zinc deficiency may be found in malabsorptive states. Zinc deficiency can complicate both intestinal pancreatic malabsorptive states and may manifest itself in a variety of ways. ranging from typical skin lesions to more subtle findings, such as immune dysfunction or impaired night vision. It is important that health-care personnel be aware of the multiple presenting features of zinc deficiency. So that it can be recognized and treated in an early state.

McCracken GH, Jr. see Eichenwald HF

MacDonald KL, Cohen ML. Epidemiology of travelers' diarrhea: current perspectives. Rev Infect Dis 1986 May-Jun;8(suppl 2):S117-21

Diarrhea is the most common illness affecting international travelers. During 1983, an estimated 286 million people traveled abroad worldwide, and a substantial number probably experienced travelers' diarrhea. Investigators have examined many strategies to prevent or treat this problem, including the administration of prophylactic or therapeutic antimicrobial agents. Identification of the characteristics that make certain travelers more likely to develop diarrhea can lead to prevention of the illness to improved understanding of endemic diarrheal disease in developing countries. Travelers' diarrhea, a syndrome with a spectrum of clinical symptoms, is most frequently characterized by watery diarrhea, cramps, and nausea. The highest attack rates have been reported in travelers from the United States or northern Europe to less-developed, particularly tropical, countries. Among travelers from

less-developed countries, diarrhea has been correlated with higher socioeconomic status. The findings that country of origin and socioeconomic status may affect the frequency of previous exposures to enteric pathogens suggest that persons with prolonged exposure acquire immunity and are at lower risk of developing travelers' diarrhea. Although few studies have shown a clear correlation between the eating of specific foods and the development of travelers' diarrhea, the syndrome has been associated with eating in public places. (Modified authors' abstract)

McGarry M see Feachem R

McHenry MC, Weinstein AJ. Antimicrobial drugs and infections in ambulatory patients. Some problems and perspectives. Med Clin North Am 1983 Jan;67(1):3-16

McIntosh MA see Finkelstein RA

MacIntyre PB. The short bowel. 8r J Surg 1985 Sep; (suppl 72): S92-3

McKee A <u>see</u> Pearson DJ

McRae JR see Metz SA

MacLean WC, Jr. see Brown KH

Mahalanabis D, Patra FC. In search of a super oral rehydration solution: can optimum use of organic solute-mediated sodium absorption lead to the development of an absorption promoting drug? J Diarrhoeal Dis Res 1983 Jun;1 (2):76-81

A hypothesis is proposed that may lead to the development of a super oral rehydration solution (ORS). Available literature is critically reviewed. Suggested are possible ways of further enhancing the absorption of sodium and water from the small intestine in acute diarrhea, through water soluble organic solute-linked transport, without imposing an osmotic penalty due to unabsorbed organic compounds. These ways are: (1) use of suitable polymers, such as polysaccharides and proteins; (ii) use of mixtures of rapidly absorbed organic molecules, such as d-hexoses and amino acids; and (iii) use of complex and variable mixtures of polysaccharides, proteins, protein hydrolysates, oligo-and monosaccharides, di- and tri-peptides, and amino acids. Based on these principles, optimum concentrations of organic compounds in ORS and use of compounds other than glucose (including sucrose, glucose polymer, and rice powder) are discussed. Through clinical trials, it already has been shown that addition of the amino acid glycine to standard glucose-containing ORS enhance absorption of sodium and water, without imposing an osmotic penalty. To explore the possibility of further enhancing the absorption of ORS, clinical trials were conducted in infants and children, who had moderate-to-severe dehydration due to diarrhea caused by rotavirus Vibrio chelense caused by rotavirus. diarrhea Vibrio cholerae. enterotoxigenic Escherichia coli and others. The control group received standard WHO-recommended ORS with glucose, while one study group received the same solution with added glycine, and the other received ORS in which 20 g of glucose was replaced by 50 g of popped rice powder for each liter of fluid. Compared to the control groups, in the groups treated with rice ORS and glycine-glucose ORS, respectively, there was a significant reduction of 3 variables: diarrheal stool output, by 49% and 50%; mean duration of diarrhea,

by 30% in each group; and volume of ORS needed to treat them, by 36% and 43% respectively. In addition to replacing diarrheal losses, these 2 study solutions led to reduced magnitude and duration of diarrhea. Thus, the 2 solutions assumed the role of highly effective absorption-promoting drugs. The results were superior to the effects of antisecretory drugs so far tested in humans, the future prospect of the hypothesis is discussed. A series of animal and human studies in vivo is in progress to define the optimum composition of a "Super ORS".

Mahalanabis D, Choudnuri AB, Bagchi NG, Bhattacharya AK, Simpson TW. Oral fluid therapy of cholera among Bangladesh refugees. Johns Hopkins Med J 1973 Apr;132(4):197-205

In this classic paper, the effectiveness of oral rehydration solution (ORS) in treating cholera without hypovolemic shock was confirmed by a crucial field trial during a massive cholera outbreak in the summer of 1971 among Bangladesh war refugees at Bongaon, West Bengal, India. Extremely adverse logistic and administrative conditions prevailed. Some 3,703 patients, including severe cases treated initially with limited intravenous (i.v.) supplies mild-to-moderately severe cases treated with ORS alone, were admitted to the Bongaon treatment center. The overall case-fatality ratio was 3.6%, which compared favorably with the results of standard i.v. therapy in well-organized modern treatment centers. Moreover, a special demonstration unit treated 1,190 of these patients, with a case-fatality ratio of only 1%. The ORS contained sodium 90 mmol/1, bicarbonate 30 mmol/1 and chloride 60 mmol/1, with 22 g/1 of glucose, prepackaged for mixing with water in the field. supplementation was given orally on an individual basis. Sample surveys during the epidemic showed that 38% of the patients were under age 6. About 79% of the 108 rectal swabs yielded Vibrio cholerae, with 92% of the isolates being the classical biotype. Advantages of ORS included local availability of ingredients, reduced cost, ease of administration, safety in the hands of inexperienced personnel after only brief instructions, early accessibility of treatment, and reasonable effectiveness when given early in the disease course. In severe cases, considerable sparing of i.v. fluids resulted from the adjunct use of ORS.

Mahalanabis D, Merson MH, Barua D. Oral rehydration therapy - recent advances. World Health Forum 1981;2(2):245-9

One of the most important advances in diarrheal disease research has been the discovery that dehydration associated with acute diarrhea, irrespective of etiology and age, can be treated orally with a simple glucose-electrolyte solution. This paper discusses the scientific basis of oral rehydration therapy, its nutritional benefits, and experiences gained in treatment centers of different countries. The authors stress the need to develop national diarrheal disease control programs to ensure continued availability of oral rehydration solutions (ORS) and the training and education of health workers and families so that diarrhea-related mortality and malnutrition can be prevented.

Mahalanabis D see Rohde JE

Mahmoud AA. A physician's guide to the diagnosis of common parasitic infections. Med Clin North Am 1983 Jan;67(1):253-8

Malavolti M see Fromm H

Mani V. Idiopathic ulcerative colitis - clinical problems, controversies and what's new. Trop Gastroenterol 1986 Oct-Dec;7(4):147-56

Manning PA. Involvement of cell envelope components in the pathogenesis of Vibrio cholerae: targets for cholera vaccine development. Vaccine 1987 Jun;5(2):83-7

Manning PA see Guidolin A

Mansour TE. Serotonin receptors in parasitic worms. Adv Parasitol 1984;23: 1-36

Manuca M see Alexandrescu M

Mara D see Feachem R

Mara DD see Feachem RG

Marks J. The relationship of gastrointestinal disease and the skin. Clin Gastroenterol 1983 Sep;12(3):693-712

Marks MI see San Joaquin_VH

Marshal WC see Candy DCA

Marshall BJ. Campylobacter pyloridis and gastritis. J Infect Dis 1986 Apr; 153(4):650-7

The successful isolation of Campylobacter pyloridis from human gastric mucosa in 1982 marked the turning point in the long but unremarkable career of this organism. At the present time, infection with C. pyloridis is found in many patients undergoing gastroscopic examination and in asymptomatic adults. presence confers an approximately 10-fold risk of developing ulceration. A study in 1983 revealed that the bacteria were present in 70% of 40 patients with gastric ulcers and in 90% of 70 patients with duodenal ulcers. The bacteria were still present in patients whose ulcers had healed with cimetidine therapy but who still had gastritis. It is the most probable cause of active chronic gastritis, a disorder associated with nearly all forms of chronic dyspepsia. It is associated with a forgotten form of acute bacterial gastroenteritis causing hypochlorhydria. Its ability to digest urea and produce ammonia in the stomach is a metabolic disorder of particular relevance to patients with renal or hepatic disease. Observation of the healing and relapse rates of duodenal ulcers following the eradication of C. pyloridis, and studies of antibiotic therapy for patients with chronic vague gastrointestinal syndromes will be helpful in estimating the importance of this new pathogen.

Marshall T see Feachem RG

Martinez RJ see Portnoy DA

Martinez-Baez M see Martinez-Palomo A

Martinez-Palomo A. The pathogenesis of amoebiasis. Parasitol Today 1987 Apr; 3(4):111-8

Martinez-Palomo A, Martinez-Baez M. Selective primary health care: strategies

for control of disease in the developing world. X. Amebiasis. Rev Infect Dis 1983 Nov-Dec;5(6):1093-1102

"The human parasite, Entamoeba histolytica, has a worldwide distribution. The majority of infected individuals suffer few symptoms; nonetheless, a variable proportion experience dysentery, hepatic abscesses, or invasion of other extraintestinal organs. Invasive amebiasis is a major health problem, particularly in certain developing countries. The aim of this review is to clarify, as much as possible, controversial issues about the biology of the parasite, the clinical features of amebiasis, and the estimations of morbidity and mortality rate as the basis for defining strategies for control of amebiasis by primary health care workers. The eradication of invasive amebiasis lies basically in potentially political and economic actions at the governmental level. Preventive and control measures include (1) provision of safe water supplies and sanitation, (2) health education, (3) adequate diagnosis and correct treatment of invasive amebiasis, and (4) implementation of surveillance and control programs." (Authors' abstract)

Martorell R see Chandra RK

Maskell DJ see Dougan G

Mata L, Urrutía JJ, Simhon A. Infectious agents in acute and chronic diarrhea of childhood. In: Lebenthal E, ed. Chronic diarrhea in children. New York: Raven Press. 1984:237-52

This work describes the agents of acute and chronic diarrhea of childhood. Enterotoxigenic Escherichia coli (ETEC) and rotaviruses stand out as the main agents associated with acute diarrhea. In the National Childrens' Hospital in Costa Rica, chronic diarrhea is the main cause of death, being also associated with severe protein-energy malnutrition (PEM). The agents found in chronic diarrhea are generally the same as those diagnosed in acute diarrhea, but their relative frequency varies. Shigella and parasites, especially Giardia and Entamoeba histolytica, are more prominent in poor urban and rural children. E. coli, Campylobacter, Yersinia, among others, are also often responsible for chronic diarrhea. Coronaviruses proved to be associated with chronic diarrhea in India. Persistence of Shigella infection in small children is discussed. A retrospective analysis of agents in acute and chronic diarrhea was carried out. Results showed the presence of pathogens in the following manner: 41% of the patients had single infections, 14% had double, and 3.7% had triple infections. The multiple pathogenic organisms were found in the stools of children with severe PEM and diarrhea. Observations indicate that chronic diarrhea does not occur in infants who are breast-fed from birth. Administration of allergenic food with early weaning, failure of the host to cope with infection, failure to diagnose the exact causal agent, all contribute towards establishing chronic diarrhea. Multinational collaborative studies on the infecticus etiology of acute and chronic diarrhea are recommended.

Mata LJ see Chandra RK

Mathan VI. Small intestine failure. In: Taylor TG, Jenkins NK, eds. Proceedings of the XIII International Congress of Nutrition. London: Libbey, 1986:671-4

A variety of conditions associated with maldigestion and malabsorption of nutrients can produce different degrees of small intestinal failure leading to

malnutrition. Intestinal failure often follows a small intestinal resection. Resection of 1/3 to 1/2 of the small bowel is consistent with the adequate maintenance of oral nutrition. When less than 25% of the small bowel remains, the patient needs special nutritional management. Small intestinal resection for conditions, such as Crohn's disease or scleroderma, usually results in severe malabsorption as the disease may affect the remaining bowel. Following small intestinal resection, the incidence of diarrhea decreases by preserving the ileocecal valve and thus protecting against bacterial overgrowth in the remaining small intestine. Dehydration, electrolyte losses, and acid-base disturbances need to be immediately corrected in the post-operative period when watery diarrhea with stool volume as high as 10 liters. At this stage, oral feeding aggravates diarrhea and restriction of oral feeding reduces diarrhea significantly. Frequent liquid diets in small amounts is beneficial. It is concluded that minor degrees of small intestinal failure have a significant impact on the nutritional status of population groups.

Mathias JR, Clench MH. Review: pathophysiology of diarrhea caused by bacterial overgrowth of the small intestine. Am J Med Sci 1985 Jun;289(6): 243-8

"The bacterial overgrowth syndrome constitutes an intestinal problem involving alterations in motility and injury to the brush border and mucosa. The overgrowth of bacteria also causes secretion, malabsorption, and maldigestion. These alterations result in a clinical syndrome that manifests itself as weight loss, malabsorption of specific nutrients, and (usually) diarrhea. There are known causes of bacterial overgrowth, such as intestinal diverticuli or surgical procedures involving a vagotomy, but in our experience most cases remain idiopathic. This review evaluates the mechanisms of bacterial overgrowth, as currently understood, and specifically addresses the known causes of diarrhea that results from bacterial contamination of the small intestine." (Authors' abstract)

Mathis RK see Cohen S

Matseshe JW, Phillips SF. Chronic diarrhea: a practical approach. Med Clin North Am 1978 Jan;62(1):141-54

Many patients who present with chronic diarrhea are not found to have an important organic disease. This review presents a logical and practical approach for treatment based on a definition of chronic diarrhea, brief consideration of the relevant pathophysiology, and a scheme for the clinical evaluation of patients. The history, the physical examination and the initial laboratory tests should lead to a provisional diagnosis, with respect to the organic or functional origin, and the location of disease in the small or the large bowel. Tests used to investigate chronic diarrhea may include further macroscopic. microscopic, and bacterial examination proctosigmoidoscopy: blood tests; barium enema: specific malabsorption, inflammatory bowel diseases, carcinoma of the large bowel. parasitic infections, and metabolic disorders; and determination of serum concentration of gastrointestinal hormones. Specific causes of chronic diarrhea, including functional diarrhea, irritable colon, inflammatory bowel disease, large bowel carcinoma, amebic colitis, giardiasis, disorders, and gastrointestinal hormone-producing tumors, are Patients with suspected functional diarrhea should be investigated at least by stool analysis, proctosigmoidoscopy, and barium enema. Functional diarrhea is common, and these patients are no less susceptible to severe disease than is

the rest of the population. Treatment of various forms of chronic diarrhea is also outlined.

Matts SG see Pillai DK

Mauel J. Mechanisms of survival of protozoan parasites in mononuclear phagocytes. Parasitology 1984 Aug;88(pt 4):579-92

May JT. Antimicrobial properties and microbial contaminants of breast milk - an update. Aust Paediatr J 1984 Nov;20(4):265-9

"A review of recent studies of antibacterial, antiviral and antiprotozoan factors in human breastmilk is presented. Also reviewed are the microbial contaminants that have been detected in human milk with a particular focus on cytomegalovirus and rubella virus, both of which have recently been shown to infect infants via breast milk." (Author's abstract)

Mayberry JF. Some aspects of the epidemiology of ulcerative colitis. Gut 1985 Sep; 26(9); 968-74

Mbere N see Feachem RG

Mebus CA see Torres-Medina A

Meeuwisse GW. High sugar worse than high sodium in oral rehydration solutions. Acta Paediatr Scand 1983 Mar;72(2):161-6

"The literature on oral sugar-electrolyte solutions for treatment of acute diarrhoea is reviewed. Several trials have shown that the solution proposed by the WHO for developing countries containing inter alia 90 mmol/l of sodium and 111 mmol/1 of glucose is safe for short-term oral rehydration. When used in this manner there is no risk for development of hypernatraemia. The surplus base of the solution is not essential and, furthermore, other anions e.g. acetate may be substituted for bicarbonate. Other modifications of the WHO formula have also been successfully tried, e.g. sucrose 4% (117 mmol/l) instead of glucose 2% (111 mmol/l). A somewhat lower concentration of sucrose may, however, prove to be better. Most acute childhood diarrhoeas are not mediated by enterotoxin and thus not of the secretory type, but temporary malabsorption is common. Therefore, the amount of carbohydrate in oral sugar-electrolyte mixtures should be limited. Osmotic diarrhoea due to carbohydrate mixtures should malabsorption is a more likely cause of hypernatraemia in dehydrated children than too much dietary sodium. In developed countries, prepacked sugar-electrolyte mixtures are mainly designed for moderately sick children treated at home. There is no reason to raise the carbohydrate content of these mixtures above that of the WHO formula, but the sodium content must be lower. For most situations in home treatment 50 mmol/l of sodium will be adequate." (Author's abstract)

Mehta S. Investigative approach towards chronic diarrhoea in infants and children. Indian Pediatr 1977 Apr;14(4):303-8

Mekalonos JJ. Cholera toxin: genetic analysis, regulation, and role in pathogenesis. Curr Top Microbiol Immunol 1985;118:97-118

This chapter reviews the existing state of knowledge on the genetics of toxinogenesis in <u>Vibrio cholerae</u> and speculates on the function of the toxin in

the pathogenesis of cholera and other related diarrheal diseases. toxin is a multimeric protein, composed of two subunits, A and B, present in the holotoxin in a ratio of 1:5. The A- and B-subunit genes are arranged in a single transcriptional unit (the $\underline{\mathsf{ctx}}$ AB operon). The nucleotide sequence of the $\underline{\mathsf{ctx}}$ AB operon has provided information regarding translational control in ctx expression and the mechanism of toxin secretion. The observed duplication and amplification of the ctx genetic element, repetitive sequence-1, and the 7-bp repeated sequence in the ctx promoter region have helped establish that repetitive DNA is no longer a topic confined to eukaryotic molecular biology. The cloned toxR gene was shown to complement the defect present in several independently isolated hypotoxinogenic mutants of strain 5698, increasing the toxin production by these strains at least 300- to 1000-fold. In addition to positive control, mediated by toxR, there is also evidence of other regulatory loci influencing cholera toxin expression. The observation that regulatory gene exists in nontoxinogenic as well as toxinogenic strains of V. cholerae has provided another system for studying the evolution of regulatory systems, controlling the expression of accessory genetic elements. Finally, the characterization of <u>ctx</u> site-specific mutants <u>in vivo</u> has provided evidence that cholera toxin plays a more complex role in pathogenesis than previously envisioned.

Mendeloff AI see Calkins BM

Mensch AH see von Graevenitz A

Merson MH. The global problem of acute diarrhoeal diseases and the WHO Diarrhoeal Diseases Control Programme. In: Takeda Y, Miwatani T, eds. Bacterial diarrhoeal diseases. Tokyo: KTK Sclentific Publishers, 1985:1-10

Merson MH see Feachem RG

Merson MH see Litvinov SK

Merson MH see Mahalanabis D

Merson MH see Snyder JD

Metcalf AM, Phillips SF. Ileostomy diarrhoea. Clin Gastroenterol 1986 Jul;15 (3):705-22

Metz SA, McRae JR, Robertson RP. Prostaglandins as mediators of paraneoplastic syndromes: review and up-date. Metabolism 1981 Mar; 30(3): 299-316

Meyer-Kawohl R, Bockermuhl J. [Clostridium difficile-induced enterocolitis: pathogenesis, clinical course, epidemiology and laboratory diagnosis]. Immun Infekt 1986 Apr:14(2):63-7

Midthun K see Kapikian AZ

Milla PJ. Disorders of electrolyte absorption. Clin Gastroenterol 1982 Jan;11(1):31-46

Milla PJ. Intestinal motility and its disorders. Clin Gastroenterol 1986 Jan;15(1):121-36

Miller C see Feachem R

Miller DP, Everett ED. Bacterial enteritis. Missouri Med 1983 May;80(5): 241-8

The incidence of bacterial causes of acute diarrhea, the major clinical manifestations of specific bacterial pathogens and the results of stool-culture survey, performed at the University of Missouri-Columbia Health The authors also describe a clinical Sciences Center, USA, are provided. approach to the diagnosis and treatment of bacterial diarrheas. The bacterial causes of diarrhea can generally be divided into those that produce illness by toxigenic or by direct invasive mechanisms. The former is characterized by watery, large-volume diarrhea and the absence of fever, while the latter is reflected by small-volume stools, often with blood, the presence of fecal leukocytes, and fever. A wide variety of organisms are capable of producing these illnesses. The clinical features resulting from the common agents are presented. At the University of Missouri-Columbia Health Sciences Center, stool cultures were prospectively surveyed over a 15-month period. hundred specimens were received from 533 patients, both adults and children. The overall numbers of bacterial pathogens isolated were low (4.9%), but represent several species, including <u>Salmonella</u> sp., <u>Shigella</u> sp., <u>Yersinia</u> enterocolitica, and <u>Campylobacter</u> sp. <u>Special</u> studies for toxigenic agents were not included in the survey. The presence of fecal leukocytes led to a 9-fold increase in isolation of bacterial pathogens. Even though the University is largely a tertiary-care hospital, this survey reflects the spectrum of organisms that may be seen by practicing physicians. Because of the relatively low yield of bacterial agents, the cost of stool culture and the time involved, it has been suggested that patients selected for culturing of stools should be drawn judiciously. From the review of the agents and the illness patterns, the following criteria would seem appropriate for patient selection: (a) febrile patients with diarrhea; (b) fecal leukocytes present in the stool; (c) diarrhea lasting longer than 5-7 days; and (d) common-source outbreaks (this may involve culturing the suspected vehicle as well as the stools). In general, therapy should be directed toward supportive care. Antimicrobials are indicated only in a few instances. Antibiotics are of proven benefit in patients with shigellosis and have been recommended for nontyphoidal salmonellosis only in infants, immunocompromised patients and when bacteremia is documented.

Minami H, McCallum RW. The physiology and pathophysiology of gastric emptying in humans. Gastroenterology 1984 Jun;86(6):1592-1610

The physiological and pathophysiological aspects of gastric emptying are highlighted, and the clinical problems of gastric motility discussed. Significant advances in the study of gastric motility have been made in recent years. The newer diagnostic techniques and drug applications have been particularly impressive. Dual isotope scintigraphy has provided a tolerable, noninvasive method for defining and quantifying the gastric handling of liquids and solids in various clinical situations. Its reproducibility and patient acceptance will allow more precise evaluation of various treatment regimens in gastroparetic disorders. A better understanding of the part played by gastric emptying dysfunction in diseases, such as gastroesophageal reflux, gastric ulcer, and anorexia nervosa, may lead to more innovative therapeutic approaches to these disorders. A brief account is presented on drugs that delay gastric emptying. Therapy of gastroparesis is also discussed. Metoclopramide has revolutionized the therapy of gastric retention. Its efficacy has been demonstrated in gastroesophageal reflux disease, diabetic gastroparesis,

post-operative gastric stasis, delayed gastric emptying in anorexia nervosa, and idiopathic gastroparesis. The usual dose of metoclopramide is 10 mg, taken 15-30 min before each meal and at bedtime. Domperidone, a benzimidazole derivative, is a specific peripheral dopamine antagonist that stimulates the gastrointestinal tract. Initial therapeutic studies with domperidone have been encouraging. Exciting pharmacologic advances will be predicted on the correlation of pharmacology with electrical, mechanical, and transit aspects of gastric pathophysiology.

Mindel A see Allason-Jones E

Minette HP. Epidemiologic aspects of salmonellosis in reptiles, amphibians, mollusks and crustaceans - a review. Int J Zoonoses 1984 Jun;11(1):95-104

Mir GN see DiJoseph JF

Mirelman D. Ameba-bacterium relationship in amebiasis. Microbiol Rev 1987 Jun;51(2):272-84

"It is generally recognized that there are nonpathogenic and pathogenic strains of Entamoeba histolytica and that the differences between them may be related in part to the type of bacterial species with which they become associated in the human intestine. The development of methods for cultivating the parasite axenically in vitro has made it possible to study the interactions between amoebae-bacteria and mammalian host cells. The present knowledge of the mechanisms and molecules involved in the intercellular recognition and the effect they have on virulence of E. histolytica as well as on the relation between isoenzyme patterns (zymodemes) and pathogenicity, is critically reviewed and discussed." (Author's abstract)

Misiewicz JJ. Human colonic motility. Scand J Gastroenterol 1984;19 (suppl 93):43-51

"Abnormalities of colonic motility are believed to play a major role in many alimentary disorders. Progress in devising suitable therapies has been hampered by imperfect understanding of the complex factors that control colonic motility. Intrinsic and extrinsic nerves, endocrine and paracrine factors and the nature of the luminal contents all affect colonic motor activity (propulsive or segmental) in various degrees, the relative importance of which is difficult to assess. Recent evidence concerning these influences and abnormalities of colonic motility in various diseases is reviewed." (Author's abstract)

Misra BS see Aggarwal P

Mitchell GF. Injection versus infection: the cellular immunology of parasitism. Parasitol Today 1987 Apr;3(4):106-11

Modler S see Perman JA

Molla A, Molla AM, Sarker SA, Khatoon M, Rahaman MM. Effects of acute diarrhea on absorption of macronutrients during disease and after recovery. <u>In:</u> Chen LC, Scrimshaw NS, eds. Diarrhea and malnutrition: interactions, mechanisms, and interventions. New York: Plenum, 1983:143-54

Molla A. Molla AM. Sarker SA. Malabsorption in enteric infection; a

nutritional cost in children with diarrhea. In: Chagas C, Keusch GT, eds. The interaction of parasitic diseases and nutrition, 22-26 October 1985. Vaticana: Pontificia Academia Scientiarum, 1985:71-80.

This review evaluates the important findings regarding nutrient malabsorption possible mechanisms in children during enteric Malabsorption of fat, vitamin B_{12} and xylose were found during the acute stage, and, for some time, after recovery from diarrhea. Infants suffering from chronic diarrhea were found to lose larger amounts of nitrogen and fat through passage of stools. The amount of nitrogen loss was related to the severity of the diarrheal attack. The sources of the stool nitrogen were food, invading microbes, shedding of the gastrointestinal mucosa and leakage of plasma proteins in the gut. Xylose absorption during the acute stage and two weeks after recovery showed that the differences between the two stages of diarrhea were significant. Marked differences could be observed according to the In the acute stage, a reduced nitrogen absorption was etiological agent. observed in diarrheas of all etiologies but more severely so in cases with Shigella infection. Mean calorie absorption in acute diarrhea was affected in children infected with rotavirus. Mean absorption of carbohydrate in rotavirus diarrhea was 78%, which was sufficient to permit rehydration of patients with rehydration solution prepared with sucrose. glucose or Enterotoxigenic E. coli (ETEC) and Shigella-associated diarrhea resulted in reduced growth rates. More than 80% of the children over 3 years of age suffered from lactose malabsorption although children under 6 months of age did not, unless there was a recent history of diarrhea. The prevalence of lactose malabsorption in infants of 7-18 months was significantly associated with recent episodes of diarrhea. Malabsorption secondary to intestinal parasites varied according to the type of parasite, age of the patient and severity of infection. Nutrients which were affected by parasitic infection included fat, lactose, zinc, vitamin A and vitamin B12. Protein-losing enteropathy which contributed to the development of malnutrition was found in patients with giardiasis. Reduced intake, nutrient malabsorption and increased energy output were considered to be the major contributing factors in causing malnutrition. It is concluded that mortality from diarrheal diseases due to bacterial and viral pathogens is a major contributing factor to the overall mortality of children in developing countries.

Molla A see Molla AM

Molla AM, Molla A, Sarker SA, Rahaman MM. Food intake during and after recovery from diarrhoea in children. $\underline{\text{In}}$: Chen LC, Scrimshaw NS, eds. Diarrhea and malnutrition: interactions, mechanisms, and interventions. New York: Plenum, 1983:113-23

This chapter reviews the literature on the effect of an episode of diarrhea on food intake, noting that voluntary food intake is decreased substantially ($\approx 20-70\%$ protein and caloric reduction) during illness. Breastmilk intake is not reduced. There is also substantial evidence that food should not be withheld from patients, as substantial nutrients are absorbed and there is no evidence of prolongation of diarrhea by feeding. The chapter summarizes a study performed by the authors, in which diarrhea due to 4 organisms (Vibrio cholerae, rotavirus, enterotoxigenic Escherichia coli, and Shigella) was studied. Shigella was demonstrated to have the most marked decrease in caloric intake in the acute phase, whereas rotavirus was associated with the most prolonged decrease in caloric intake.

Molla AM, Sarker SA, Hossain M, Molla A, Greenough WB, III. Rice-powder electrolyte solution as oral therapy in diarrhoea due to <u>Vibrio cholerae</u> and <u>Escherichia coli</u>. Lancet 1982 Jun 12;1(8285):1317-9

To replace sucrose or glucose in a standard oral rehydration solution (ORS). rice powder was used in a case-control study to see whether a cereal-based electrolyte solution is effective as a rehydration medium in diarrhea. Of 124 patients with acute diarrhea due to <u>Vibrio cholerae</u> or <u>Escherichia coli</u>, 61 were treated with standard sucrose-electrolyte solution and 63 with an electrolyte solution containing 30 g of rice powder per liter and electrolytes as recommended by the WHO (sodium 90 mmol/l; chloride 80 mmol/l; potassium 20 mmol/l and bicarbonate 30 mmol/l). In vitro hydrolysis converts 80-86% of the rice powder into glucose. Therefore, 30 g of rice powder was given so that at least 20 g of glucose would be liberated in the intestinal lumen. treatments were compared by measuring the rate of purging, change in body weight, serum-specific gravity, urine output and post-hydrolysis sugar content of the stool. The proportions of successfully treated patients in the rice-powder group were 80% for cholera patients and 88% for E. coli patients, which was no different from the results in patients receiving the sucrose-electrolyte solution. The results are also comparable to those of other oral replacement solutions. Failure was due to rates of purging that exceeded the patients' ability to drink enough replacement solution. advantages of using rice powder in the ORS were seen. It was speculated that intraluminal digestion of rice powder liberated glucose gradually without causing the osmotic drag of fluid from the vascular space to the gut lumen resulting in osmotic diarrhea, as is seen when sucrose or glucose exceeds the recommended amount in ORS. This study suggests that treatment with rice powder or any other cereal appropriate for a specific geographical area may permit simultaneous rehydration and nutritional rehabilitation in diarrhea patients with malnutrition.

Molla AM see Ahmed HS

Molla AM see Molla A

Molla AM see Rohde JE

Monico R see Dionigi R

Monsur KA. Epidemiology of <u>Escherichia coli</u> - an important but neglected field [editorial]. J Diarrhoeal Dis Res 1985 Sep; 3(3):128-30

Monsur KA. How this happened? [editorial]. J Diarrhoeal Dis Res 1983 Mar;1 (1):3-4

Montgomerie JZ see Neilands JB

Montrose MS see Shane SM

Moot FR, de Graaf FK. Molecular biology of fimbriae of enterotoxigenic Escherichia coli. Curr Top Microbiol Immunol 1985;118:119-38

Moon MW. Mechanisms in the pathogenesis of diarrhea: a review. J Am Vet Med Assoc 1978 Feb 15;172(4):443-8

Morger H, Steffen R, Schar M. Epidemiology of cholera in travellers, and

conclusions for vaccination recommendations. Br Med J 1983 Jan 15;286(6360): 184-6

"All cases of cholera imported to Europe and North America between 1975 and 1981 were reviewed to assess the danger of cholera for visitors to endemic areas. Data were obtained from the health authorities of the respective countries. From a total of 129 cases notified to the World Health Organization detailed reports were obtained on 117 patients. Of these, 66 (56%) were immigrants, refugees from endemic areas, or foreign workers returning from leave in their native countries. Only 51 (44%) were citizens of countries of Europe or North America. The incidence per journey for foreign travellers visiting Africa or Asia was about 1 in 500,000. Stay in hospital was always short, and fewer than 2% of patients died. In view of the minimal risk and lack of reliability of cholera vaccination, such protection is not indicated for ordinary tourists visiting developing countries." (Authors' abstract)

Moriarty KJ, Turnberg LA. Bacterial toxins and diarrhoea. Clin Gastroenterol 1986 Jul:15(3):529-43

"Bacteria and their toxins are responsible for an enormous burden of diarrhoeal disease. Knowledge about the toxins and their mechanisms of action is limited. Thus, although considerable information is available about the mechanism of action of cholera toxin and a small number of heat-stable enterotoxins, information on the role and action of many others is incomplete. The demonstration of a toxic effect in a test system does not necessarily imply that activity is relevant to the pathogenesis of the diarrhoea. On the other hand, the absence of a toxic effect in experimental systems does not eliminate the possibility that a toxin is responsible for a particular organism's clinical effects. This is a field of active research and much more work is clearly required." (Authors' abstract)

Morisset R see Kurstak E

Morris JA, Jr., Selivanov V, Sheldon GF. Nutritional management of patients with malabsorption syndrome. Clin Gastroenterol 1983 May;12(2):463-74

Morris JG see Levine MM

Morris RJ. Religion and medicine: the cholera pamphlets of Oxford, 1832, 1849 and 1854. Med Hist 1975 Jul;19(3):256-70

Three great cholera epidemics of 19th century Britain generated huge literature on the effects on individual towns and districts. The principal means of transmission of cholera was through water polluted by the excreta of affected patients. The low-lying parts of Oxford provided the ideal environment for the disease to spread. In 1832, 86 people died. In 1849, 44 died, a reduction in part caused by better drainage. The total rose in 1854 to 78, some of the increase being due to the occupation of houses on the poorest land. On each of these epidemics, 3 cholera pamphlets were written. The first pamphlet displayed a blend of moral and physical explanation. The writer advised the Board of Health to look "for moral and religious guidance as to various indulgences, imprudent negligences and generally as to things done or omitted either positively or probably injurious to health". After surveying the "peculiarities of the soil, sanitation, water and atmosphere" of the areas worst affected, he blamed the disease on bad drainage, crowded housing, lack of

ventilation, dirty streets, and "the destitute state of the poor". recommended better street cleansing, improved sewers and water supply, and "the amelioration of the conditions of the poor". The second cholera pamphlet was a carefully tabulated statistical summary of the epidemic, and then a street-by-street account of the cases, with a description of the condition of the areas affected. The statistics were presented to make comparisons possible between 1832 and 1849. The authors of the Oxford report on the 1849 epidemic found little relationship between cholera and occupation, but a strong link between cholera and poverty; 122 of the 144 cases were "proper objects of charity", as were 140 of 174 in 1832. The Oxford doctors then went on to these local findings and their implications as part of a campaign for improvement of drainage, cleansing, sewerage, and water supply in Oxford. memoir of the 1854 cholera at Oxford was published after the third epidemic. The author combined the religious and scientific factors and recognized the value of Snow's theory that cholera is transmitted by water polluted by cholera patients' excreta. As long-term remedies, the author suggests sanitary reforms to be accompanied by a wide program of religious, educational and intellectual improvement.

Morrison DC. Bacterial endotoxins and pathogenesis. Rev Infect Dis 1983 Sep-Oct;5(suppl 4):S733-47

"Recent advances in endotoxin biochemistry, cell biology, and -immunopathology are reviewed. Discussion is focused on the endotoxin-unresponsive C3H/HeJ mouse and the contributions the studies of this mouse have made to current concepts of mechanisms of endotoxin action. The extension of the observations documented with this experimental model provide a firm foundation for productive future research into the precise role of bacterial endotoxins in the protection against, and exacerbation of, gram-negative bacterial sepsis."

Morton RE see Reisman T

Mosalova NM. [Cytochemical characteristics of lymphocytes in patients with chronic enterocolitis]. Vrach Delo 1985 Mar;(3):81-3

Mosley WH, Khan MU. Cholera epidemiology – some environmental aspects. Prog Water Technol 1979; $11\{1-2\}:309-16$

Details of longitudinal, community-based, epidemiological studies from endemic areas in Bangladesh are given. These lay emphasis on the essential link between the transmission of cholera and the exposure to specific environmental situations. Cholera has a distinctive epidemiological pattern. Epidemics, even in endemic areas, are sharply localized in time and place. The "place" specificity points to the essential requirement for water to facilitate the transmission of disease. The age, sex, occupational risks of disease are primarily related to exposure to contaminated water. Cholera is rarely seen in infants aged under one; the highest incidence in the endemic areas is among children, their age ranging from 4 to 8 years; in epidemics in newly infected areas, adult males are typically the prominent victims. Selective exposure to contaminated water can account for most of these patterns. This exposure may, however, involve small doses of Vibrio cholerae ingested with water while rinsing the mouth, cleaning utensils, or with fresh vegetables, or fish, especially shellfish. The epidemiological pattern of cholera contrasts greatly with that of other diarrheal diseases. There is need of a clear

epidemiological picture of the predominant routes of transmission of each etiological agent.

Motil KJ, Grand RJ. Nutritional management of inflammatory bowel disease. Pediatr Clin North Am 1985 Apr;32(2):447-69

This paper reviews the epidemiology and etiology of malnutrition and growth failure in inflammatory bowel disease. Chronic inflammatory bowel disease represents two intestinal disorders of childhood: ulcerative colitis and Because of the clinical significance of the symptoms Crohn's disease. associated with these diseases, nutritional considerations have a major impact in the therapeutic management of inflammatory bowel disease in childhood. Individual nutrient deficiencies may occur in children but more frequently there is protein-energy malnutrition with progressive growth retardation. etiology of malnutrition in inflammatory bowel disease is multifactoral and may excessive intestinal dietary intake, inadequate Malabsorption or its malabsorption, and increased nutrient requirements. complications are more common in patients with long-standing Crohn's disease and weight-for-age deficits. The indications for nutritional intervention in inflammatory bowel disease include the provision of primary nutritional support during active inflammatory disease, the treatment of individual nutrient deficiencies, and the reversal of malnutrition and growth arrest. Nutritional disorders and their complications in chronic inflammatory bowel disease can be prevented by monitoring anthropometric and laboratory indices, and by promptly instituting enteral or parenteral nutritional rehabilitation when indicated.

Moudgil KD, Narang BS. Pathogenesis of typhoid fever. Indian J Pediatr 1985 Jul-Aug;52(417):371-8

This paper describes the pathogenesis of typhoid fever. The incubation period of typhoid fever is generally 7-14 days, but can vary from 3 to 60 days. mean duration of untreated illness is about 4 weeks. The outcome of interaction between the typhoid bacilli (Salmonella typhi) and humans is determined during the early hours after ingestion of the organisms. site where the organisms penetrate the gastrointestinal tract to reach the blood stream is not known with certainty. S. typhi reach the small intestine shortly after ingestion. The organisms then penetrate the mucosa with minimal epithelial destruction and enter the intestinal lymphatics to be carried to the blood stream. Organisms re-enter the blood stream producing continuous bacteremia for days or weeks which initiates the symptomatic phase of infection. The normal flora of the upper intestinal tract is a protective enhances mechanism against invasion by S. typhi. Malnutrition The host defence mechanism in typhoid susceptibility to typhoid infection. The host defence mechanism in typhoid fever is not clearly understood yet. Humoral and cellular immune responses are known to develop during typhoid fever. The role of endotoxin is discussed.

Mozin MJ <u>see</u> Loeb H

Muller JM, Keller HW, Brenner U, Walter M, Holzmuller W. Indications and effects of preoperative parenteral nutrition. World J Surg 1986 Feb;10(1): 53-63

Muller R <u>see</u> Feachem RG

Murray BE. Resistance of Shigella, Salmonella, and other selected enteric

pathogens to antimicrobial agents. Rev Infect Dis 1986 May-Jun;8(suppl 2): \$172-81

"Antimicrobial agents are commonly used therapeutically and prophylactically for travelers' diarrhea. Resistance of enteric pathogens to these agents may prevent the success of such therapy, with the result depending upon the level of resistance and the drug concentrations achieved in the gastrointestinal tract. Data from a number of geographic locations were collected to determine whether consistent trends exist and whether predictions can be made regarding the susceptibility of various enteric pathogens worldwide. These data showed marked variability in the prevalence of resistance. Among Shigella, percentage of strains resistant to commonly used agents varied within following ranges: ampicillin, 7% (Dacca) to 87% (Thailand); tetracycline, 11% (Sri Lanka) to 91% (Mexico); and trimethoprim-sulfamethoxazole, 0 (Dacca, 1980) to 55% (Dacca, 1984). Resistance in Salmonella strains showed a similar marked variability. Few strains of enterotoxigenic Escherichia coli (<10%) were resistant to trimethoprim-sulfamethoxazole. Relatively recent isolates of all pathogens examined tended to be more resistant than earlier isolates to trimethoprim-sulfamethoxazole as well as to other agents." (Author's abstract)

Nair GB see Pal SC

Nalin DR. Oral replacement of water and electrolyte losses due to travellers' diarrhoea. Scand J Gastroenterol 1983;18(suppl 84):95-8

"An oral rehydration solution (ORS) containing glucose, sodium and potassium chloride, and sodium bicarbonate, forms the basis of an oral rehydration therapy (ORT) regime which is detailed. The regime can be modified for adults, older children, younger children and neonates, and may be used in combination with other methods of augmented fluid intake such as juices and plain water supplements. Milk and soft foods should be added to the regimen. Guidelines for patients include methods of assessing rehydration. The harmful effects of 'therapeutic' starvation are stressed." (Author's abstract)

Narang BS see Moudgil KD

Navin TR, Juranek DD. Cryptosporidiosis: clinical, epidemiologic, and parasitologic review. Rev Infect Dis 1984 May-Jun;6(3):313-27

"Cryptosporidium, an intestinal protozoan parasite, is a well-known cause of diarrhea in animals but has been recognized only recently as a cause of human disease. Since 1976, 58 cases of cryptosporidiosis in humans have been reported; 18 of the patients had normal immune function, and 40 had various immunologic abnormalities, the most common of which, acquired immune deficiency syndrome (AIDS), occurred in 33 patients. Patients with normal immune function had self-limited diarrhea, but patients with immunologic abnormalities often developed severe, irreversible diarrhea; 22 patients have died. The diagnostic of cryptosporidiosis can now be made noninvasively, but increased diagnostic proficiency has led to little improvement in control or treatment of the disease. Although 23 compounds have been evaluated in experimentally infected animals and 20 drugs have been used in human clinical trials, no effective chemotherapeutic agent for cryptosporidiosis has been identified to date." (Authors' abstract)

Navin TR. Cryptosporidiosis in humans: review of recent epidemiologic studies.

Eur J Epidemiol 1985 Jun:1(2):77-83

"Since 1976, when <u>Cryptosporidium</u> was first recognized as a human pathogen, understanding of the epidemiology of this protozoan parasite has increased substantially. This review discusses 14 recently published studies of the occurrence of cryptosporidiosis in developed and developing countries and compares their findings with those of previous epidemiologic reports. These studies show that cryptosporidiosis is an important public health problem worldwide. Although cryptosporidiosis was first recognized as a problem in immunosuppressed patients, persons with normal immunologic function are also affected. This appears to be especially true for children in developing countries; of 1,035 children with diarrhea reported in several surveys, 79 (7.6%) had <u>Cryptosporidium</u>. Findings from studies that included both asymptomatic as well as symptomatic persons have demonstrated that <u>Cryptosporidium</u> infections are rare in persons without symptoms, suggesting that <u>Cryptosporidium</u> should not be thought of as an opportunistic parasite. Early studies documented the potential for animal-to-human transmission of <u>Cryptosporidium</u>, but it is now clear that many, perhaps most, <u>Cryptosporidium</u> infections in humans are not acquired directly from infected animals. Although alternate modes of transmission are just beginning to be explored, evidence indicates that person-to-person spread is important." (Author's abstract)

Nazer H. Astrovirus gastroenteritis. J Trop Pediatr 1985 Apr;31(2):67-71

Astrovirus-associated gastroenteritis is a short illness. This paper reviews the morphology, epidemiology and pathogenicity of astrovirus and outlines the clinical features, diagnostic techniques and management of astrovirus-associated gastroenteritis. Serial stool examinations can establish the duration of astrovirus excretion. The introduction of more sensitive diagnostic techniques can lead to more detailed and accurate assessment of astrovirus pathogenicity. There is usually a mild degree of dehydration without any appreciable systemic disturbances. Conventional oral rehydration therapy is effective in most patients.

Neilands JB, Bindereif A, Montgomerie JZ. Genetic basis of iron assimilation in pathogenic <u>Escherichia coli</u>. Curr Top Microbiol Immunol 1985;118:179-95

Nelson JD. Etiology and epidemiology of diarrheal diseases in the United States. Am J Med 1985 Jun 28;78(suppl 6B):76-80

"Accurate data on the frequency of acute diarrheal illness and the distribution of pathogens are not available for several reasons, including the facts that only a small fraction of cases come to the attention of physicians and that available diagnostic tests establish an etiology in only about half of these. In a survey of three groups of patients in a community (upper-middle class and lower class outpatients and hospitalized infants), a possible cause was found in fewer than 20 percent of outpatients, and rotavirus accounted for the majority. However, asymptomatic colonization with rotavirus is very common in early infancy, and demonstration of the antigen correlates with a causative role in diarrhea in only about one-half of cases. Data on cases of shigellosis over a 15-year period in Dallas compared with the Centers for Disease Control national data demonstrate that epidemiologic patterns in any given community can differ substantially from the national data. Day-care centers are important sources of Giardia and Shigella infections in the community. The epidemiologic roles of newly reported causes of diarrhea (Campylobacter

laridis, Blastocystis hominis, Cryptosporidium species, and Aeromonas hydrophila) and being explored. Generalizations about etiology and epidemiology of pathogens in diarrheal disease are invalid unless the population group is defined socioeconomically and geographically." (Author's abstract)

Nelson JD, Kusmiesz H, Shelton S. Oral or intravenous trimethoprimsulfamethoxazole therapy for shigellosis. Rev Infect Dis 1982 Mar-Apr;4(2): 546-50

This paper reviews the previously published studies of oral trimethoprim-sulfamethoxazole (TMP-SMX) therapy for shigellosis, presenting new data on intravenous TMP-SMX therapy and commenting on post-therapy asymptomatic carriage of Shigella. A review of the literature on treatment of shigellosis with TMP-SMX summarizes comparative studies of 149 cases and noncomparative studies of 147 cases. Bacteriologic and clinical success rates were >90% and TMP-SMX compared favorably with other drugs. Eleven children hospitalized for severe Shigella colitis were given TMP-SMX intravenously as initial therapy. Bacteriologic and clinical responses were comparable to those observed with oral TMP-SMX. Pharmacokinetic studies revealed substantial differences between children and adults; a higher ratio of SMX to TMP (50:1) was found in children. TMP-SMX can be regarded as the drug of choice for shigellosis in areas where ampicillin resistance is prevalent.

Netchvolodoff CV, Hargrove MD, Jr. Recent advances in the treatment of diarrhea. Arch Intern Med 1979 Jul;139(7):813-6

Recent advances in the treatment of diarrhea, both acute and chronic, are discussed. Often, treatment is not indicated in individuals who have acute diarrhea that improves spontaneously in one or two days. Treatment is indicated if the diarrhea causes a large volume of fluid loss, is severe from the onset, is accompanied by blood in the stool, or persists without improvement for more than 24 h. Parenteral and oral fluid replacements, drugs that decrease intestinal motility, and antibiotic therapy for specific microorganisms are also discussed. An exact diagnosis is essential for proper management of patients with chronic diarrhea. A complete history and physical examination, stool examination for blood, leukocytes, parasites, and fat, proctoscopic examination, roentgenograms of the stomach, small intestine, and colon are required for almost all patients. Although treatment of patients with functional diarrhea is difficult, drugs, such as diphenoxylate hydrochloride and anticholinergic agents, may provide partial symptomatic relief. Causes, diagnoses and therapy in different types of organic diarrhea are also outlined.

Neter E. Enteropathogenicity: recent developments. Klin Wochenschr 1982 Jul 15;60(14):699-701

"Recent studies have added important new information to our understanding of the pathogenesis and etiology of diarrheal disease. Vibrio cholerae produces a heat-labile enterotoxin, affecting cyclic AMP. A very similar heat-labile enterotoxin is produced also by certain strains of Escherichia coli as well as by Citrobacter, Klebsiella, and Aeromonas. E. coli may also produce a heat-stable enterotoxin, stimulating guanylate cyclase activity. In order to produce the pathologic effects, E. coli first attaches to epithelial cells of the intestinal tract by means of pili or surface antigens. Enterotoxin can be

demonstrated by both in vivo and in vitro tests, but none are yet suitable for routine diagnostic laboratories. A third mechanism whereby \underline{E} , \underline{coli} causes diarrhoeal disease consists of enteroinvasiveness. Campylobacter, Yersinia, and Clostridium difficile have been added to the list of enteric pathogens of man." (Author's abstract)

Neumann V, Wright V. Arthritis associated with bowel disease. Clin Gastroenterol 1983 Sep;12(3):767-95

Ngokwey N see Jelliffe EFP

Nicholl CG, Polak JM, Bloom SR. The hormonal regulation of food intake, digestion, and absorption. Annu Rev Nutr 1985;5:213-39

Niemegeers CJE see Awouters F

Nime FA, Burek JD, Page DL, Holscher MA, Yardley JH. Acute enterocolitis in a human being infected with the protozoan <u>Crytosporidium</u>. Gastroenterology 1976 Apr:70(4):592-8

"A 3-year-old child with severe acute self-limited enterocolitis was found on rectal biopsy to be infected with the protozoal parasite <u>Cryptosporidium</u>. This organism is known to infect a variety of vertebrates, but this is the first report of infection by <u>Cryptosporidium</u> in a human being. Both light and electron microscopic findings in the rectal biopsy are reported. It is suggested, on the basis of the severity of the clinical symptoms, and on the pathological changes in the rectum, that the organism in this case is likely to have been the cause of the enterocolitis and thus to have been a pathogen rather than a commensal. The source of the infection in this child could not be established. The value of sigmoidoscopy and biopsies is noted in this condition and as a general method for determining the etiology of a gastrointestinal infection in cases where other studies are negative." (Authors' abstract)

Norrby SR. Problems in evaluation of adverse reactions to β -lactam antibiotics. Rev Infect Dis 1986 Jul-Aug;8(suppl 3):S358-70

"Despite their high degree of safety, B-lactam agents cause adverse reactions. This article deals with the types of adverse reactions to various β -lactam agents that have been reported and, especially, with the difficulties involved in monitoring and evaluating these reactions. Comparisons of the results of studies of the same drug carried out in various countries show striking differences in the incidence of adverse effects reported. A reason for this variation is the lack of strict definitions of the events that should be Only rarely are all the unexpected events regarded as adverse effects. occurring in patients in clinical studies reported, but the investigators are allowed to make subjective judgments about which reactions are related to the drug tested and are thus reportable. With such procedures there is an inherent risk of overlooking unexpected adverse effects. Also, in the analysis of laboratory adverse reactions the rule that "one finds what one looks for" applies. It seems obvious that if stricter rules for registration of adverse effects had been applied, the toxic effects of various β -lactams, such as the nephrotoxicity of cephaloridine and the coagulopathies associated with moxalactam, would have been detected much earlier." (Author's abstract)

Nunoue T. Epidemiological aspect of viral diarrhea in tropical area. Jpn J Trop Med Hyg 1985 Mar:13(1):51

Gastroenteritis needs to be successfully treated in the tropical areas, because higher mortality rates are prevailing there than in the temperate and developed countries. Recent studies have revealed that viral infection plays an important role in diarrheal disease. Rotavirus, which consists of 2 subgroups and at least 4 serotypes, is a major cause of viral diarrheas, but one of top 3 infectious agents, together with Escherichia coli and Salmonella. Rotavirus is frequently isolated from the stools of children with diarrhea in tropical countries, e.g. 17% in Mexico, 10% in Guatemala, 26% in Costa Rica, 41% in Venezuela, 21% in Equador, 50% in Nigeria, 18% in Central Africa, 27% in Ethiopia, 26% in south India, 24% in Bangladesh, 30% in Thailand, 40% in Malaysia and Indonesia, 67% in Vanuatu, and 17% in the Philippines. In Metro Manila, Philippines, rotavirus excretion is as frequent as 43%. A rotavirus vaccine given orally may prove effective in preventing rotavirus diarrhea. (Modified author's abstract)

The nutritional origin of cataracts. Nutr Rev 1984 Nov: 42(11):377-9

Nye FJ. Travelers' diarrhoea. Clin Gastroenterol 1979 Sep;8(3):767-81

Oblapenko GP see Litvinov SK

O'Brien AD, Holmes RK. Shiga and Shiga-like toxins. Microbiol Rev 1987 Jun; 51(2):206-20

"Shigella dysenteriae type 1 (Shiga's bacillus) produces a potent toxin, called Shiga toxin that was discovered at the beginning of this century. Shiga toxin is the prototype for a family of related toxins called Shiga-like toxins (or Vero toxins) made by Shigella sp., Escherichia coli, and several species Vibrio, Salmonella, and Campylobacter. These toxins all have cytotoxic, enterotoxic, and lethal activities, and they can be separated into at least two serologically distinct groups. Most strains of E. coli that produce large amounts of Shiga-like toxins have been isolated from stools of patients with diarrhea, bloody diarrhea, hemorrhagic colitis, or hemolytic uremic syndrome. Some of these highly toxinogenic E. coli strains belong to enteropathogenic serotypes associated with diarrheal disease in infants, whereas others are of the enterohemorrhagic serotype 0157:H7. Production of Shiga-like toxins is often controlled by bacteriophage conversion in E. coli, and a single E. coli strain can synthesize more than one kind of Shiya-like toxin. The primary goals of this review are to summarize recent advances concerning the structure. mode of action, immunochemistry, and genetics of the Shiga and Shiga-like toxin family. The circumstantial data which implicate these toxins as potential and for ma.ior virulence factors for shigellae enteropathogenic enterohemorrhagic strains of E. coli are also critically reviewed." abstract)

O'Brien TF. Resistance of bacteria to antibacterial agents: report of Task Force 2. Rev Infect Dis 1987 May-Jun;9(suppl 3):S244-60

O'Connor RW see Col NF

O'Connor TW. Pseudomembranous enterocolitis: a historical and clinical review. Dis Colon Rectum 1981 Sep;24(6):445-8

"Pseudomembranous enterocolitis (PMEC) was first documented in 1893. Since this initial description, confusion has reigned in the medical literature concerning its nature and differentiation from such entities as necrotizing enterocolitis and staphylococcal enterocolitis. Since the 1950s, volumes have been written on PMEC and its association with a multitude of different antibiotics. PMEC has generally been used as somewhat of a "waste-basket" designation, being applied to any postoperative patient who develops significant diarrhea while on broad-apectrum antibiotics. More recently, a resurgence of interest in PMEC has led to its recognition as a specific disease entity and to a greater understanding of its etiology. The current review traces the history of PMEC, distinguishes it from similar disease processes, and describes its clinical presentation, diagnosis, and management. PMEC is particularly distinguished from antibiotic-associated diarrhea and certain forms of antibiotic-associated colitis." (Author's abstract)

O'Donnell S see Levine MM

Old DC. Bacterial adherence. Med Lab Sci 1985 Jan; 42(1):78-85

In the past 20 years, bacterial adherence has developed into a very active area of research not only among microbiologists and epidemiologists investigating infectious diseases but also among those involved with the broader aspects of microbial ecology. The ability of bacteria to stick to surfaces is an important determinant influencing the successful colonization of different niches. Adhesion of bacteria to the mucosal surfaces of different hosts is likely to be of particular importance in the early stages of many bacterial infections. A knowledge of the molecular basis of interactions between bacterial adhesions and host receptors may lead to improved means to control and treat bacterial infections in man and animals by using receptor analogues and adhesion vaccines. (Modified author's abstract)

Olsen WA see Ryan ME

Ooms L. Alterations in intestinal fluid movement. Scand J Gastroenterol 1983;18(suppl 84):65-77

"Fluid loss in diarrhoea is caused by alterations in the basic mechanisms of transport both in the small intestine and in the colon. The role of blood flow is discussed with reference to anatomy and blood flow distribution, fluid transport, luminal pressure and intestinal motility. The control of intestinal fluid transport and of absorption and secretion processes by intracellular and extracellular mediators is reviewed. Treatment of disorders of fluid transport consist of fluid replacement therapy or the use of antidiarrhoeal agents with high specificity and safety, such as loperamide. The effects of interactions between loperamide and bacterial toxins, theophylline and prostaglandins on the antisecretory activity of loperamide are detailed." (Author's abstract)

Ooms L, Degryse A. Pathogenesis and pharmacology of diarrhea. Vet Res Commun 1986 Sep;10(5):355-97

Oral cholera vaccines [editorial]. Lancet 1986 Sep 27;2(8509):722-3

Ordal GW. Bacterial chemotaxis: biochemistry of behavior in a single cell. CRC Crit Rev Microbiol 1985;12(2):95-130

Orenstein WA see Hinman AR

Othenjiann R see Wormann 8

Owen RL, Brandborg LL. Mucosal histopathology of malabsorption. Clin Gastroenterol 1983 May:12(2):575-90

Ozeretskovskaya NN. Intestinal parasitic infections. Scand J Infect Dis 1982;(suppl 36):46-51

Page DL see Nime FA

Pal SC, Nair GB. Epidemiology of campylobacteriosis in developing countries. ICMR Bull 1984 Jul;14(7):1-4

This review paper summarizes a brief overview of the epidemiology of <u>Campylobacter</u> enteritis in developing countries on the basis of existing data. The <u>discussion</u> includes prevalence, age and sex distribution, seasonality, clinical profile, and modes of transmission of campylobacteriosis. The roles of animal and human reservoirs of <u>Campylobacter</u> species are also outlined. The review shows that the epidemiology of <u>Campylobacter</u> enteritis in developing countries differs markedly from those of the developed countries. In the developed countries, the disease is acquired primarily through the consumption of contaminated food and drinks, the environment playing a less significant role. While in developing countries, the environment with its high potential for host contamination appears to play a major role in the onset of the disease. Poor sanitary conditions and improper personal hygiene seem to lead to greater exposure to the organism.

Palumbo PE see Smith SM

Paneth N. Hypernatremic dehydration of infancy: an epidemiologic review. Am ϑ Dis Child 1980 Aug;134(8):785-92

Discussed here is the occurrence of hypernatremic dehydration, defined as a serum sodium level of 150 mmol/l or higher, in the clinical setting of diarrheal dehydration in otherwise healthy children. Almost all clinical reports comment on the relatively high mortality associated with hypernatremic dehydration. Infants with hypernatremic dehydration usually have a distinct clinical appearance. They have fewer signs of dehydration, and more signs of central nervous system dysfunction - irritability, lethargy, coma, convulsions. The rate of development of hypernatremia plays an important role in the degree of central nervous system dysfunction. Hypernatremic dehydration can be produced by a variety of causes or combinations of causes. Only tentative conclusion can be drawn from the limited epidemiological information available. It seems unlikely that the source of variance in serum-sodium concentration in dehydration is due to differences in the infecting agent. The fact that both hypernatremic dehydration and nonhypernatremic dehydration have peak incidences in the same age group at the same time of year suggests that the same etiological agents are operative. A strong line of evidence implicates dietary factors. One study found a statistically significant correlation between the sodium concentration and the total osmolarity of the formulas generally fed to infants and their risk of having hypernatremic dehydration. Many studies of hypernatremic dehydration have noted a greater tendency for such infants to be febrile. Stool electrolyte values might be expected to differ in hypernatremic dehydration and nonhypernatremic dehydration. But, the variance in serum-sodium concentration in diarrhea does

generally not correlate with stool-sodium concentration. A few studies have noted that infants with hypernatremic dehydration are more likely to have an explosive onset of disease. Future research efforts should be directed at the elucidation of the relationship between hypernatremic dehydration and diet as well as between hypernatremic dehydration and of the more recently discovered enteric pathogens.

Patra FC see Mahalanabis D

Patterson M, Schoppe LE. The presentation of amoebiasis. Med Clin North Am 1982 May;66(3):689-705

Many people around the world harbor Entamoeba histolytica or a parasite closely resembling it. This, combined with the high degree of mobility, means that outbreaks and sporadic cases of amebiasis will continue to plague the society. This article presents the pathogenicity of the disease, reviews the pathology and the variety of ways amebiasis may appear, and discusses the problems of diagnosis. clinical presentations of parasitologic The (noninvasive-carrier state, dysentery, nondysenteric colonic disease, and ulcerative post-dysenteric colitis) and extraintestinal amebiasis (amebic liver abscess and cutaneous amebiasis) have been discussed. The most reliable method for diagnosing intestinal amebiasis appears to be the demonstration of hematophagenous trophozoites either in stools or from materials aspirated from lesions seen at the time of proctoscopy. Since morphologic diagnosis may be difficult or impossible in amebiasis, the finding of an immunologic response can be of significant help in diagnosis as well as in epidemiologic study. The results of 4 commonly used serologic tests are comparable, and will detect most cases of symptomatic amediasis. It has been suggested that the available serologic tests are adequate for most clinical situations.

Pearson DJ, McKee A. Food allergy. Adv Nutr Res 1985;7:1-37

Pearson RD, Hewlett EL, Guerrant RL. Tropical diseases in North America. DM 1984 Mar:30(6):1-68

Penner JL. The genus <u>Campylobacter</u>: a decade of progress. Clin Microbiol Rev 1988 Apr:1(2):157-72

Peppercorn MA see Baker RW

Perman JA, Modler S. Role of the intestinal microflora in disposition of nutrient in the gastrointestinal tract. J Pediatr Gastroenterol Nutr 1983:2(suppl 1):S193-6

Persistent diarrhoea in children in developing countries: memorandum from a WHO meeting. Bull WHO 1988;66(6):709-17

"This Memorandum summarizes current knowledge of the epidemiology, etiology, and pathophysiology of persistent diarrhoea and describes current approaches to its management. A number of research topics are presented which focus especially on improving understanding of the causes of persistent diarrhoea and on developing more effective methods for treatment and prevention." (Authors' abstract)

Persson PG, Ahlbom A, Hellers G. Crohn's disease and ulcerative colitis.

review of dietary studies with emphasis on methodologic aspects. Scand J Gastroenterol 1987 May;22(4):385-9

Pettersson U see Hyypia T

Phillips AD see Candy DCA

Phillips SF see Matseshe JW

Phillips SF see Metcalf AM

Phillips SM see Brown KR

Pickering LK, Bartlett AV, Woodward WE. Acute infectious diarrhea among children in day care: epidemiology and control. Rev Infect Dis 1986 Jul-Aug;8 (4):539-47

"The incidence of diarrhea among children in day care centers is highest for those under three years of age. Limited studies indicate that diarrhea occurs more frequently among children enrolled at these centers than among age-matched children cared for at home or in family day care. Most reported outbreaks have been caused by rotavirus, Giardia, Shigella, or combinations of organisms. Children in day care centers commonly excrete enteropathogens in the absence of symptoms; the significance of this phenomenon in transmission is An association between higher rates of diarrhea and selected characteristics of centers - the most important of which is the presence of nontoilet-trained children - has been shown. The contamination of hands. The contamination of hands, communal toys, and other classroom objects as well as a lack of infection control measures play a role in the transmission of enteropathogens in outbreaks of diarrhea in day care centers. Spread of infection from nontoilet-trained children in centers to their families is common. Potential ways of dealing with this situation include education, development, implementation, and enforcement of regulations; and use of infection control measures. However, the effectiveness of specific control measures has not been systematically evaluated." (Authors' abstract)

Pickering LK. Evaluation of patients with acute infectious diarrhea. Pediatr Infect Dis 1985 May-Jun;4(suppl 3);S13-19

Pickering LK. Infections in day care. Pediatr Infect Dis J 1987 Jun;6(6): 614-7

"Infections in children in day care are common but can be limited by several measures which include education of providers and staff in standards of hygiene, maintenance of basic techniques of infection control, appropriate use of the physical facilities of the day care facility and maintenance of recommended immunization schedules of children and staff." (Author's abstract)

Pickering LK. Problems in diagnosing and managing giardiasis. Pediatr Infect Dis 1985 May-Jun;4(suppl 3):S6-10

Pickering LK. Rotaviruses infection. Pediatr Infect Dis 1985 May-Jun;4(suppl 3):S2-6

Rotavirus infection produces significant morbidity and mortality in humans, mostly in children aged under 2. In this review, particular attention has been

of rotaviruses, clinical and epidemiologic the description manifestations of rotavirus infection, diagnosis, treatment and prevention of the disease. Rotaviruses, a genus of the family reoviridae, have an inner and outer capsid, and possess a genome consisting of 11 segments of double-stranded ribonucleic acid. Most rotaviruses from mammalian and avian species share a common antigen detected by complement fixation and immunofluorescence, and possess a subgroup antigen which separates most strains into two subgroups. Rotavirus usually produces sporadic episodes of diarrhea rather than large outbreaks. Rotavirus infection accounts for about 15 to 50% of acute diarrhea cases presenting to hospitals in tropical countries and 35 to 60% in temperate areas. Incidence of rotavirus diarrhea is higher in cooler months in temperate climates, whereas a lack of seasonality has been reported from the tropical countries. Rotavirus has a mean incubation period of 2 days, and the usual duration of fecal excretion is 8 to 10 days. The usual duration of the illness is 5 to 7 days. Rotaviruses are assumed to be transmitted by the fecal-oral route. The disease occurs in all age groups with the peak occurrence in children aged 6 to 24 months. Stools are usually watery in children with rotavirus diarrhea, and do not usually contain blood or fecal leukocytes; mucus is occasionally seen. Stools collected on the third to fifth days after onset of illness are most likely to contain the virus. The ability to diagnose of rotaviruses, and to rotavirus, to study the epidemiology characteristics of individual strains depend on availability of rapid detection assays. The enzyme-linked immunosorbent assay for rotavirus antigen in stool has proved useful and applicable in field settings. A number of enzyme immunoassays have been developed, and they are easy to use, reliable and more A commercially available latex microscopy. sensitive than electron agglutination test could be used to screen large numbers of stool specimens or for routine diagnostic requirements in laboratories. The treatment of patients with rotavirus diarrhea includes prevention and/or correction of dehydration using oral glucose-electrolyte solutions. Attempts are being made to develop an effective rotavirus vaccine.

Piggot PJ, Hoch JA. Revised genetic linkage map of <u>Bacillus</u> <u>subtilis</u>. Microbiol Rev 1985 Jun;49(2):158-79

Pillai DK, Matts SG. Chronic inflammatory bowel disease--a review. Br J Clin Pract 1983 May;37(5):165-72

Pirie A. Vitamin A deficiency and child blindness in the developing world. Proc Nutr Soc 1983 Jan;42(1):53-64

Pitlik S, Berger SA, Huminer D. Nonenteric infections acquired through contact with water. Rev Infect Dis $1987\ Jan-Feb; 9(1):54-63$

In recent years, water has been recognized as an important vehicle for extraintestinal infection. Although water is an obvious vehicle of gastrointestinal illness, there has been no comprehensive review of infectious diseases acquired through nonenteric, physical contact with water. A variety of pathogens are acquired through occupational, recreational, and even therapeutic contact with water. The nature of nonenteric waterborne diseases is often determined by the ecology of aquatic pathogens. Such infections are basically two types: superficial, involving damaged or previously intact mucosa and skin; and systemic, often serious infections that may occur in the setting of depressed immunity. A broad spectrum of aquatic organisms, including viruses, bacteria, fungi, algae, and parasites, may invade the host through

such extraintestinal routes as the conjunctivae, respiratory mucosa, skin, and genitalia. Humans are brought into contact with waterborne organisms through circumstances of employment, recreation, travel, and even hygiene or medical intervention. Infection may remain localized to the portal of entry or progress to severe and often fatal disease. (Modified authors' abstract)

Polak JM see Nicholl CG

Poley JR. Causes of chronic diarrhea in infants and children. Postgrad Med 1970 Dec:48(6):143-7

This paper discusses the causes of chronic infantile diarrhea and their diagnosis. A reliable feeding history of the patient helps diagnosis by enabling the physician to put the newer diagnostic tests to their optimal use. In addition, growth data should be obtained whenever possible. The symptoms and diagnosis of various types of diarrhea caused by dietary proteins and carbohydrates are discussed. Both chronic ulcerative colitis and Crohn's disease may be associated with chronic or recurrent diarrhea. It is suggested that careful proctoscopy and X-ray study of the intestine may assist in differentiating ulcerative colitis from Crohn's disease. The use of drinking water, containing a significant amount of sulfates in infant formula, may also lead to diarrhea which can be severe, and being usually watery. The World Health Organization, therefore, recommends that water with a sulfate content of more than 400 mg/l is unsafe for infant feeding.

Polin RA. Monocional antibodies against microorganisms. Eur J Clin Microbiol 1984 Oct;3(5):387-98

Pollock RV, Carmichael L. Caninê viral enteritis. Recent developments. Mod Vet Pract 1979 May:60(5):375-80

Pomerantz RJ see Ho DD

· Ponette E see Vantrappen G

Poppensiek GC, Kahrs RF. Twenty-five years of progress in understanding major infectious diseases of dairy cattle. J Dairy Sci 1981 Jun:64(6):1443-64

Porter P, Linggood MA. Development of oral vaccines for preventing diarrhoea caused by enteropathogenic Escherichia coli. J Infect 1983 Mar;6(2):111-21

Escherichia coli is well established as a leading etiological agent of severe diarrheal disease. Intensive systems of rearing food animals have contributed greatly to increasing the prevalence of this disease. In all species of host, evidence for intestinal adhesiveness of \underline{E} , coli is to be expected, because enteropathogenic strains multiply rapidly in the small intestine during the course of the disease. The fact that the K88 antigen is host-specific indicates the presence of a unique receptor on the intestinal mucosa. This receptor has been shown by immunofluorescence studies to be predominantly in the brush border of the enterocyte and to a lesser extent in those parts of the mucous membrane which are not readily accessible from the lumen. It is to be expected that antibodies to the bacterial adhesions would be an important means of immunoprophylaxis. Although certain molecular characteristics of adhesion are essential for establishing infection, an antibody that specifically blocks adhesion is not essential for maintaining protection of the host. Thus,

although K88 antigen is common to many O serotypes that are enteropathogenic for the pig, antibodies to it are not the essential protective factor in this species. Observations on the natural passage of infection from sows to piglets emphasize the importance of carefully considering routes of immunization for inducing passive antibody. Unlike parenteral vaccines, incorporating oral vaccines in animal feed is less likely to encourage new variants of the K88 antigen. The antibody-mediated activity may lead to a reduced concentration of virulence plasmids in the microbial population, not only in the intestinal tracts of individual animals but in the environment as a whole. Although this has obvious relevance to the intensive rearing of farm animals, it may relate also to human beings, especially to those young children living in unhygienic conditions. The principle that plasmids may be permanently eliminated by the application of immunology has exciting implications for the control of other plasmid-mediated characteristics, such as antibiotic resistance.

Portnoy DA, Martinez RJ. Role of a plasmid in the pathogenicity of <u>Yersinia</u> species. Curr Top Microibol Immunol 1985;118:29-51

Powell DW. Enterotoxigenic diarrhea: mechanisms and prospects for therapy. Pharmacol Ther 1984;23(3):407-16

In the past decade, three evolving concepts have given hope for the development of effective antidiarrheal therapy. First has come the realization that ' diarrheal diseases, at least those causing significant morbidity and mortality, are diseases of water and electrolyte metabolism and not of altered _intestinal motility. Secondly, there is a growing understanding of the mechanisms of intestinal absorption and secretion. Lastly, there is new knowledge of how various agents promote intestinal water and electrolyte absorption or inhibit secretion. Because much of the impetus for studies in this field comes from the knowledge of the high mortality of diarrheal diseases in the Third World have been so important to and because the bacterial enterotoxins understanding of electrolyte transport in the gut, it is only fitting that concepts of mechanisms and prospects of therapy be discussed in a symposium on This article briefly reviews • enterotoxins and enterotoxigenic bacteria. mechanisms of intestinal secretion and discuss 4 promising categories of (modulators), calmodulin and calcium antisecretory drugs: neurotransmitters inhibitors, anti-inflammatory drugs, and opiates. Purification of bacterial exotoxins has allowed an understanding of how the agents stimulate cells. Coupled NaCl influx processes and chloride secretory mechanisms have been shown to be affected by exotoxin-stimulated increases in cyclic nucleotides, as well as by increases in intracellular calcium and arachidonic acid metabolites. Catecholamines, somatostatin, phenothiazines, nonsteroidal anti-inflammatory drugs, and opiates appear to be the most promising of the antisecretory drugs. While the mechanism of action of these agents remain to be determined, there is significant hope that effective antisecretory drugs will emerge in the near future.

Powers C see Janda JM

Prasad AS. The role of zinc in gastrointestinal and liver disease. Clin Gastroenterol 1983 Sep;12(3):713-41

Pratt EL see Darrow DC

Pressman J see Gertler S

Prichard RK. Interaction of host physiology and efficacy of antiparasitic drugs. Vet Parasitol 1985 Aug;18(2):103-10

Pringot J, Bodart P. Inflammatory diseases of the small bowel. Verh K Acad Geneeskd Belg 1984:46(5):309-49

Since the classic contributions published by Marshak and Lindner (1970). radiological understanding of inflammatory diseases of the small intestine has continued to progress. In this review, inflamatory diseases of the small intestine are considered as a whole and the concept of inflammatory bowel disease is considered in its broadest sense, including the diseases in which specific or nonspecific inflammation plays a determining role in the radiological appearance. Their radiological description is based on personal experience and recent literature: emphasis is placed on the differential diagnosis between Crohn's disease and other nosological entities. semiology of the intestinal form of Crohn's disease is specified. The spatial distribution of lesions (grading, assymetrical distribution on the mesenteric border) and the concept of the intermediate segment, whose importance in diagnosing the disorder is acknowledged, are also specified. The differential diagnosis includes certain tumors (lymphomas), carcinoid tumors, metastases, etc. as well as other inflammatory disorders. Aside from Crohn's disease, a specific or infectious acute ileitis, tuberculosis, segmental ischemia and radiation lesions are the ones most often encountered. Nonstenosing lesions are characterized by nodular-type thickening of the mucosa, aphthoid ulcers, or large ulcers and lymphoid hyperplasia, which is not constant.

Pritchard DI. Antigens of gastrointestinal nematodes. Trans R Soc Trop Med Hyg 1986;80(5):728-34

Nematodes, occupying the gastrointestinal tract of man, are known to release an array of chemicals into their environment. To combat effectively the potentially debilitating diseases, caused by infection with these organisms, attempts must be made to (a) define the parasite products chemically, (b) determine their ability to induce protective immunity (or to counter a protective immune response), and (c) establish their potential for the diagnosis of infection. Whilst it has become clear that "antigens" can be derived from within the parasite and from the turnover of external cuticular components (the term "ES" must include both), further work is necessary to establish the significance of these molecules for the survival of the parasite. In this context, a number of questions need to be answered in the near future. For example, how important is the hookworm protease to parasite nutrition? Can vaccines, using the genetically engineered and purified enzymes, generate Will the stichocyte secretions of Trichuris trichiura protective immunity? prove to be as immunogenic as those of Trichinella spiralis? (Surprisingly, little has been published with regard to the presence of enzymes in stichocyte secretions). Are gastrointestinal nematodes on the way out? (Modified author's abstract)

Proia RL see Eidels L

Protein and fat losses in infants with prolonged diarrhea. Nutr Rev 1982 Nov; 40(11):335-7

Quinn TC, Bender BS, Bartlett JG. New developments in infectious diarrhea. DM $1986\ Apr; 32(4):165-244$

"The past decade has produced exciting advances in our knowledge of infectious diarrhea. The list of potential enteric pathogens continues to grow with both the identification of new organisms, e.g. E. coll serotype 0157:H7, and the characterized microorganisms, e.g. previously recognition that gastrointestinal disease also cause avium-intracellulare. can Multiple investigations have led to a greater immunosuppressed patients. understanding of the epidemiology and clinical features of these illnesses. Application of modern biological techniques has yielded a number of diagnostic assays that are just now becoming clinically relevant. New drugs and treatment modalities for enteric infections have been developed and are now being Despite these advancements, however, evaluated in clinical trials. questions still remain unanswered. For example, the etiology of the debilitating diarrhea in AIDS patients in whom no known intestinal pathogen can mechanism of diarrhea The unknown. be identified remains Cryptosporidium, Isospora, and Giardia remains elusive. And last, will intestinal vaccines be effective in preventing diarrheal disease? Certainly, the next decade of research in enteric diseases should prove to be even more (Authors' abstract) interesting than the last."

Rabbani GH. Cholera. Clin Gastroenterol 1986 Jul;15(3):507-28

Rabbani GH. Drug treatment of infectious diarrhea in children. World Pediatr Child Care 1986;2(3):243-54

This paper highlights current knowledge and practices in the clinical management of pediatric diarrhea, specifically examining the role of drugs in the treatment of diarrheal diseases caused by viral, bacterial, and protozoal pathogens. Two types of drugs are used in the treatment of infectious diarrhea, namely, the antimicrobial and antisecretory agents. Diarrhea, caused by Vibrio cholerae, Shigellae, Entamoeba histolytica, Giardia lamblia, and Campylobacter jejuni, may benefit from the treatment with antimicrobial drugs. Antisecretory agents include loperamide, chlorpromazine, nicotinic acid, aspirin, indomethacin, and chloroquine. These interfere with intracellular biochemical mechanism(s) responsible for transport of water and electrolytes across the intestinal epithelium. Their clinical usefulness is not yet proven. (Modified author's abstract)

Rabbani GH see Greenough WB, III

Rahaman MM. Diarrhoea in Bangladesh: an overview of research conducted between 1962-1984. In: Tzipori S, Barnes G, Bishop R, Holmes I, Robins-Browne R, eds. Infectious diarrhoea in the young: strategies for control in humans and animals; proceedings of an International Seminar on Diarrhoeal Disease in South East Asia and the Western Pacific Region, Geelong, 10-15 Feb 1985. Amsterdam: Elsevier, 1985:69-72

This paper presents an overview of diarrhea-related research conducted in Bangladesh between 1962 and 1984 at the International Centre for Diarrhoeal Disease Research, Bangladesh (ICDDR,B). The development of oral rehydration therapy (ORT) is discussed with the use of the "Dhaka" solution containing physiologically balanced electrolytes. The decline in the use of the whole-cell cholera vaccine is reported. The classical vaccines were found to be useless for all practical purposes in studies carried out at the Matlab Field station of the ICDDR,B. Findings of research show that Shiyella species is a major cause of diarrhea, while noncholera Vibrio, enterotoxigenic

Escherichia coli, and rotavirus are considered as important causative agents of diarrhea. Dehydration due to rotavirus could be corrected by oral rehydration solution. Some inexplicable phenomena of classical and El Tor cholera in Bangladesh are discussed. Tetracycline-resistant Vibrio cholerae infection was found to be rare either in the laboratory or in nature. Search for new and effective vaccines against cholera and other diarrheal pathogens are continuing through vaccine trials at field level and experimentation at the laboratories of the ICDDR, B. The "Dhaka" solution for ORT is recommended for use in cholera patients.

Rahaman MM, Wahed MA. Direct nutrient loss in diarrhea. <u>In:</u> Chen LC, Scrimshaw NS, eds. Diarrhea and malnutrition: interactions, mechanisms, and interventions. New York: Plenum, 1983:155-60

This paper describes the use of alpha-1 antitrypsin as a marker protein derived from serum, to assess the diagnosis of protein-losing enteropathies and in the measurement of protein loss in diarrheal diseases. Hypotheses to explain the different pathogenesis of the serum protein losses observed in diarrhea due to Escherichia coli, Vibrio cholerae, Shigella, and rotavirus are offered.

Rahaman MM. Shigellosis: an old disease with new faces [editorial]. J Diarrhoeal Dis Res 1984 Dec;2(4):208

Rahaman MM see Briscoe J

Rahaman MM see Huq M1

Rahaman MM see Molla A

Rahaman MM see Molla AM

Ramband JC, Hautefeuille M, Ruskone A, Jacquenod P. Diarrhoea due to circulating agents. Clin Gastroenterol 1986 Jul;15(3):603-29

Rambaud J-C. Small intestinal lymphomas and alpha-chain disease. Clin Gastroenterol 1983 Sep;12(3):743-66

Ramia S. Transmission of viral infections by the water route: implications for developing countries. Rev Infect Dis 1985 Mar-Apr;7(2):180-8

"The "enteric" virus group comprises >100 different viruses. These viruses typically infect the cell lining of the alimentary canal and are discharged in very large numbers in the feces of infected persons. Contamination of water supplies by enteric viruses represents an important source of viral infection. communities, particularly in developing countries. depend sewage-polluted sources for their recreational and drinking water. conventional methods of sewage and water treatment have proved inefficient in the removal and inactivation of most enteric viruses, great concern has been raised over the impact of waterborne infection on the health of such communities. Current evidence implicating drinking and recreational water supplies in the transmission of nonbacterial gastroenteritis and hepatitis A virus and adenovirus infections is overwhelming. Waterborne transmission of other enteric viruses is also possible. Effective antiviral drugs are generally unavailable, and current vaccines can control only a limited number of viral infections; therefore, provision of uncontaminated water is a basic

requirement in raising the standard of health in affected communities." (Author's abstract)

Rampton DS, Hawkey CJ. Prostaglandins and ulcerative colitis. Gut 1984 Dec; 25(12):1399-413

Rampton DS see Hawkey CJ

Rams H, Rogers AI, Ghandur-Mnaymneh L. Collagenous colitis. Ann Intern Med 1987 Jan; 106(1): 108-13

"Two hypotheses have been proposed to explain the pathogenesis and cause of the increased subepithelial collagen deposition that occurs in patients with collagenous colitis, a rare disease of unknown cause. One hypothesis considers an inflammatory origin, and the other, a local abnormality of collagen synthesis. An analysis of clinical, endoscopic, and histologic findings from one of our patients and from previously published cases suggests that collagenous colitis is a form of inflammatory bowel disease characterized by localization of the initial injury to the superficial subepithelial zone and with subsequent fibrosis in that area. The data shows a spectrum of clinical and histologic changes that represent different stages in the evolution of the inflammatory process." (Authors' abstract)

Rand KH see Gotz VP

Randeria JD. Malnutrition and cancer. J Environ Pathol Toxicol Oncol 1985 Jul;5(6):103-14

Rao AV <u>see</u> Jenkins DJA

Rapid laboratory techniques for the diagnosis of viral infections. WHO Tech Rep Ser 1981;(661):1-60

Rask-Madsen J. Eicosanoids and their role in the pathogenesis of diarrhoeal diseases. Clin Gastroenterol 1986 Jul;15(3):545-66

"Eicosanoids are unsaturated fatty acid compounds derived from 20-carbon 'essential' fatty acids, the most important being arachidonate. Both cyclooxygenase and lipoxygenase products of arachidonate are abundant in the human gut, and their biological effects include modulation of fluid and electrolyte secretion, motor activity, mucosal blood flow, and cytoprotection, in addition to chemotaxis and immune response in inflammation. In health, these lipid mediators reinforce or synergize normal homeostatic mechanisms that could proceed in their absence. Receptors for control of intestinal secretion can be divided into two major classes, one of which triggers the production of cyclic AMP and another, which initiates phospholipid breakdown and arachidonate An intimate connection appears to exist between phospholipid metabolism; cytosolic Ca2+ levels, electrogenic anion secretion, Na+ pump rate, electroneutral Na+/H+ exchange activity, and intracellular pK. Ca2+-dependent secretagogues affect fluid and electrolyte transport in the small and the large bowel by increasing Ca_2^+ entry and Ca_2^+ mobilization through stimulation of eicosanoid formation, prostaglandins of the E type being the most important. Secretory diarrhoea may be thought of, therefore, as cellular Ca_2^+ intoxication. Uncontrolled formation of eicosanoids, perhaps with a changed spectrum of arachidonate metabolites, may not only be the source of diarrhoea

associated with mucosal inflammation, but may also be critical for cell proliferation resulting in abnormal cell differentiation, which seems to be the link between long-standing inflammatory bowel disease and the increased risk of colonic neoplasia. A better understanding of the pathophysiological role of eicosanoids in diarrhoeal disease has allowed reinterpretation of the rationale behind current therapy." (Author's abstract)

Rask-Madsen J, Bukhave K. Prostaglandins and chronic diarrhoea: clinical aspects. Scand J Gastroenterol 1979:14(suppl 53):73-8

This paper reviews the literature on the biological actions of prostaglandins (PG) with respect to the role of PGE and PGF in the pathogenesis of diarrhea. The review is largely concerned with physiological aspects which particularly relevant to the therapeutic application of PG inhibitors in the treatment of chronic and relapsing diarrhea. synthesized in virtually every organ including the small intestine. The E-type appears to elicit secretion of salt and water, while the F-type seems to promote intestinal motility -- at least in pathological conditions; but a physiological role has not yet been defined for either of them. Since the effect on transepithelial ion transport is similar to that produced by cholera toxin, the adenylate cyclase cyclic adenosine 3'.5'-monophosphate (cAMP) system has been implicated in the mechanism of PG action. Although PGE mimics the effects of cholera toxin and cAMP, not all experimental observations are explicable in terms of the theory which links PGs to cAMP. pharmacological concentration (10⁻⁶-10⁻⁴ M) activates intestinal cyclase, and the action of PGE on cAMP concentration is additive to that of On the other hand a dose-dependent relationship between cholera toxin. exogenous PGE2 and changes in ionic movements has been demonstrated in the short-circuited human jejunum in vitro during blockade of endogenous PG-biosynthesis with indomethacin (threshold response at $10^{-11} \, \mathrm{M}$; half maximal response at $10^{-9} \, \mathrm{M}$). Important therapeutic implications are suggested by the successful treatment of the diarrhea observed in the irradiation syndrome, specific food intolerance. syndrome, Crohn's irritable bowel colitis, and intestinal pseudo-obstruction with PG-synthetase inhibitors, such as indomethacin, aspirin and related drugs, or nutmeg. PGs also appear to be involved in the pathogenesis of diarrhea in pancreatic cholera, the carcinoid syndrome, and medullary carcinoma of the thyroid. Effects have been made in assaying primary PG levels in peripheral plasma for clinical use. The demonstration of a marked in vitro production during the sampling procedure seems to explain the conflicting data in accordance with the known ability of the lungs to remove the majority of PGs from circulation. However, local PG-release is reflected in the intestinal secretions which may be sampled for analysis without laborious precautions.

Rask-Madsen J. The role of eicosanoids in the gastrointestinal tract. Scand J Gastroenterol 1987;22(suppl 127):7-19

"Exploring the role of eicosanoids in the gastrointestinal tract entails fundamental problems of methodology and interpretation. Most important are the difficulties inherent in the choice of an experimental design which prevents non-specific stimulation of eicosanoid formation, because any perturbation of cell membranes will initiate eicosanoid synthesis. In addition to cyclic nucleotides, prostaglandins may serve as intracellular mediators for the stimulus of secretion coupling via intracellular free calcium in the gastrointestinal epithelial cells. By contrast, the effects of

supraphysiological doses of prostaglandins parallel those AMP-dependent secretagogues such as VIP, which increases calcium through activation of the mucosal adenylate cyclase. The question of whether patients develop gastric or duodenal ulcers as a result of a prostaglandin deficiency remains open. The synthetic prostaglandin analogues available commercially for anti-ulcer therapy appear to be unable to accelerate the healing of peptic ulcers unless hey are administered in antisecretory doses, and are unlikely to have a substantial effect on patients with bleeding from ulcerative lesions in Prostaglandins of the E type mediate, at least the gastro-duodenal mucosa. partly, the diarrhoea associated with a large number of clinical conditions and various pharmacological agents. Several types of secretory diarrhoea respond to drugs that inhibit prostaglandin biosynthesis. Whether eicosanoids are mediators, or merely epiphenomena, of inflammation in ulcerative colitis and Crohn's disease remains unclear. Improved knowledge of their functional role of eicosanoids has nevertheless allowed a reinterpretation of the rationale behind current therapy. Uncontrolled formation of eicosanoids may not only be the source of diarrhoea in colonic inflammation, but may also be critical for cell proliferation and the development of dysplasia in long-standing disease." (Author's abstract)

Ravdin JI, Guerrant RL. A review of the parasite cellular mechanisms involved in the pathogenesis of amebiasis. Rev Infect Dis 1982 Nov-Dec;4(6):1185-1207

"Amebiasis, a significant cause of morbidity and mortality on a global scale, is caused by invasion of the colonic mucosa by the cytolytic protozoan parasite Entamoeba histolytica. A review of the investigations into the mechanisms by which the parasite causes invasive disease is presented, including the relevant history, epidemiology, and pathology of amebiasis, the biology of the parasite, in vivo and in vitro studies of the pathogenesis of disease, the host immune responses, and possibilities for prevention and biologic control. New insights into the cell biology of \underline{E} . histolytica, especially its adherence and cytolytic properties, offer several new approaches to biologic control of this invasive, destructive parasitic infection." (Authors' abstract)

Ravich WJ, Bayless TM. Carbohydrate absorption and malabsorption. Clin Gastroenterol 1983 May:12(2):335-56

Read NW. Diarrhee motrice. Clin Gastroenterol 1986 Jul;15(3):657-86

"The gut is a long convoluted tube, in which food is processed and nutrients, salt and water are absorbed. The degree of absorption depends to a large extent on the degree of contact between the luminal contents and the absorptive epithelium. Motor activity can influence the degree of absorption because it regulates the degree of contact with the epithelium and it may also induce secretion by a reflex mechanism. Many factors that induce diarrhea are associated with "abnormal" and highly propagative forms of motor activity that can clear material through the yut, allowing insufficient epithelial contact for absorption. These propulsive motor patterns may be provoked by distension of the yut with fluid, but they can also occur in response to diarrheagenic factors when there is minimal distension. Patients who complain of increased frequency, urgency and incontinence but pass normal stool volumes often have an abnormality in the motor activity of the anorectum. Thus, the generation of abnormal or propagated forms of motor activity must be regarded as an important component of the pathogenesis of all types of diarrhea, and an increased stool volume can be regarded as the end result of a vicious spiral that may start

with a primary abnormality in either motor activity or epithelial transport." (Author's abstract)

Read NW. The relationships between colonic motility and transport. Scand J Gastroenterol 1984;19(suppl 93):35-42

"The salvage of unabsorbed nutrients, salt and water in the colon requires the integration of functions of bacterial digestion and epithelial transport with motor activity. However, the motor and transport functions of the colon are poorly understood owing to the inaccessibility of that organ. This paper discusses some of the ways in which motor activity may influence colonic absorption and vice versa, using examples taken from studies carried out in the small intestine. In particular, the effect of factors such as convection, transit time, reflex secretion and blood flow are discussed." (Author's abstract)

Read NW. Speculations on the role of motility in the pathogenesis and treatment of diarrhoea. Scand J Gastroenterol 1983;18(suppl 84):45-63

"Comparatively little attention has been given to the role of gastrointestinal motility in the pathogenesis and treatment of diarrhoea. Here the relationship between motor activity and absorption in the small intestine has been assessed, as has that between small intestinal secretion and motility, and between colonic salvage and motility. It is suggested that diarrhoea should be considered as a disturbance of intestinal flow, involving disturbances in both motility and transport. The role of antimotility agents (particularly opiate-like agents such as loperamide) is reviewed. The most successful antidiarrhoeal agents are those that combine a reversal of epithelial secretion with an action on motility." (Author's abstract)

Recent advances in cholera research: Memorandum from a WHO meeting. Bull WHO 1985;63(5):841-9

Vibrio cholerae Ol is a major cause of diarrheal disease in large parts of Asia and Africa. The seventh pandemic of cholera is still continuing (92 countries have so far been affected), and other organisms related to V. cholerae 01 are being reported with increasing frequency as the cause of diarrheal outbreaks as well as endemic diarrhea. Recent research has considerably advanced the understanding of how cholera is transmitted, the mechanisms by which V. cholerae 01 causes disease, and the functioning of the local intestinal immune response by which individuals can be protected from infection. One result of these advances has been the development of experimental cholera vaccines, derived from nonliving and attenuated live strains, which promise to become a new tool for the control of cholera. These matters, which were reviewed by the WHO Scientific Working Group on Bacterial Enteric Infections at a meeting in Geneva, Switzerland, in September 1984, are described together recommendations for future research.

Reddy V. Interaction between malnutrition and diarrhoea with particular reference to pediatric practice. Indian J Gastroenterol 1985 Jul;4(3):183-6

Protein-energy malnutrition and diarrheal diseases are major health problems in developing countries contributing to high morbidity and mortality in children. Both these conditions basically arise from poverty, and the mutual interaction between the two compounds the situation. Repeated episodes of diarrhea

aggravate malnutrition and, conversely, malnutrition worsens diarrhea and increases case fatality. Effective intervention must necessarily deal with Symptomatic response to an infection and the both problems simultaneously. disease outcome depends on the balance between two opposing forces of pathogen virulence and host defense. Nutrition is one of the important factors that can modify the resistance and tip the balance towards morbidity and mortality. Promotion of breast feeding and proper weaning practices are critical for infant nutrition. Adequate feeding during illness is important to maintain good nutrition and calls for a change in the traditional dietary practices. Supplementary feeding programs have been implemented on a wide scale to meet the existing calorie gap in the diet of preschool children. Nutrition services should be integrated with other child care services, like immunization and medical care, for obtaining optimum benefits. Oral rehydration therapy is effective in the treatment of diarrhea and dehydration, and this simple and inexpensive measure can bring down diarrheal mortality considerably. Prevention of diarrheal diseases, however, requires environmental approaches. Personal hygiene, improvement in water supply and sanitation are essential to interrupt the transmission of diseases. Ultimately, malnutrition and diarrhea may be effectively controlled only through overall socioeconomic development.

Reisman T, Morton RE, Rogers AI. Gastroenterology: a hypothetical case of chronic diarrhea incorporating a management self-test. Postgrad Med 1976 Feb;59(2):203-10

Chronic diarrhea may be a manifestation of any of the several local and systemic disorders. A thorough history and physical examination of a patient with this complaint is mandatory. Appropriate laboratory and radiological studies should be obtained after the attending physician has outlined a rational approach to the problem. This paper reports a hypothetical case of chronic diarrhea in which questions concerning management are interposed at points in the work up where they would arise in practice. The questions and answers should enable the physicians to formulate a rational approach to the diagnosis and treatment of chronic diarrhea.

Reller LB see Blaser MI

Reller LB see Janoff EN

Rennels MB, Levine MM. Classical bacterial diarrhea: perspectives and update — Salmonella, Shigella, Escherichia coli, Aeromonas and Plesiomonas. Pediatr Infect Dis 1986 Jan; 5(1): 591-100

The primary goal of this discussion or bacterial diarrhea is to highlight some of the major advances in knowledge and consider the relative importance of the various bacteria from a global perspective. The bacteria, Salmonelia, Shigella, Escherichia coli, Aeromonas, and Plesiomonas, are covered in this review. Emphasis is, however, placed on current understanding of mechanisms of pathogenesis as they determine the clinical manifestations of infection. A major portion of the review concentrates on the diarrheagenic classes of Escherichia coli, because this area has received intensive research attention. Impressive advancements in the understanding of the mechanisms of diarrhea and of the epidemiologic importance of these "classical" bacterial enteropathogens have been accomplished, but many areas need further exploration. The recent development of gene probes and the enzyme-linked immunosorbent assay for the identification of enteropathogenic \underline{E} , \underline{coli} , enterotoxigenic \underline{E} , \underline{coli} and

enteroinvasive \underline{E} . \underline{coli} will enhance immeasurably the ability to carry out large-scale epidemiologic studies which are still needed to clarify the global importance of these pathogens in infantile diarrhea. For some of these organisms, pathogenic mechanisms are not incompletely understood, and the role of antibiotics is not well established. The goal of prevention through vaccination is coming nearer through the delineation of virulence factors, immunity and the construction of attenuated strains through deoxyribonucleic acid recombination technology. These advances should not, however, detract attention from the primary reason that these bacteria continue to be a major cause of childhood morbidity and mortality in the developing world, which is again due to the lack of adequate sanitation. Efforts to provide clean water, safe waste disposal, and hygiene education need to proceed in conjunction with development of vaccines. (Modified authors' abstract)

Rennels MB see Levine MM

Research on improving infant feeding practices to prevent diarrhoea or reduce its severity: Memorandum from a JHU/WHO meeting. Bull WHO 1989;67(1):27-33

"In developing countries the highest incidence of diarrhoea occurs among infants who are given fluids or foods to supplement or replace breast-feeding, and numerous studies have been conducted to examine the relationship between feeding practices during the weaning period and the risk of diarrhoea and malnutrition. This Memorandum summarizes current knowledge about the potential impact of improved infant feeding practices on diarrhoeal morbidity and mortality and describes experiences gained with interventions to promote improved practices. Further research activities to examine the role of improved infant feeding practices in the control of diarrhoeal diseases and to identify more effective approaches to the promotion of such practices in the context of a public health programme are also proposed. Finally, methodological issues concerning the design, implementation, and analysis of intervention studies are reviewed." (Authors' abstract)

Reves RR see DuPont HL

Rikkers LF see Thompson JS

Riviere JE, Boosinger TR, Everson RJ. Inorganic arsenic toxicosis in cattle. Mod Vet Pract 1981 Mar;62(3):209-11

"In 4 occurrences of arsenic poisoning in cattle, the principal clinical sign was acute hemorrhagic diarrhea attributable to hemorrhagic gastroenteritis. Arsenic concentrations in the liver, kidney and rumen contents varied. In one occurrence, arsenic in the hair of affected survivors was assayed at 0.8-3.40 ppm vs 0.09-0.10 ppm in randomly selected control samples of hair. Sudden death was the only clinical sign in another occurrence in which gastric contents contained arsenic at 671 ppm. In another occurrence, arsenic poisoning caused lesions similar to those of salmonellosis." (Authors' abstract)

Robertson RP see Metz SA

Robins-Browne R see Levine MM

Robins-Browne RM. Traditional enteropathogenic <u>Escherichia coli</u> of infantile diarrhea. Rev Infect Dis 1987 Jan-Feb; 9(1):28-53

"First described in 1885, Escherichia coli gradually emerged as a cause of infantile diarrhea. Currently, four pathogenic categories of diarrheagenic E. recognized: enteropathogenic (EPEC), enterotoxigenic (ETEC), ve (EIEC), and enterohemorrhagic (EHEC). Of these, ETEC and EPEC enteroinvasive (EIEC), and enterohemorrhagic (EHEC). are important causes of pediatric diarrhea, especially in developing countries. are characterized by their production of well-defined characteristics, EPEC strains have been identified by serotyping only; recent laboratory and clinical investigations have shown that these strains constitute a distinctive group of pathogenic microorganisms displaying characteristic producing pathognomonic cultured epithelial cells and adherence to histopathologic changes in experimentally infected animals and naturally infected children. The pathogenicity of most strains of EPEC is associated with the carriage of a 55- to 70-megadalton plasmid that specifies the production of an adherence factor. EPEC strains may also owe pathogenicity to the production of at least one enterotoxin, possibly a Shiga-like cytotoxin." (Author's abstract)

Rocha MP, Burrichter PJ, Blodgett RC. Effect of chrysotherapy on the lower gastrointestinal tract: a review. Semin Arthritis Rheum 1987 May;16(4):294-9

Rodrick GE, Hood MA, Blake NJ. Human <u>Vibrio</u> gastroenteritis. Med Clin North Am 1982 May;66 (3):665-73

Many gaps concerning the epidemiology of Vibrio infections exist. seawater and raw or improperly cooked seafood serve as the principal vehicles for the transmission of non-Vibrio cholerae 01, \underline{V} . parahaemolyticus, \underline{V} . vulnificus, \underline{V} . alginolyticus, \underline{V} . metschnikovii, and group F (EF6) vibrios. Seafood, properly cooked, prevents infection. More specifically, consumption of seafood is safe if the food is cooked at temperatures adequate to sterilize them and if cross-contamination after cooking is prevented. addition, such cooked seafood should be kept at either temperatures too cold (less than 4°C) or too hot (greater than 60°C) for multiplication of vibrios. Traditional methods of cooking are inadequate for the inactivation of many evidence suggests that people Recent epidemiologic pre-existing liver disease (cirrhosis, hepatitis, or leukemia) should avoid eating raw or improperly cooked seafood because of the high risk of Vibrio infections. In addition, the risk of <u>Vibrio</u> infection may be minimized by not eating raw shellfish collected during the warm summer and fall months. Cases involving non-V. cholerae 01, V. parahaemolyticus, V. vulnificus, and V. alginolyticus have increased sharply during the past 10 years. Increased awareness of epidemiologists, microbiologists, and physicians concerning the existence of these potentially pathogenic vibrios and their suspected settings will lead to a better understanding of the epidemiology and diagnosis of Vibrio infections in the future. (Modified authors' abstract)

Rogers AI see Rams H

Rogers AI see Reisman T

Rogers MF. AIDS in children: a review of the clinical, epidemiologic and public health aspects. Pediatr Infect Dis 1985 May-Jun;4(3):230-6

Rohde C see Cvjetanovic B

Ronde J, Northrup RS. Diarrhoea: a nutritional disease. J Indian Med Assoc 1987 Jul;85(7):196-202

Rohde JE. Selective primary health care: strategies for control of disease in the developing world. XV. Acute diarrhea. Rev Infect Dis 1984 Nov-Dec; 6(6): 840-54

"Diarrhea claims the lives of at least five million children per year and is a major contributor to malnutrition in developing countries. A variety of infectious agents cause diarrhea through several mathogenic mechanisms. Bacteria such as Shigella can directly invade the intestinal mucosa, and those similar to <u>Vibrio cholerae</u> or enterotoxigenic <u>Escherichia coli</u> can produce toxins that alter cellular absorption and secretions. Rotavirus appears to cause 30%-40% of diarrhea in toddlers. cause 30%-40% of diarrhea in toddlers. Diarrhea frequently accompanies parasitic infestation and infections in other parts of the body. Several methods, including improvement of water supplies and sanitation administration of vaccines and antibiotics, may be useful in preventing mortality and morbidity due to diarrhea. Oral rehydration to replace fluids and electrolytes, accompanied by attention to early feeding and encouragement of breast feeding, is the most effective and economical intervention currently available." (Author's abstract)

Rohde JE, Cash RA, Guerrant RL, Mahalanabis D, Molla AM, Valyasevi A. Therapeutic interventions in diarrhea. <u>In</u>: Chen LC, Scrimshaw NS, eds. Diarrhea and malnutrition: interactions, mechanisms, and interventions. New York: Plenum, 1983:287-95

This work is a concise review on therapeutic diarrhea management, laying special emphasis on the immediate need for fluid and electrolyte replacement, preferably with oral rehydration solutions (ORS). The paper highlights: ORS containing 90 mmols/l of Na is effective for all diarrheas, including cholera, providing infants receive plain water, breastmilk, or diluted cow's milk; malnourished children require extra potassium. Since potassium loss in infantile diarrhea is high, higher potassium concentrations are advocated by various studies; while acidosis associated with bicarbonate loss in diarrhea to some extent can be corrected through normal renal and respiratory compensatory mechanisms over several days, it is preferable to provide some base in ORS. Acetate and citrate are acceptable alternatives to bicarbonate, since they are easier to incorporate in ORS packets or tables; two percent of glucose or 4% of sucrose is required for optimal sodium and water absorption, but such sugars provide few calories. Hence, a rice-based electrolyte solution is extremely promising; feeding should not be stopped during diarrhea and particularly during convalescence, dietary intake should be increased, through higher caloric foods and greater feeding frequency; antibiotics are only indicated for dysentery and certain other diarrheas with clear bacterial etiologies. Indiscriminate drug use can be harmful. Even among recognized pathogens, such as V. cholerae, Campylobacter, Shigella, and Salmonella, there may be special clinical circumstances where antibiotic use cannot be recommended.

Rolfe RD. Diagnosis of <u>Clostridium difficile</u>-associated intestinal disease. CRC Crit Rev Clin Lab Sci 1986;24(3):235-61

"Toxigenic <u>Clostridium difficile</u> is the major cause of antimicrobial agent-associated pseudomembranous colitis and is the etiological agent of approximately 30% of cases of nonspecific colitis and diarrhea (without colitis) induced by antimicrobial agents. In addition, <u>C. difficile</u> has been implicated in certain intestinal diseases not related to prior antimicrobial administration. <u>C. difficile</u> has been reported to be one of the most common

enteropathogens isolated from stool specimens submitted to hospital laboratories. Thus, diagnosis of \underline{C} . $\underline{difficile}$ -associated intestinal disease should not be routinely performed in diagnostic clinical laboratories. The diagnosis of \underline{C} . $\underline{difficile}$ -associated intestinal disease relies on the demonstration of either the organism or the toxin(s) in stool specimens or antibody response in serum to the toxin(s). Several selective medium are available for the recovery of \underline{C} . $\underline{difficile}$ from stool specimens. The toxin(s) of \underline{C} . $\underline{difficile}$ can be demonstrated using a variety of techniques, including biological assays as well as immunological assays. This article will review the techniques currently available to aid in the diagnosis of \underline{C} . $\underline{difficile}$ -associated intestinal disease." (Author's abstract)

Rollin RE <u>see</u> Fettman MJ

Rolston KV, Fainstein V. Cryptosporidiosis. Eur J Clin Microbiol 1986 Apr;5 (2):135-7

Romanowska E see Simmons DAR

Rosenberg I see Gitler C

Rosenberg IH, Solomons NW, Schneider RE. Malabsorption associated with diarrhea and intestinal infections. Am J Clin Nutr 1977 Aug;30(8):1248-53

An episode of diarrhea causes weight loss and temporary cessation of growth in infants and children. Diarrhea is accompanied by malabsorption of sugars, nitrogen, fats, and micronutrients. This review article examines the regularities with which diarrheal diseases of different etiologies produce malabsorption, and the mechanisms whereby infectious diarrhea may produce a transient, but significant malabsorption syndrome. Also discussed are possible nutritional implications of malabsorption in diarrhea. The nutritional costs of malabsorption may pose a major threat if diarrhea becomes chronic or recurrent. The hydrogen breath test for carbohydrate malabsorption does not require intubation or blood drawing. This test can be used on children to help clarify the importance of carbohydrate intolerance in the duration and perpetuation of acute diarrhea and intestinal bacterial overgrowth.

Rosenberg IH, Bengoa JM, Sitrin MD. Nutritional aspects of inflammatory bowel disease. Annu Rev Nutr 1985;5:463-84

Rossi TM. Intractable diarrhea of infancy. J Pediatr Gastroenterol Nutr 1983;2(suppl 1):S315-20

Rossi TM, Lebenthal E. Pathogenic mechanisms of protracted diarrhea. Adv Pediatr 1983;30:595-633

Rossi TM see Lebenthal E

Rowe B. The role of Escherichia coli in gastroenteritis. Clin Gastroenterol 1979 Sep;8(3):625-44

Rowe B see Gross RJ

Rowland HAK. The pathogenesis of diarrhoea. Trans R Soc Trop Med Hyg 1978;72 (3):289-302

Rowland MGM, Rowland SGJG. Growth faltering in diarrhoea. <u>In:</u> Taylor TG, Jenkins NK, eds. Proceedings of the XIII International Congress of Nutrition, Brighton, 18-23 Aug 1985. London: Libbey, 1986:115-9

Diarrhea is the most important nondietary cause of growth faltering in young children in developing countries. However, there may exist variations in the amount of diarrhea experienced by individuals, even of the same age and within the same community. Not all children who have an attack of diarrhea suffer growth impairment as a consequence. This article explores some possibilities for these variations and attempts to identify the more specific characteristics of subjects and disease episodes involved in the impact of diarrhea on growth. The main strength of interaction between diarrheal illness and growth might occur before 3 years of age. In general, boys tend to suffer higher attack rates of diarrhea than girls, for reasons largely unknown. In all studies, particularly among the younger children in underprivileged societies, the frequency and lethality of diarrhea is higher in bottle-fed, mixed-fed, or weaned children than in exclusively breast-fed children. Little attention has been given so far to the extent of influence of baseline nutritional status on the impact of diarrhea on growth. The evidence in this regard is far from conclusive. A study of Gambian children found that 63% of the diarrheal episodes were associated with weight faltering, but these variations could also have been associated with different feeding patterns. A study in rural Bangladesh showed that it was the prolonged attacks lasting more than 10 days which appeared to have the greatest effect on growth. Studies on the diarrhea-growth relationship should take into account the distinction between early transient weight loss and the less-common deficit that may be detectable months later. That an effect can be discerned in some children long after the original illness could be due to chronic dietary constrains, persisting pathology impairing gut function, or both. Community studies are in general based on fairly frequent questionnaire surveys and tend to document diarrheal morbidity fairly comprehensively. Most hospital studies are carried out on -children, selected for admission on the basis of more or less severe dehydration, or severe concomitant malnutrition. This aspect should be considered when trying to relate various findings and particularly when trying to determine the basis for interventions or strategies aimed at reducing malnutrition during diarrhea.

Rowland MGM. The weanling's dilemma: are we making progress? Acta Paediatr Scand 1986;(suppl 323):33-42

"In many developing countries the weanling child (the breastfed child who is regularly receiving additional food) still suffers a high level of morbidity and mortality from diarrhoeal disease. The initiation of weaning is a critical event. No clear strategy exists for substantially enhancing the breast milk output of demand feeding mothers in underprivileged communities. Remarkably little progress has been made in our ability to advise mothers, either on a collective or an individual basis, as to when they should supplement the diet of their breastfed offspring, one continuing problem being the failure to adopt appropriate growth standards for infants. Furthermore there has been little attempt to improve traditional weaning foods in terms of consistency, shelf life and bioavailability of nutrients. Increasing insights into the normal growth pattern of breastfed infants and knowledge of localised appropriate traditional food technology remain grossly underexploited." (Author's abstract)

Rowland SGJG see Rowland MGM

Rowley D, La Brooy J. Intestinal immune responses in relation to diarrhoeal diseases. J Diarrhoeal Dis Res 1986 Mar:4(1):1-9

Although there are no wholly satisfactory vaccines against enteric infections. there is still evidence from a good number of vaccine studies, of a measure of immunity following their administration. As the practical relevance of intestinal immunity in diarrheal disease relates to the possibility of developing effective immunization programs for the control of gut infections, this review focuses on insights into the functioning of the immune system particularly relevant to this goal. There is positive evidence from recent work that antigens, introduced into the gut, effect their immune stimulation after entering Peyer's patches. The role of Peyer's patches as a site where antigen is sampled has been discussed. The consequence of an antigenic stimulus to the intestine is IgA production. The immune response generated in the intestine spans T-lymphocytes and at least indirectly, macrophages. primary stimulus to the intestinal immune system is best delivered by oral immunization. The immune response pattern generated by an immune stimulus at the gut differs from that which occurs when antigen is injected and reaches the The normal response to orally immune system first by a different route. administered antigen is the development of a local IgA response and the development of systemic or oral tolerance. The effector pathways that may play a role in immunity to gut infections include those that may act within the lumen of the gut such as those dependent on IgA which have well been documented, and others which may act in the gut wall against invading organisms, some of which have been only superficially examined. Approach to the generation of useful vaccines against enteric infections has changed in recent years directly as a result of increased knowledge on the pathways by which local immunity is generated and the effector pathways against pathogens. With the powerful tools of biotechnology now available, effective vaccines against many of the pathogens which cause disease at secretory surfaces, such as the intestine, lung, and genito-urinary tract, may soon be tested.

Rowley D see La Brooy J

Ruiz-Palacios GM see Walker RI

Ruiz-Palacious GM. Norfloxacin in the treatment of bacterial enteric infections. Scand J Infect Dis 1986;(suppl 48):55-63

"Diarrhoeal diseases are a major cause of illness and death among infants and young children, especially in developing countries. They are also the principal cause of illness in tourists who travel to these countries. Overall, the main causes of diarrhoea are bacterial. Campylobacter, enteropathogenic E. coli, Shigella and enterotoxigenic E. coli are, in this order, the most common bacterial enteropathogens isolated in developing countries, and Campylobacter and Shigella in developed countries. Even though the cornerstone of the treatment of diarrhoea is oral rehydration, especially in children, the better knowledge of the pathogens involved and the mechanisms by which these organisms produce diarrhoea has brought the reconsideration of antibiotic use, which just a few years ago was considered unnecessary except for Shigella. The increasing number of Shigella and Salmonella typhi resistant to ampicillin and co-trimoxazole and the recognition of Campylobacter, an enteropathogen normally resistant to these drugs as a major cause of diarrhoea, makes it necessary to investigate new agents against these three enteropathogens. Antimicrobial susceptibility studies done in Mexico and other parts of the world have shown

that norfloxacin, a new quinolone, is very active against multi-resistant S. typhi and Shigella and against Campylobacter, with MIC values of approximately 0.8 mg/l. Furthermore, it achieves high concentrations in faeces after oral administration. Clinical studies of norfloxacin used in inflammatory diarrhoea and in prophylaxis of traveller's diarrhoea, have demonstrated it to be an effective and well-tolerated drug. Norfloxacin has great potential for the treatment of bacterial enteric infections reflected by its broad in vitro activity as well as its effectiveness in initial clinical trials." (Author's abstract)

Ruskone A see Ramband JC

Russell RI. Intestinal adaptation to an elemental diet. Proc Nutr Soc 1985 Feb;44(1):87-93

Ruzicka LT, Hansluwka H. Mortality transition in South and East Asia: technology confronts poverty. Pop Dev Rev 1982 Sep:8(3):567-88

This paper presents an overview of the crucial issues revolving around attempts to decrease mortality in South and East Asia from 1950 to 1980, and the resulting trends. The review begins with the fact that in all low-income countries in this area, deaths were reduced, both directly through health care interventions and indirectly through improvements in living standards. However, it notes that mortality levels and the pace of mortality decline varied greatly from country to country and recent evidence suggests that in the 1970s. in some of the countries, the earlier promising trend has slowed, while in others stagnation or even reversal may have occurred. The authors suggest that mortality decline in the past was produced both by economic and social improvements, and by diffusion of public health measures and preventive medicine. In individual countries, the roles of these two factors are difficult to separate, due to the complexity of the interrelationship. In most cases, the statistical data needed to even attempt such an evaluation are sadly inadequate or lacking. The conclusion from various studies and evaluations of experimental programs appears to be that both public health programs and improved living standards are important, and that they are likely to be most effective in combination. It is suggested that the main questions for the 1980s are not of the either/or type, but concern the type of programs and development that, in a given country's specific circumstances, will have the greatest effect on morbidity and mortality. Moreover, within the framework of the primary health care package recommended by the WHO, program types and services will vary from country to country. Also, in some countries the emphasis of development programs should be on reducing existing inequalities in opportunities for education and employment. The indirect impact of such programs on morbidity and mortality, in the long run, may be more important especially for the low-income strata. The shortcomings of public health services, their inadequate distribution and the misallocation of limited health resources in many countries have been pointed out by many publications of the It must be realized, the authors maintain, that lack of basic health care is not a health problem, but rather a handicap in the fight against health problems. At the root of many countries' health problems is the poverty of a large proportion of their people, and the accompanying deficiencies in nutrition, housing, sanitation and literacy - the conditions that must be redressed along with improvements in health services. Action in one area alone may alleviate some manifestations of the health problem, but will not, in the long run, provide a significant and permanent solution.

Ryan ME, Olsen WA. A diagnostic approach to malabsorption syndromes: a pathophysiological approach. Clin Gastroenterol 1983 May;12(2):533-50

Rybalko SL see Zhalko-Titarenko VP

Sack DA. Treatment of acute diarrhoea with oral rehydration solution. Drugs 1982 Jan-Feb;23(1-2):150-7

This paper reviews some of the principles of oral rehydration, and outlines practical methods for using oral rehydration solution (ORS). Though based on experiences at the International Centre for Diarrhoeal Disease Research, principles applicable. Ву are generally Bangladesh, the glucose-facilitated absorption of sodium and water, ORS is capable rehydrating patients with acute watery diarrhea of different etiologies. Even for mechanism during diarrhea-induced intestinal hypersecretion, this absorption remains intact. ORS does not stop the secretion nor slows down the It replaces the needed volume, base and potassium, thus eliminating metabolic consequences of diarrhea. All etiologies, age groups, and severities of diarrheal diseases respond to ORS therapy, unless there exists very severe diarrhea, shock, and dehydration, requiring initial intravenous (i.v.) therapy, followed by ORS. Antibiotics are useful against specific diseases, but neither these nor antimotility drugs should be used routinely. Being a simple, inexpensive, convenient diarrhea therapy, ORS is particularly relevant to developing countries. However, its use also should be encouraged in developed countries, where ORS can largely replace i.v. hydration in diarrheal diseases.

Sack D see Svennerholm A-M

Sack DA see Ahmed ZU

Sack RB. Antimicrobial prophylaxis of travelers' diarrhea: a selected summary. Rev Infect Dis 1986 May-Jun;8(suppl 2):S160-6

Prophylaxis of travelers' diarrhea with antimicrobial agents began more than a decade before the major causative agents of the disease were identified. Clinical observations had suggested that the syndrome was most likely caused by enteropathogenic bacteria, and on that basis, most of the antimicrobial agents This paper reviews the tested for prophylactic efficacy were nonabsorbable. published controlled studies of the prophylaxis of travelers' diarrhea in which the following drugs have been used: neomycin, nonabsorbable sulfonamides, streptotriad, doxycycline, erythromycin, and mecillinam. These studies have shown that antimicrobial prophylaxis can be highly effective in preventing episodes of travelers' diarrhea. The protection, however, lasts only as long as the drugs are being taken; there is no evidence that subclinical infections None of the data suggest that occur while the drug is being taken. antimicrobial prophylaxis increases of infection the probability In these studies, few subjects drug-resistant enteric pathogens. experienced adverse drug reactions; however, the number of subjects has too small to allow determination of accurate incidence data. Antimicrobial prophylaxis for travelers' diarrhea can be effectively and safely used on an individualized basis for persons traveling to areas of high risk. author's abstract)

Sack RB. Antimicrobial prophylaxis of travellers' diarrhoea: a summary of

studies using doxycycline or trimethoprim and sulfamethoxazole. Scand J Gastroenterol 1983:18(suppl 84):111-7

"Antimicrobial therapy of travellers' diarrhoea with doxycycline or combination of trimethoprim and sulfamethoxazole is effective only while the drugs are being taken. There is no evidence that sub-clinical infections or exposures lead to the development of immunity while the drugs are being taken. When the drugs are withdrawn, an increase in diarrhoea attack rate occurs in those persons who were previously taking the drug. Therefore, the drugs are only effective for short-term use and would not be indicated in persons travelling for long periods of time. Both drugs can be taken once a day. are therefore convenient to use. There are adverse side-effects with these drugs: the possibility of developing a skin rash with TMP-SMX or photosensitivity reaction with doxycycline must be balanced against the protection afforded. Although both drugs have an effect on normal enteric flora, there is no evidence to suggest that this renders the individual more susceptible to other enteric pathogens." (Author's abstract)

Sack RB. Escherichia coli and acute diarrheal disease. Ann Intern Med 1981 Jan:94(1):129-30

Sack RB. Human diarrheal disease caused by enterotoxigenic <u>Escherichia</u> coli. Annu Rev Microbiol 1975:29:333-53

Saif LJ, Smith KL. Enteric viral infections of calves and passive immunity. J Dairy Sci 1985 Jan;68(1):206-28

At least 8 viruses have been identified. 4 within the last 5 years, that produce diarrhea and pathological intestinal lesions in experimentally inoculated calves. Coronavirus and rotavirus are frequently associated with the neonatal calf diarrhea syndrome, but the etiologic role of the newly identified viruses is undefined. All diarrheal viruses replicate within small-intestinal epithelial cells, resulting in variable degrees of villus atrophy. Immunity against these viral infections must, therefore, be directed toward protection of the susceptible intestinal epithelial cells. Since most of these viral infections occur in calves aged under 3 weeks, passive lactogenic immunity within the gut lumen plays an important role in protection. This report reviews methods of boosting rotavirus antibody responses in bovine mammary secretions and analyses passive and active immunity in calves supplemented with colostrum and challenged by rotavirus. Results indicate that rotavirus immunoglobulin G1 antibodies in colostrum and milk are elevated after intramuscular and intramammary vaccination of pregnant cows with an Ohio Agricultural Research and Development Center rotavirus vaccine, but not after intramuscular immunization with a commercial rota-coronavirus vaccine. Feeding colostrum from intramuscular plus intramammary immunized cows to newborn calves challenged by rotavirus prevent the development of diarrhea and shedding of rotavirus. (Modified authors' abstract)

Saif LJ see Horzinek MC

Sakai S, Ito T. [Campylobacter infections]. Nippon Saikingaku Zasshi 1985 May;40(3):563-80

Samadi AR, Islam MR, Aziz KMS. ICDDR, B model for treatment of diarrhoeal diseases. Dhaka: International Centre for Diarrhoeal Disease Research.

Bangladesh, Jul 1982. 12 p. ([CDDR,B special publication no. 19)

Diarrheal diseases are a major health problem in developing countries and so is lack of resources and trained personnel to cope with health problems. This paper presents the model for treatment of diarrheal diseases followed at the International Centre for Diarrhoeal Disease Research, Bangladesh, with particular reference to its organization, administration, staffing pattern, procedures for screening patients and methods of treatment. This includes the procedure for practical management of patients which is outlined in a functional chart. This model provides an example of running a large diarrheal disease treatment center by trained paramedics and auxiliaries, under supervision of a physician. The model also provides information about the successful use of oral rehydration solution in treating a majority of dehydrated patients. In any developing country where limitation of resources and trained personnel exist, such a model can be easily adopted for the treatment of diarrheal illness.

Sands RL see Casemore DP

San Joaquin VH, Marks MI. New agents in diarrhea. Pediatr Infect Dis 1982 Jan-Feb;1(1):53-65

Sankaranarayanan YS, Santhanakrishnan BR. Chronic diarrhoea in infancy & childhood: rationalised approach and guidelines for dietary management. Pediatr Bull 1986 Jul;8(1):95-102

Sansonetti PJ, d'Hauteville H, Formal SB, Toucas M. Plasmid-mediated invasiveness of <Shigella-like> Escherichia coli. Ann Microbiol (Paris) 1982 May-Jun;133(3):351-5

Invasive Escherichia coli is a Shigella-like microorganism which cause a dysenteric syndrome through invasion of the human colonic epithelium. This paper describes the contribution of plasmid(s) in the virulence of invasive E. coli. All invasive E. coli strains, irrespective of serotype, were found to harbor a large plasmid of approximately 140 megadalton (Mdal). Spontaneous variants of serotypes 0143 and 0124 had lost this plasmid and had become avirulent as determined by their inability to penetrate HeLa cells and to provoke keratoconjunctivitis in guinea pigs. A plasmid was still present in some of these strains, although consistently smaller than those observed in the virulent isolates (i.e. 100 Mdal and less). This suggests that deoxyribonucleic acid sequences involved in the invasive process had been deleted. Virulence of these strains were restored when pWR110, a Tn5-labeled virulence plasmid of Shigella flexneri was transferred to them. These results demonstrate for the first time that invasive E. coli strains, irrespective of their serotype, harbor a 140-Mdal plasmid which is necessary for epithelial cell penetration. This work also demonstrates that S. flexneri and invasive E. coli share a common extrachromosomal control of their ability to penetrate into cells and are closely related when compared to S. sonnei.

Santhanakrishnan BR <u>see</u> Sankaranarayanan YS

Sanyal SC. NAG Vibrio toxin. Pharmacol Ther 1983;20(2):183-201

Saran M. Dabral M. Srivastava RN. Sharma VK. Epidemiology of human rotavirus diarrhoea -- a review. J Indian Assoc Commun Dis 1982 Sep-Dec;5(3-4):50-7

The major findings in rotavirus epidemiology are outlined. Human rotavirus is responsible for as much as 60-80% of acute diarrhea in 6-24-month old children, especially in winter, antibodies against rotavirus have been demonstrated in more than 90% of children aged 5, indicating a previous infection. Vomiting, diarrhea, and mild temperature with mild-severe dehydration are common in human rotavirus diarrhea. Management consists of rehydration therapy by fluids and electrolytes. Antibiotics are not useful. Development of an oral, live attenuated polyvalent vaccine for producing enteric and humoral immunity against human rotavirus diarrhea will be ideal to prevent morbidity and mortality in developed as well as developing countries.

Sarkar U. Community participation in the control of diarrhoea. J Indian Med Assoc 1987 Jul;85(7):210-2

Sarker MR see Ahmed ZU

Sarker SA see Molla A

Sarker SA see Molla AM

Sattherwhite TK, DuPont HL. Infectious diarrhea in office practice. Med Clin North Am 1983 Jan;67(1):203-20

Sauer KT see Guerrant RL

Saw SP. The causes and economic significance of enteric infections in domestic animals. In: Tzipori S, Barnes G, Bishop R, Holmes I, Robins-Browne R, eds. Infectious diarrhea in the young: strategies for control in humans and animals; proceedings of an International Seminar on Diarrhoeal Disease in South East Asia and the Western Pacific Region, Geelong, 10-15 Feb 1985. Amsterdam: Elsevier, 1985:160-2

The causes and economic significance of enteric infections in domestic animals in Burma are described. The livestock sector in Burma involves mainly small-holding farmers who play an important role in economic development. About 70% of the total employment is in the agricultural sector. Cattle and buffalo provide invaluable draft service. Pigs, poultry, sheep and goat rearing is a subsidiary activity of the small farmer. Various kinds of infectious diarrhea have mainly contributed to the calf mortality (9.1%) and piglet mortality encountered annually. Many microorganisms, protozoa, and parasites were identified as causative agents of diarrhea in young animals. Faulty management and feeding systems also contributed to diarrhea. Pathogens identified in pigs were <u>Salmonella</u> cholera suis and <u>Escherichia coli</u>, and <u>Salmonella typhimurium</u> in pigs and <u>cattle</u>. <u>Treponema hyodysenteriae</u> have also been reported in swines. Transmissible gastroenteritis, viral infection and frequent rotavirus infection were found in piglets and calves. Diarrhea due to other bacterial or protozoal agents is discussed. Trichuris suis and Ascaris suum were common parasites found in many pig farms. Ascaris was found cattle and buffaloes. Economic significance of enteric infections discussed. Components and aspects of Burma's diarrheal disease control program are also given. The emphasis of the present control measure is on improving the herd hygiene, better housing and efficient management.

Sbarbati A see Giorgi PL

Schar M see Morger H

Schiller LR see Feldman M

Schlafer DH see Torres-Medina A

Schmidt NJ. Rapid viral diagnosis. Med Clin North Am 1983 Sep;67(5):953-72

Rapid progress in viral diagnosis has made viral examinations much more practical and useful in clinical settings. This has allowed successful intervention in the treatment of patients or their contacts or in the control of viral diseases in the community. The major emphasis in rapid viral diagnosis has been on the development of methods which permit direct detection of virus or viral antigen in the clinical specimen, thus obviating the need to cultivate the virus in a living host system. This permits detection of viruses that cannot be cultivated, and detection of virus in specimens in which the agent is no longer infectious. Direct methods used for viral detection include electron microscopy and various immunoassays which are based on demonstrating reactivity of viral antigen in the specimen with known viral Perhaps, the most important advances in viral isolation and identification have been in the application of immunoassays to the more rapid identification of viruses isolated in cell cultures or other laboratory host systems. reliability of all specific viral identification procedures depends on the use of high-quality viral antisera. Some of the problems previously encountered in preparing satisfactory viral immune reagents are being overcome through the produced highly specific monoclonal antibodies availability of cell-hybridization techniques. Monoclonal antibodies to human IgM have been produced for use as "capture" antibodies for assay of viral IgM, and the availability of anti-u reagents of this source should overcome previous problems of specificity, consistency, and supply, and should facilitate standardization and development of reliable assays for IgM antibodies to viral and other microbial agents. A variety of rapid and relatively simple tests have been developed in recent years for the determination of immunity status to certain viruses, and some of these assays are now commercially available. reliability of some of these antibody assays has improved through the incorporation of more suitable controls and through clearer interpretations of test results.

Schneider RE see Rosenberg IH

Schoch PE, Cunha BA. Aeromonas. Infect Control 1984 Nov;5(11):542-4

The genera Aeromonas and Plesiomonas are Gram-negative, cytochrome oxidase-positive, asporogenous, facultatively anaerobic rods that ferment carbohydrates with the production of acid or acid and gas, and are members of the family Vibrionaceae. The aeromonads are ubiquitous organisms whose habitats include the soil, salt and fresh surface water, sewage, and a number of aquatic animals and plants. Their survival in the environment depends on a continued supply of moisture and organic matter. Although Aeromonas hydrophila has been reported in fecal specimens from healthy individuals, investigators do not agree that it is a normal inhabitant of the gastrointestinal tract. Aeromonas hydrophila was first implicated in cases of severe gastroenteritis in 1961 in Colombia, and since then, cases have been reported in Europe, Africa, India, Asia, and North America. Isolates of Aeromonas and Plesiomonas are generally nonfastidious in nature and require no special precautions in specimen collection and transport to ensure recovery. These organisms usually grow on most laboratory media, including blood agar, enteric selective and

differential media and infusion broths. Most strains of Aeromonas beta-hemolytic on blood agar and the majority do not ferment lactose on MacConkey agar. Aeromonas infections in the hospital setting are rare. Of the 5 Aeromonas species, A. hydrophila is the species most frequently associated with human infection. Aeromonas species are pathogens of low virulence. Aeromonas may be present in the fecal flora of a compromised host. It is not part of the normal fecal flora of immunocompetent hosts. Wound infections due to Aeromonas provide the other possible hospital reservoir for these organisms. Aeromonas causes an interesting variety of infections in the community, particularly severe wound infections following traumatic water/soil injuries. Gastroenteritis and enteric fevers have been described in association with this organism. Aeromonas produces a short-lived gastroenteritis with a watery Ordinarily, this is a self-limiting illness. With the exception of Aeromonas gastroenteritis which is usually a self-limiting disorder that requires no treatment. Aeromonas infections ordinarily are treated with a wide variety of antimicrobials. The most active antibiotics against these organisms include aminoglycosides. the chloramphenicol, the tetracvclines. trimethoprim-sulfamethoxazole, and currently the third generation cephalosporins.

Schoeneman MJ see Feld LG

Schoppe LE see Patterson M

Schooling JB see Guerrant RL

Schreifer DS see Wolf JL

Schuster MM see Tucker H

Sciortino CV see Finkelstein RA

Scrimshaw NS see Keusch GT

Seal SC. Centenary of discovery of cholera <u>Vibrio</u> [editorial]. Indian J Public Health 1983 Jan-Mar:27(1):1-4

On the occasion of the centenary of the discovery of Vibrio cholerae by Robert Koch of Germany, the author discusses the reasons why cholera is still menacing Asian countries, particularly India, despite 50 years of effort to curtail the problem. A number of mistakes committed by serological researchers and health authorities, particularly regarding classification, are discussed. For example, vibrios, including El Tor, not agglutinated by serum raised against the so-called "true cholera <u>Vibrio</u>" were regarded as noncholeragenic. The author concludes that attempts to control cholera should place more importance on human carriers.

Selivanov V see Morris JA, Jr.

Sen PC, Banerjee H. Diarrhoea - the great killer of infants and children. Your Health 1983 Jan;32(1):9-14

This paper discusses various aspects of diarrheal diseases in infants and children. Such etiological agents as bacteria, viruses, and parasites are described. Improved personal hygiene, using boiled water, encouraging breast

feeding, providing health education to mothers, etc. are indicated for the prevention of diarrhea. Management of diarrhea by oral rehydration, using the WHO-recommended solution or home-made fluids, and specific therapy with different antibiotics, are also outlined.

Sepulveda B. Amebiasis: host-pathogen biology. Rev Infect Dis 1982 Nov-Dec;4 (6):1247-53

"Invasive amebiasis caused by Entamoeba histolytica, and particularly amebic liver abscess, is a major public health problem in Mexico and some other countries because of the high incidence and mortality due to this disease. This paper first discusses the pathogenic effect of E, histolytica and the defensive response of the host and then reports studies concerning experimental induction of protective immunity to amebic infection. pathogenic effect of E, <u>histolytica</u> is probably initiated by a lectin-mediated adhesion of trophozoites to target cells; the adhesion is followed by cytopathic activity and phagocytosis by the ameba. The defensive response is characterized by humoral and cellular immune reactions. Humoral immunity manifests itself by specific circulating antibodies useful in the diagnosis and seroepidemiology of amebiasis. Cellular immunity is shown by characteristic reactions. Experimental induction of immunoprophylaxis with E. histolytica antigens represents the first stage in the development of a vaccine against E. histolytica for use in humans." (Author's abstract)

Sepulveda B. Progress in amebiasis. Scand J Gastroenterol 1982;17(suppl 77): 153-64

This review presents a summary of the principal advances in the following areas: 1) the morphology and biology of the parasite; 2) the reaction of host as demonstrated by the immune response and distinctive features of amebic lesions: 3) diagnosis; and 4) treatment of amebiasis. There is information concerning the biochemical composition of the parasite as well as some of its functions, such as motility endocytosis and pathogenic action. Both humoral and cell-mediated immunity participate in the immune response. The humoral immune response in human invasive amebiasis as well as in experimental models is characterized by the rapid appearance of circulating antiamebic antibodies. These specific antibodies can easily be detected by various techniques, and their identification is particularly useful in the diagnosis of the disease and in seroepidemiological surveys of amediasis. There are two distinct features of intestinal and hepatic amebic lesions. first is that when there is a moderate inflammatory reaction in early lesions, necrosis generally predominates over inflammation in late lesions. The second is that amebic lesions heal without the formation of scar tissue, even in the case of extensive lesions such as those that occur with hepatic abscess ameboma of the colon. Rectosigmoidoscopy, identification of hematophagous trophozoites in rectal scrapping, detection of antiamebic antibodies in serum, diagnostic scintillography useful procedures. are bns Ultrasonography and computerized tomography with diagnostic efficiency of 90% respectively are two diagnostic methods developed recently. Metronidazole and its congeners are the drugs of choice in the therapy of invasive amebiasis. The demonstration that different antigens obtained from Entamoeba histolytica-induced antiamebic protective immunity in various animal species and the confirmation of the innocuousness of these antigens suggest the possibility of obtaining an antiamebiasis vaccine for use in humans.

Sethabutr O see Echeverria P

Sever JL. Infectious diseases and immunizations. Rev Infect Dis 1982 Jan-Feb;4(1):136-46

Shanahan F see Targan SR

Shane SM, Montrose MS. The occurrence and significance of <u>Campylobacter</u> <u>jejuni</u> in man and animals. Vet Res Commun 1985 Jul;9(3):167-98

"Campylobacter jejuni, which is now recognized as a discrete species, is a gram negative, microaerophilic, thermophilic, nalidixic acid sensitive, hippurate positive pathogen requiring special selective media for propagation. The organism is widely distributed in avian species, experimental and companion animals and in humans. Mammalian campylobacteriosis is characterized by an enterocolitis of variable severity. The prevalence of the condition relatively high in young individuals, in underdeveloped countries and in subjects with diarrhea. food animals, especially poultry, are reservoirs of the organism and infection occurs following consumption of untreated surface water, unpasteurized milk, incompletely cooked meat or other contaminated food products. Close contact with infected immature companion animals is a significant cause of campylobacteriosis in children and direct intrafamilial transmission and occupational infection have Campylobacteriosis attributable to <u>C. jejuni</u> is a condition of emerging significance which arises principally from deficiencies in hygiene inherent in the environment and in the food chain which extends from domestic animals to the consumer." (Author's abstract)

Shanmugan R. An intervened Poisson distribution and its medical application. Biometrics 1985 Dec;41(4):1025-9

Sharma VK see Saran M

Sharp GW see Donowitz M

Sharp GWG see Donowitz M

Sharp JCM. Infections associated with milk and dairy products in Europe and North America, 1980-85. Bull WHO 1987;65(3):397-406

"Outbreaks of infection associated with milk and other dairy products in Europe and North America from 1980 to 1985 are reviewed. Salmonella spp. and Campylobacter spp. were the most commonly identified etiological agents, while other infections of animal origin, in particular listeriosis and yersiniosis, were increasingly reported. Most infections were attributed to untreated cows' milk or cheese, but also increasingly to contaminated "heat-treated" products. Heat-treatment is highly effective in controlling foodborne disease, but may be insufficient if not complemented by high standards of hygiene throughout production and processing. Large community outbreaks of salmonellosis, listeriosis, and yersiniosis in Canada, Sweden, the United Kingdom, and the USA that were associated with contaminated "heat-treated" liquid milk, powdered milk, or cheese emphasize the vulnerability of dairy produce." (Author's abstract)

Sheldon GF see Morris JA, Jr.

Shelton S see Nelson JD

Shetty RK see Wagh MG

Shiau YF. Clinical and laboratory approaches to evaluate diarrheal disorders. CRC Crit Rev Clin Lab Sci 1987;25(1):43-69

"Diarrheal disorders are the result of excessive fluid and electrolyte loss through the gastrointestinal tract. Many different underlying mechanisms are known to cause diarrhea. Fordtran suggested that in secretory diarrhea osmolality of stool water should be accounted for by its electrolyte contents. Therefore, the osmotic gap between the measured osmolality and that estimated from electrolyte contents should be small. In osmotic diarrhea, due to the presence of the osmotic agent, there should be a greater gap between the measured and the estimated osmolalities. Osmotic gaps varying from 100 to 40 mOsm have been used arbitrarily in literatures to define the underlying pathogenesis. Because of the uncertainty, the usefulness of these measurements remains in question. In this article, methods used to measure stool osmolality and electrolyte contents are reviewed. Limitations of these measurements are discussed. Measurements derived from various diarrheal disorders revealed that the basic concepts put forward by Fordtran are corrected. However, we found that the osmotic gaps (measured osmolality - 2 [Na+K] in secretory diarrheal disorders are frequently negative numbers. In osmotic diarrhea, the osmotic gap (greater than 160 mOsm) is substantially greater than the figures used in the literature. In many diarrheal disorders the osmotic gap falls between the two extremes and the pathogenesis is multifactorial in origin. Under these circumstances, stool osmolality and electrolyte measurements provide little insight into the underlying mechanism causing the diarrhea. Furthermore, stool contains many biologically active organisms which can alter the stool osmolality. Unless these effects are appreciated, an inaccurate interpretation of these measurements may result." (Author's abstract)

Shields DS see Guerrant RL

Shimonishi Y see Takeda Y

Shiner M. Autoimmunity in gastrointestinal diseases. Arq Gastroenterol 1986 Apr-Jun;23(2):99-103

This article is an update on the importance of some gastrointestinal diseases, such as ulcerative colitis, celiac disease, and some types of diarrhea, in It is that likely autoimmune response. gastroenteropathies, like gastritis, inflammatory bowel disease, and celiac disease associated with long-standing hyperimmune activity, acquire a state of immune tolerance which may eventually lead to a loss of recognition of self antigens and thus to an autoimmune disease state. The induction of immune tolerance to dietary antigens has been well documented experimentally and has also been linked to genetic MHC-associated disease. The antigens may not only be dietary but also bacterial, viral, or other, as yet unknown, factors. could, therefore, speak of autoimmune phenomena induced as secondary to chronic inflammatory gut disease and contrast these with the protracted diarrheas and autoimmune organ-specific disease in infants. From the practical point of view, autoimmune disease associations are important in the recognition and investigation of extraintestinal complications of chronic gastrointestinal affections. The immunological processes underlying the induction of autoimmune

disease is not yet clearly understood. It may implicate abnormalities in T-cells (suppressor/cytotoxic or contra-suppressor) or B-cells (overstimulation idiotypic network or direct suppression of antibody-mediated inhibition). It is also not understood in exactly what way genetic factors cause the disturbance of this type of aberrant immunoregulation. The mechanism whereby autoimmune disease develops is, therefore, hypothetical to date. is, however, of interest that in the chronic diarrhea of childhood malnutrition with immune deficiency (involving associated predominantly) autoimmune disease is apparently unknown. This would provide additional evidence that T-cells are involved in autoimmune disease. correlation with autoimmune disease without digestive symptoms is (rheumatoid arthritis, Hashimoto's thyroiditis, anklosing spondylitis, and so on). Recognizing such interrelations should be followed by search of extradigestive symptoms of autoimmune gastrointestinal diseases, allowing a better understanding of the immunologic phenomena involved. Gastrointestinal disease and autoimmunity, local or general, has been reported for almost 20 years. The immunological processes involved are only just beginning to be It is hoped that this paper will stimulate gastroenterologists to undertake more intensive investigations into this subject.

Shmerling DH see Goriup U

Shuval HI, Yekutiel P, Fattal B. An epidemiological model of the potential health risk associated with various pathogens in wastewater irrigation. Wat Sci Technol 1986:18(10):191-8

"The primary goal of this study has been to carry out a rigorous, critical review and evaluation of all available documented epidemiological evidence with the aim of determining the significant, quantifiable, health effects on population groups directly or indirectly exposed to wastewater irrigation: through occupation; by residing in contiguous communities or by consuming the sewage irrigated crops. Based on these findings, we have developed an epidemiological model predicting the degree of risk associated with various groups of pathogens. Based on theoretical consideration of the model and confirmation by the empirical evidence collected and analyzed in this study, we have concluded that the risk of pathogen transmission by irrigation with raw wastewater for most developing countries is according to the following descending order: high-helminths (Ascaris, Trichuris, Ankylostoma and Taenia), lower-bacteria (Typhoid and Cholera), and lowest-viruses. In general, it can be stated that based on both the theoretical and empirical findings of this study, there appears to be a basis for relaxing the conventional effluent quality standards for unrestricted crop irrigation. A quideline of no helminths in 1 liter of water and a log mean of 1.000 fecal coliforms/100 ml is proposed," (Authors' abstract)

Shuval HI, Fattal B, Yekutiel P. State of the art review: an epidemiological approach to the health effects of wastewater reuse. Wat Sci Technol 1986;18 (9):147-62

"The reuse of wastewaters for agricultural irrigation can be a means of reducing the pollution of surface waters including those in coastal areas used for bathing. The wastewater stream of a community carries within it the complete spectrum of pathogenic bacteria, viruses, protozoa and helminths which are endemic in the community. These pathogens can survive sufficiently long in the soil or on crops to infect, at least in theory, persons coming in direct

contact. Thus, it is important to establish a sound epidemiological basis for health regulations related to the reuse of wastewater in agriculture. paper presents the theoretical epidemiological considerations that should serve as the basis for a predictive model of the potential risks associated with quantifiable credible. irrigation. Empirical data from wastewater epidemiological studies have been reviewed to validate the theoretical model. For developing countries, the authors have concluded that the ranking of pathogens as to the degree of risk associated with wastewater irrigation is as follows: high risk - helminths, medium risk - bacteria and protozoa and low In order to effectively reduce the concentration of these risk – viruses. pathogens wastewater treatment is recommended as the most effective control strategy." (Authors' abstract)

Siegenbeek van Heukelom J. Physiological aspects of absorption and secretion in intestine. Vet Res Commun 1986 Sep;10(5):341-54

Silva J, Jr., Fekety R. <u>Clostridia</u> and antimicrobial enterocolitis. Annu Rev Med 1981;32:327-33

Simhon A see Mata L

Simmons DAR, Romanowska E. Structure and biology of <u>Shigella flexneri</u> 0 antigens. J Med Microbiol 1987 Jun;23(4):289-302

The species, Shigella flexneri, comprises a serologically heterogeneous group dysentery bacilli whose 0 antigens are polysaccharide-lipid A-polypeptide-lipid B complexes strictly comparable in their gross structural and biological properties to the analogous antigens present in Gram-negative bacilli of the family Enterobacteriaceae. The lipopolysaccharide (LPS) component of these antigens comprises two distinct regions - a common basal structure shared by all \underline{S} . flexneri serotypes (except serotype 6) and O-specific side-chains that determine the serological specificity cross-reactivity of the whole antigen. In an earlier review, structural and genetic aspects of the biosynthesis of the \underline{S} . $\underline{flexneri}$ 0 antigens were considered in detail. The conclusions drawn at that time about the structural changes involved in smooth to rough (S+R) mutation, in the production of X and Y variants, and in the modification of type-specificity by lysogenic conversion remain valid today. However, some of the structures themselves require to be revised, largely as a result of methylation studies performed during the past decade by Lindberg and his colleagues. This review summarizes the evidence for these currently accepted structures and indicates how these studies have elucidated the biology of the <u>S</u>. $flexneri\ 0$ antigens. These studies are of intrinsic value in that they have solved the molecular basis of serological specificity and cross-reactivity, elucidated the biochemical and enzymic basis of smooth to rough (S+R) mutation and A+B variation, defined the structural relationship and biosynthetic pathways of smooth and rough antigens, and indicated the mechanisms involved in the genetic control of S. flexneri O-antigen biosynthesis. It seems reasonable to predict that similar studies will continue to reveal structural and genetic with other organisms relationships between different genera that will ultimately clarify the evolutionary history of Enterobacteriaceae.

Simon GL, Gorbach SL. Intestinal flora in health and disease. Gastroenterology 1984 Jan;86(1):174-93

Simon GL, Gorbach SL. Intestinal microflora. Med Clin North Am 1982 May;66

Simpson TW see Mahalanabis D

Singh J see Aggarwal P

Singh V see Broom S

Sitrin MD see Rosenberg IN

Skirrow MB see Butzler JP

Slater PE see Ellenoweig AY

Sleisenger MH see Freeman HJ

Smith KL see Saif LJ

Smith SM, Palumbo PE, Edelson PJ. <u>Salmonella</u> strains resistant to multiple antibiotics: therapeutic implications. Pediatr Infect Dis 1984 Sep-Oct;3(5):

Snyder IS see Cavalieri SJ

Snyder JD, Merson MH. The magnitude of the global problem of acute diarrhoeal disease: a review of active surveillance data. Bull WHO 1982;60(4):605-13

"Data from 24 published studies were analysed in order to estimate the annual morbidity and mortality from acute diarrhoeal disease in the developing world. Twenty-two of the studies involved frequent surveillance through home visits to families in communities; the other two were multi-country studies in which diarrhoea mortality was calculated on the basis of death certificate information. Morbidity rates were found to be highest in the 6-11-month age group, while the mortality rates were greatest in infants aged under 1 year of age and children 1 year old. For children aged under 5 years old, the median incidence of diarrhoea was 2.2 episodes per child per year for all studies and 3.0 episodes per child per year for the studies that had the smallest populations and most frequent surveillance. Using 1980 population estimates, the estimated total yearly morbidity and mortality from diarrhoeal disease for children under 5 years of age in Africa, Asia (excluding China), and Latin America were 744-1,000 million episodes and 4.6 million deaths." abstract)

Snyder JW see Buchino JJ

Solomons NW see Keusch GT

Solomons NW see Rosenberg IH

Sory M-P see Cornelis &

Southal H see Feachem RG

Soutter VL, Kristidis P, Gruca MA, Gaskin KJ. Chronic undernutrition/growth

retardation in cystic fibrosis. Clin Gastroenterol 1986 Jan;15(1):137-55

Spaan WJ see Horzinek MC

Sparling PF. Bacterial virulence and pathogenesis: an overview. Rev Infect Dis 1983 Sep-Oct;5(suppl 4):S637-46

"Bacterial virulence is the result of many different attributes, which often contribute to different steps in the complicated series of events we recognize as infection. With the use of selected examples, an overview of the stages of infection common to many bacterial infections (attachment, proliferation, tissue damage, invasion, and dissemination) is presented. Expression of virulence depends on a large number of host variables, including nonspecific and specific immune defenses, and interruption of the process of infection is undoubtedly possible at many different steps. Future developments will depend on better definition of the biochemistry, genetics, and immunology of the bacterial factors involved in pathogenesis." (Author's abstract)

Srivastava RN <u>see</u> Saran M

Steele JH see Du Pont HL

Steffen R. Epidemiologic studies of travelers' diarrhea, severe gastrointestinal infections, and cholera. Rev Infect Dis 1986 May-Jun;8(suppl 2):S122-30

"A retrospective survey, which is based on interviews conducted between 1975 and 1984 with 20,000 European tourists returning from 15 destinations in various climatic zones, demonstrates that travelers' diarrhea is the most frequent health problem encountered by travelers in the tropics. The incidence varied from 4% to 51%, depending on the destination. High-risk groups were persons younger than 30 years, adventurous travelers, and travelers with preexisting gastrointestinal illnesses. Illness acquired at various geographic regions showed only minor differences in chronology and symptomatology. The clinical course of travelers' diarrhea was usually short and mild. Additionally, by longitudinal and retrospective analyses, the incidence and prognosis of gastrointestinal infections of greater severity that were acquired after a short stay in a developing country, such as giardiasis, amebiasis, typhoid fever, and cholera, were evaluated; typhoid fever and cholera, in particular, were found to be quite rare." (Author's abstract)

Steffen R. Epidemiology of travellers' diarrhoea. Scand J Gastroenterol 1983;18(suppl 84):5-17

"Travellers' diarrhoea is the most frequent health problem encountered during a stay in developing countries. A recent study based on interviews with 16,568 charter-flight passengers returning to Europe from 13 destinations in various climatic regions provides epidemiological data on a worldwide scale. Significant differences in diarrhoeal incidence varied not only between individual destinations, but also between hotels in the same area. Travel characteristics, and a record of former tropical journeys influenced the incidence to a minor degree. Persons under 30 were more often affected than older travellers. Within international groups meeting in developing countries, the risk varied according to the patient's country of origin, with the residents of industrialised nations being most often affected. The various

regions show nonessential differences in chronology and symptomatology. This is consistent with quantitative rather than qualitative geographical variations in causative agents. Potentially life-threatening gastrointestinal infections, such as typhoid and cholera, are very rarely acquired by tourists." (Author's abstract)

Steffen R, Heusser R, DuPont HL. Prevention of travelers' diarrhea by nonantibiotic drugs. Rev Infect Dis 1986 May-Jun;8(suppl 2):S151-9

"Travelers have resorted to a variety of drugs for prevention of diarrhea. No beneficial prophylactic effect has been confirmed for halogenated hydroxyquinolines, lactobacilli, antimotility drugs, ethacridine, and various other agents. In contrast, bismuth subsalicyate (BSS) in liquid form reduced the incidence of diarrhea in students from the United States living in Mexico and in tablet form in volunteers challenged by enterotoxigenic Escherichia coli. In tourists visiting various developing countries, a randomized, double-blind study was conducted in which 390 persons received a total of 2.1 or 1.05 g of BSS daily or placebo in tablet form in two doses. BSS reduced the incidence of diarrhea by 41% in the high-dose group and by 35% in the low-dose group without causing important adverse reactions." (Authors' abstract)

Steffen R see Morger H

Steinhoff MC. Rotavirus: the first five years. J Pediatr 1980 Apr;96(4): 611-22

This article summarizes a portion of a large body of data on rotavirus accumulated during 1975-1980. The human rotavirus is spherical, 70 nm in diameter, and its name is derived from its distinctive double capsid structure. Its microscopic appearance with the wide hub, short spokes, and thin rim makes it look like a wheel. Its double-stranded ribonucleic acid (RNA) genome is in 11 segments, and it contains an RNA polymerase. The human rotavirus is a hardy virus, resisting a temperature of 56° C, pH of 3, and most detergents and solvents. The following points summarize the evidence presented: (1) they are often found in high concentration in the stools of diarrheic children who are aged under 2; (2) the virus has been cultured with difficulty and undergone a number of passages in organ and cell culture; (3) the human virus will cause disease in colostrum-deprived newborn rhesus monkeys, piglets, lambs, calves, and can be transmitted serially in piglets, lambs, and calves; and (4) human infants and experimental animals, all demonstrate serologic response to infection with this virus. After the initial reports from Australia, UK, and Canada of human rotavirus in diarrheal stools, the virus was reported from many areas, including Japan, USA, Central and South America, Africa, India, Bangladesh, Singapore, and New Guinea. Although the early reports alleged the rarity of human rotavirus disease at ages under 6 months or above 5 years, there have been subsequent reports of neonatal and adult disease. However, the adult human rotavirus disease is often mild, perhaps because many of such episodes are reinfections. A patchy irregularity of the mucosal surface, shortening and blunting of villi, distorted microvilli of absorptive cells, and a decrease in duodenal disaccharidases are the major pathologic findings. destruction of mature enterocytes results in reduced levels of disaccharidases and a decrease in the absorptive surface of the small bowel. The diarrhea, caused by human rotavirus, is probably due to decreased absorption; increased secretion does not appear to be involved. Specific serum IgM is produced in response to primary infection. In some adults, an early IgG elevation has been

noted. The presence of serum antibody is not fully protective. The illness is characterized by the abrupt onset of vomiting and diarrhea. Mild-to-moderate found frequently. The course of human dehydration was gastroenteritis is usually short in hospitalized children. The fever and vomiting resolve in the first day or two, but the diarrhea may last up to 8 days. Diagnosis of human rotavirus infection rests on the demonstration of the virus in the stool or on serologic methods. As with all diarrheal disease. fluid replacement remains the most important aspect of therapy for human Human milk provides some degree of protection rotavirus gastroenteritis. against the disease, through both specific antibodies and nonspecific factors. The existence of multiple serotypes and the recognition of both asymptomatic and repeat infections indicate that the intricacies of immune protection must be better understood.

Steinhoff MC. Viruses and diarrhea--a review. Am J Dis Child 1978 Mar;132 (3):302-7

"A critical analysis of the literature shows that only a few viral agents have been proved by accepted criteria to cause acute gastroenteritis. Rotavirus, of the RNA family reoviridae, is the cause of most childhood gastroenteritis. The parvovirus-like agents (Norwalk, Hawaii, MC, and W) are proved agents of epidemic gastroenteritis. There is good evidence that the Marcy, FS, and Niigata agents were the causes of gastroenteritis outbreaks. Thirty, percent of all acute gastroenteritis episodes in large investigations are still etiologically unaccounted for. Much work remains to be done to define the pathogenesis and immunology of viral gastroenteritis." (Author's abstract)

Sternberg N. Evidence that adenine methylation influences DNA-protein interactions in Escherichia coli. J Bacteriol 1985 Oct;164(1):490-3

Stevens DP. Selective primary health care: strategies for control of disease in the developing world. XIX. Giardiasis. Rev Infect Dis 1985 Jul-Aug;7(4): 530-5

"Giardia lamblia infects millions of individuals throughout the world. In developed countries it appears primarily in waterborne epidemics of diarrhea. In developing countries, it is endemic, but only a small proportion of those infected appear ill. This flagellate parasite infects the small intestine of its host and may cause malabsorption and malnutrition, particularly among infants and young children. Little is known about the extent of illness caused by this parasite because few epidemiologic studies have been done, diagnosis is difficult, and <u>Giardia</u> carriers frequently are simultaneously infected with other pathogens. Control measures include intermittent treatment of those infected and improved water supply and sanitation. Efforts to control individual infection can only be successful on a temporary basis. The greatest progress in control should derive from efforts to develop an effective vaccine." (Author's abstract)

Strober W. Animal models of inflammatory bowel disease - an overview. Dig Dis Sci 1985 Dec:30(suppl 12):S3-10

Suchy FJ see Balistreri WF

Suchy FJ see Buchino JJ

Sudo R. Aiba S. Role and function of protozoa in the biological treatment of

polluted waters. Adv Biochem Eng Biotechnol 1984;29:117-41

Summary of recommendations and conclusions: vaccines. <u>In</u>: Priorities in biotechnology research for international development; proceedings of a workshop, Washington, D.C. and Berkeley Springs, 20-26 Jul 1982. Washington, D.C.: Board on Science and Technology for International Development Affairs, National Research Council, 1982:1-7

Sunoto. Diarrhoeal problems in Southeast Asia. Southeast Asian J Trop Med Public Health $1982 \, \mathrm{Sep}; 13(3): 306-18$

In Southeast Asia, diarrheal diseases are associated with high morbidity and mortality, particularly among children aged under 5. The magnitude of the diseases in some Southeast Asian nations (Indonesia, Philippines, Thailand, Malaysia, Singapore and Bangladesh) was evaluated by analyzing data from these countries. In Indonesia, morbidity rates were 430 per 1,000 population per year, of which 70% were children aged under 5. It was estimated that Indonesia in 1974 had about 50 million diarrhea cases, and 60 million episodes in 1981 with 300,000-500,000 deaths. In the Philippines, diarrhea ranked as the second morbidity cause for all age groups in 1974 (600 per 100,000), and as the second infant mortality cause (5 per 1,000). In Thailand, 93,786 diarrhea cases were hospitalized in 1980. Forty percent of these were children aged under 5. Among these patients, the morbidity rates associated with acute diarrhea. cholera and dysentery were, respectively, 458.03, 9.09 and 56.44 per 100,000 people. In Malaysia, there were outbreaks of El Tor cholera and poliomyelitis in 1971 and 1972, and in 1976, diarrhea was the number 5 cause of total hospital admissions and the number 9 cause of deaths in Malaysia. In Singapore, comparing 1975 to 1964, infant, perinatal and neonatal mortality rates dropped, respectively, from 31.2, 26.2 and 19.1 per 1,000 births to 13.9, 16.6 and 10.2 per 1,000 births. Also during this period, there was a marked reduction in deaths due to pneumonia and diarrhea respectively, from 3% and 23% to 14% and 4%. In Bangladesh, morbidity rates were highest among children and declined with age. In urban areas, the attack rate for under-5 children was 11-fold higher than for adults, but the difference was only 3-fold in rural areas. The overall attack rate implies a prevalence of 2% for the entire population, with the highest prevalence 4.5% for the under-5 groups. In a rural population of 100,000, the diarrheal attack rate was 85,000 (85.4%) episodes annually, of which 37% were cholera in the under-5 group. Mortality was highest in infants, followed by toddlers and old people. The commonest pathogens found in these countries were rotavirus, followed by enterotoxigenic Escherichia coli, Vibrio cholerae, Salmonella sp., Shigella sp., and Campylobacter sp., In addition to socioeconomic, sociocultural and poor environmental sanitation, malnutrition and a decline in breast feeding also played vital roles in causing high diarrheal disease morbidity.

Sunoto. Oral rehydration salts: a simple and appropriate tool against dehydration due to diarrhoea. Paediatr Indones 1981 Mar-Apr;21(3-4):90-100

Diarrheal disease is identified as one of the major public health problems with high morbidity and mortality particularly in children aged under 5, although recent advances in diagnosis, pathogenesis and pathophysiology of this disease have been quite significant. Oral rehydration therapy (ORT), known since the 1950's and used extensively in hospitals and treatment centers in the 1960's, has become a worldwide program, since the 1970's. This paper communicates these facts and suggests a mass campaign in favor of ORT. Nowadays more than

70 countries use ORT as a major treatment of dehydration due to diarrhea. Sodium concentration in oral rehydration solution (ORS) is one of the problems still in dispute. Extension of ORT to the use of sucrose-electrolyte solution and sugar-salt solution by using pinch or scoops has enjoyed some popularity. It has been proven that ORT has a beneficial impact in improving nutrition, saving money, and dramatically decreasing mortality. For Indonesia, it is suggested that national efforts to implement ORT must be part of the general program for delivery of basic health services. Integration of ORT with primary health care modalities, such as family planning, nutritional programs, maternal and child health, immunization, environmental sanitation, health education, etc., is also suggested. A public campaign, using all communication media, may contribute greatly in delivering the message to the people wherever they live, thereby ensuring that treatment of diarrhea begins at home with ORS.

Suskind R see Cvjetanovic B

Svennerholm A-M, Holmgren J. Oral combined B subunit - whole cell cholera vaccine. In: Holmgren J, Lindberg A, Mollby R, eds. Development of vaccines and drugs against diarrhea; proceedings of the 11th Nobel Conference, Stockholm, 1985. Lund: Studentlitteratur, 1986:33-43

"An oral cholera vaccine has been developed which consists of purified B subunit and formalin as well as heat-inactivated cholera vibrios (classical and El Tor organisms expressing i.a. Inaba and Ogawa lipopolysaccharides and fucose-binding and mannose-binding hemagglutinins). This B-subunit whole cell cholera vaccine (B+WCV) has proved to be completely safe in Swedish, Bangladeshi and American volunteers. Studies in Bangladesh have shown that immunization with B+WCV stimulated gut mucosal IgA antitoxin and antibacterial (anti-lipopolysaccharide) antibody formation and local immunological memory comparable to the immune response induced by cholera disease itself. In addition, as evident from a field-based family study orally administered B subunit alone in doses of 1-2 mg gave rise to rapid nonimmunological partial protection against disease in cholera-infected individuals by blocking cholera toxin receptors (p=0.08 vs placebo). In American volunteers who received three oral immunizations with B+WCV the vaccine-induced immune response associated with protection (p<0.01) against challenge with an ID_{100} dose of live cholera vibrios. A field trial in Matlab, Bangladesh by ICDDR, B has been initiated to (i) evaluate the protective efficacy and duration of protection against cholera afforded by both the oral combined B+WCV and its whole-cell component (WCV) alone versus an <u>Escherichia coli</u> K 12 placebo vaccine, (ii) assess the cross-protection against disease caused by LT-producing <u>Escherichia</u> coli and other toxigenic organisms (e.g. non-01 \underline{V} , cholerae and \underline{V} , mimicus) that may be afforded by the B subunit in the combined vaccine, and (iii) determine vaccine-induced cholera antibody titer rises in breastmilk in lactating mothers as these may provide protection for the breast-fed child." (Authors' abstract)

Svennerholm A-M, Jertborn M, Gothefors L, Karim A, Sack D, Holmgren J. Secretory immunity to Vibrio cholerae bacteria and cholera toxin: prospects for an improved cholera vaccine. In: Takeda Y, Miwatani T, eds. Bacterial diarrheal diseases. Tokyo: KTK Scientific Publishers, 1985:169-74

Evidence of protective immunity in cholera and the nature of protective immunity are examined in relation to secretory immunity to <u>Vibrio</u> cholerae. Recovery from disease, induced by perorally administered cholera vibrios is

associated with solid resistance for at least 3 years to rechallenge with either homologous or heterologous <u>Vibrio</u> serotypes. Studies of convalescents have shown that clinical cholera evokes a significant vibriocidal as well as enterotoxin-neutralizing antibody response in serum. Locally produced IgA antibodies are of prime importance for protective immunity against noninvasive enteric infections. The immunization efforts should be directed at identifying optimal means for stimulating intestinal antibody formation to the major protective antigens. Clinical cholera, which is followed by long-lasting immunity gives rise to secretory IgA antibody responses to toxin as well somatic antigens in the intestine. Since antibacterial and antitoxic immunity have been found to cooperate synergistically in conferring protection against experimental cholera, a future cholera vaccine should contain a combination of toxin-derived and somatic antigens, and administered so that it stimulates mucosal immunity comparable to that induced by clinical cholera infection. Research results show that two peroral immunizations of volunteers Bangladesh with a combined B-subunit whole-cell vaccine gave rise to mucosal IgA antitoxin as well as antilipopolysaccharide antibody formation and also development of a local immunologic memory which closely resembled the immune response in cholera convalescents and was superior to the response obtained by parenteral vaccination.

Svennerholm A-M see Holmgren J

Tacket C see Levine MM

Takeda T see Takeda Y

Takeda Y, Shimonishi Y, Yamamoto T, Takeda T. [Enterotoxins produced by enterotoxigenic <u>Escherichia</u> <u>coli</u>]. Tanpakushitsu Kakusan Koso 1986 Mar;31 (suppl 4):324-52

Takeda Y. "Second century of $\underline{\text{Vibrio}}$ $\underline{\text{cholerae}}$ " [editorial]. J Diarrhoeal Dis Res 1984 Mar: 2(1):1-2

Tanowitz HB, Weiss LM, Wittner M. Diagnosis and treatment of protozoan diarrheas. Am J Gastroenterol 1988 Apr;83(4):339-50

Targan S see Elson CO

Targan SR, Kagnoff MF, Brogan MD, Shanahan F. Immunologic mechanisms in intestinal diseases. Ann Intern Med 1987 Jun; 106(6):853-70

"The intestine is a unique immunologic organ that comprises an afferent and efferent compartment and provides the host with the ability to respond through several different effector mechanisms against environmental factors. We discuss mechanisms in three intestinal diseases in this overview of the mucosal immune system. Genetic and immunologic factors are important in the pathogenesis of celiac disease, which is characterized by damage to the mucosa of the small intestine with resultant malabsorption. Pathogenic microbes are important environmental agents that interact with the intestinal mucosa and initiate local immune responses. Advances in the understanding of the mucosal immune response to these pathogenic microbes have produced a clear picture of the way in which this specialized immune system works in concert with systemic immunity. As to the autoimmune nature of inflammatory bowel disease, no specific antigen has been shown to incite the inflammatory reactions and

neither the target cells nor the effector mechanism involved have been identified. Several factors exist, however, to suggest an autoimmune mechanism and the role of mucosal immunologic factors in this disease." (Authors' abstract)

Tasman-Jones C, Kay RG, Lee SP. Zinc and copper deficiency, with particular reference to parenteral nutrition. Surg Annu 1978;10:23-52

"Trace mineral metabolism has acquired renewed importance and received a new stimulus with the development of total intravenous feeding. A syndrome of acute zinc deficiency has been described in patients receiving intravenous feeding with pure amino-acid infusates. Although the rapid response to zinc therapy makes it tempting to assume that the syndrome we have recognized during total parenteral nutrition is one of pure zinc deficiency, it is very likely that this is an oversimplification. The subtle relationships between zinc and other metals, such as calcium, copper, cadmium, and selenium, need further clarification and may account for some patients with low serum zinc not developing the expected clinical manifestations. Copper deficiency also occurs, but its importance is not yet as clearly defined as that of zinc deficiency." (Authors' abstract)

Tay JS see Yip WC

Taylor DN, Echeverria P. Etiology and epidemiology of travelers' diarrhea in Asia. Rev Infect Dis 1986 May-Jun;8(suppl 2):\$136-41

Travelers' diarrhea in Asia has been studied among peace-corps volunteers in Thailand, Japanese travelers, foreign residents in Bangladesh, guests in hotels, and members of various tour groups. Rates of diarrheal attack of >50% during 4 to 6-week trips were reported for these groups. Among travelers with diarrhea, the most commonly isolated pathogen was enterotoxigenic Escherichia coli (20-34%), followed by Salmonella (11-15%), Shigella (4-7%), Campylobacter (2-5%), and Vibrio parahaemolyticus (1-13%). In 9-22% of diarrheal episodes, multiple pathogens were recovered. Among Japanese travelers, Salmonella was more commonly acquired in the Far East; Shigella and Campylobacter in the Indian subcontinent; and V. parahaemolyticus in South-east Asia. Aeromonas hydrophila and Plesiomonas shigelloides were commonly isolated from ill travelers in Thailand but less frequently from other travelers. Protozoa and Vibrio species other than V. parahaemolyticus were isolated in <5% of episodes. (Modified authors' abstract)

Taylor DN see Blaser MI

Taylor DN see Echeverria P

Taylor JA see Di Joseph JF

Tedesco FJ. Pseudomembranous colitis: pathogenesis and therapy. Med Clin North Am 1982 May;66 (3):655-64

Tedesco FJ see Fortson WC

Tharavanij S. Pathogenesis of diarrhoea caused by parasites. Southeast Asian J Trop Med Public Health 1982 Sep;13(3):331-8

This paper attempts to summarize the state of knowledge about the pathogenic

mechanisms of parasitic diarrheal agents, namely Entamoeba histolytica, Giardia lamblia, Balantidium coli, Isospora belli, Strongyloides stercoralis, Capillaria philippinensis and Trichuris trichiura. For each organism, the basic knowledge of pathogenicity is outlined.

Thimann KV. The life of bacteria: their growth, metabolism, and relationships. 2d ed. New York: MacMillan, 1963:258-88

Thompson JS, Rikkers LF. Surgical alternatives for the short bowel syndrome. Am J Gastroenterol 1987 Feb;82(2):97-106

The goals of surgical therapy in the short bowel syndrome are to slow intestinal transit, increase the area of absorption, and reduce gastric hyperacidity. Antiperistaltic segments or colon interposition benefit patients with sufficient absorptive area. Recirculating loops are associated with prohibitive morbidity and mortality. Experience with intestinal pacing is limited. Patients with dilated bowel segments may benefit from intestinal tapering or lengthening. Growing neomucosa holds promise but has not been evaluated clinically. Despite recent advances in immunosuppression, results of transplantation remain unsatisfactory. Gastric hyperacidity can be affectively controlled by H2 receptor antagonists. None of the operations for treatment of the short bowel syndrome is sufficiently safe and effective to recommend them for routine use. Operations should be performed only on selected patients to achieve specific goals. Although investigation continues, emphasis should continue to be prevention of intestinal resection conservation of as much of the intestine as possible when massive resection is necessary. (Modified authors' abstract)

Thompson LU see Jenkins DJA

Thompson WG. The irritable bowel. Gut 1984 Mar;25(3):305-20

The irritable bowel syndrome and its variants appears to affect many persons, but most sufferers do not see a doctor. This review discusses this bowel syndrome stressing developments which have occurred since 1978. Progress in the understanding of this disease is hampered by imprecise definitions, and the lack of pathophysiologic markers. There is evidence of abnormal gut motility and myoelectric activity, and a suggestion that nerves and hormones play an important role. Apparently, there is no simple gut hormone profile that will help in identifying irritable bowel syndrome patients. The endocrine responses should be correlated with other physiologic features, such as motility and myoelectric activity. Fiber deficiency is not the cause of irritable bowel syndrome, but may be a contributing factor in many cases. Bran occasionally increases symptoms of irritable bowel disease. Observations suggest that a low-fat, high-protein diet may benefit irritable bowel syndrome patients whose pain predictably follow meals. Drugs, such as antacids, antibiotics, beta blockers, and narcotics, have well-known adverse effects on the gut which trigger the irritable bowel syndrome. Physicians should precisely and positively diagnose the syndrome, so that he may explain and reassure. The disease is a great problem to doctors and patients, because many of its symptoms might indicate serious pathology, such as inflammatory bowel disease or cancer. It is a common experience that lasting cures are unusual. short-term therapeutic response to a placebo is very high, and no diet or medication consistently outperforms it. Bulking agents seem safe and are probably most effective when constipation is present. Peppermint oil shows

some promise, and anticholinergics may be tried in persistent post-prandial symptoms. It is likely that the psychic or neural interactions that lie behind the irritable bowel syndromes are complex that no drug can be expected to have a lasting, broad-spectrum effect. It is most important, in this life-long condition, that the risks of investigation and treatment do not exceed those of the disease.

Thoren A. The role of enteropathogenic \underline{E} , \underline{coli} in infantile diarrhoea: aspects on bacteriology, epidemiology and therapy. Scand J Infect Dis 1983; (suppl 37):1-51

Thorne GM. Gastrointestinal infections--dietary interactions. J Am Coll Nutr 1986;5(5):487-99

"Considerable progress has been made in understanding the complexities involved in the production of bacterial diarrheal diseases. The general mechanisms of disease that have been recognized include enterotoxigenicity, enteroadherence, and invasiveness. The interplay of epithelial cell surface receptors with the surface components of the various bacterial pathogens or their toxins will be reviewed. Knowledge of the stereospecific interactions of bacterial ligands with the eukaryotic receptors has led to the development of new strategies for prevention and therapy. The presence of foodstuffs in the intestinal lumen can contribute by a number of mechanisms to interference with the invading organism's attack on the intestinal cell surfaces. The effects of milk fat and plant lectins on the colonization of the bowel by enteric organisms is discussed." (Author's abstract)

Thorson SM see Guerrant RL

Tiangco-Torres N <u>see</u> Mabilangan LM

Tison DL, Kelly MT. Vibrio species of medical importance. Diagn Microbiol Infect Dis 1984 Sep; 2(4):263-76

The genus Vibrio is comprised of motile, Gram-negative, straight or curved rod-shaped bacteria 0.5 to 0.8 µm wide and 1.5 to 3.0 µm long. This yeaus has undergone many changes in recent years with the addition of many species of medical importance. Presently, 10 of the 25 currently-described species in the genus Vibrio have been associated with human diseases ranging from eye, ear, and wound infections, to mild or severe gastroenteritis, and to septicemia. The importance of Ol and non-Ol serovars of <u>Vibrio cholerae</u> in the United States has recently become apparent, and several marine species, in addition to \underline{V} . parahaemolyticus, have been recognized as human pathogens. \underline{V} . fluvialis, \underline{V} . furnisii, V. hollisae, and V. mimicus have recently been implicated as agents The clinical features, pathogenesis, epidemiology, and of gastroenteritis. laboratory diagnosis of diseases, produced by the genus Vibrio, have been discussed. Environmental sources and seafood are unusually important in the acquisition of <u>Vibrio</u> infections and play a prominent role in the epidemiology of <u>Vibrio</u>-associated diseases. Pathogenic <u>Vibrio</u> species can readily be isolated from clinical specimens on media routinely used in the clinical laboratory. Variability in susceptibility to antimicrobial agents among Vibrio isolates is sufficient to warrant antimicrobial susceptibility testing when antimicrobial therapy is indicated. It has been anticipated that Vibrio species will increasingly be recognized as significant human pathogens, and it is expected that additional new species with pathogenic potential in causing disease in humans will be recognized in the near future. Increased awareness of vibrios on the part of laboratory, public health, and medical personnel will be required for better diagnosis, control, and therapy of infections due to these organisms.

Tome FC see Levine MM

Tontisirin K, Valyasevi A. Protein energy malnutrition related to diarrhea in Thai children. J Nutr Sci Vitaminol (Tokyo) 1981;27(6):513-30

In 1981, Thailand had a population of approximately 47 million, of which 80--85%were living in rural areas and 41% were children aged under 14. A survey was directed towards the identification of protein-energy malnutrition (PEM), which was a major public health problem, affecting the physical growth, mental development, learning ability and immune response to infections of Thai infants It was found that, for some infants, PEM had already been and children. occurring during the intrauterine period. The average birth weights of male and female newborn infants in different hospitals of Bangkok were compared to those in the rural village of a relatively poor province. The average birth weight and length of newborn infants born in two hospitals of the province were lower than that of infants born at the hospitals of Bangkok, but higher than that of infants born in the rural villages. Although breast feeding was recognized as the best and most proper method of infant feeding, it was found that the practice of breast feeding was highest in the rural areas and had been declining constantly during the first one and a half years of life from the rural villages to the semi-rural and urban areas of Bangkok. In the urban slum area, the prevalence of PEM, based on weight-for-age, was strikingly high among infants aged under 6 months, and among the preschool children throughout the country; PEM was prevalent in 56%. The causes were identified as poor maternal health and nutrition, false food beliefs and practices, too early introduction of supplementary foods to infants, diets low in protein and fat, unhygienic preparation and handling of food, and intercurrent infections and infestations, such as diarrhea, pneumonia, and intestinal parasites. It was also shown that diarrheal disease had been for many years the first or second leading cause of death in Thai infants. In the pediatric units of most hospitals of the country, diarrheal disease in infants has accounted for 20-35% of the hospital admissions and was responsible for 20-30% of the total deaths in this age group. The interrelationship between PEM and diarrhea in infants and children was well recognized as the most critical health problem of the developing countries, and attempts to improve the nutritional status of infants and children by alleviating PEM and diarrhea were suggested.

Topalian SL, Ziegler MM. Necrotizing enterocolitis: a review of animal models. J Surg Res 1984 Oct;37(4):320-36

Torres-Medina A, Schlafer DH, Mebus CA. Rotaviral and coronaviral diarrhea. Vet Clin North Am (Food Anim Pract) 1985 Nov;1(3):471-93

"A number of different viruses can be primary pathogens in the neonatal calf diarrhea complex. By far the most common viruses causing calfhood diarrhea found throughout the world are rotaviruses and coronaviruses. Primary infection of newborn calves with either one of these viruses can cause severe intestinal alterations and diarrhea. Rotaviruses can produce high-morbidity outbreaks of diarrhea in calves under 10 days of age. Mortality is variable mainly owing to secondary bacterial infections and electrolyte imbalances.

Rotavirus infection of the small intestinal mucosa leads to loss of enterocytes of the upper third of the intestinal villi with subsequent villous atrophy and malabsorption. There is growing evidence that different rotavirus serotypes of different pathogenicity Coronavirus infections can exist. high-morbidity outbreaks of diarrhea in calves under 20 days of age, with variable mortality due to secondary complications. Coronaviruses affect not only the small intestinal mucosa, producing significant villous atrophy, but also the colon, causing a very severe intestinal damage that can lead to death due to subsequent electrolyte disturbances. All coronaviruses, associated with neonatal calf diarrhea, appear to be of the same serotype. The etiologic diagnosis of viral diarrheas of calves requires the support of the laboratory. One of the most useful diagnostic methods is the examination of fecal extracts of virus particles by electron microscopy. for the presence antigen-detection procedures like enzyme immunoassays have been found to be useful in the diagnosis of rotaviral diarrheas. The sample of choice for these diagnostic tests is a fresh fecal sample collected directly from the calf as close as possible to the onset of diarrhea. Samples from more than one calf during the outbreaks enhance the laboratory ability to establish a proper viral diagnosis." (Authors' abstract)

Toucas M see Sansonetti PJ

Townley RRW. The management of chronic or recurrent diarrhoea in childhood. Postgrad Med J 1969 Feb;45:135-46

Based on experiences with patients of predominantly Caucasian stock drawn from communities with good living standards and staying in a temperate climate, an approach to the management of diarrhea in childhood has been outlined. Since the causes of diarrhea are so diverse, a comprehensive clinical history is always required, but certain aspects of the history need to be given special emphasis. The patient's growth progress deserves careful scrutiny. During clinical examination, special attention should be given to the patient's abdomen, and the clinician should inspect one or more of the patient's stools. When the clinical features are sufficiently characteristic of a disease entity to allow a confident provisional diagnosis, then the investigatory procedures that will most definitively confirm that diagnosis should be carried out without delay. Tests, such as microscopy, hematology, stool culture, tests for stool-reducing substances, and stool pH, have been recommended. More complex investigatory procedures including the tests for intestinal absorption, radiological tests, small intestinal biopsy, the sweat test and pancreatic enzyme estimations have also been discussed. The diagnosis and treatment of some of the clinical syndromes that may cause diarrhea have been outlined.

Trissl D. Immunology of <u>Entamoeba histolytica</u> in human and animal hosts. Rev Infect Dis 1982 Nov-Dec:4(6):1154-84

"Although Entamoeba histolytica induces humoral and cellular immune responses in both human and animal hosts, there is no indication of post-infection immunity in humans; in contrast, several other mammals are protected by prior infection or immunization. The exacerbation of the disease by immunosuppression suggests a protective function of still-unknown defense mechanisms. Specific local and circulating antibodies are produced regularly during invasive amebiasis. Although serum antibodies, together with complement, are lytic to the trophozoites in vitro, the poor correlation of these antibodies with resistance contradicts a protective capacity in vivo-

The parasite may evade harm by shedding antigen-antibody complexes from its surface. Demonstration of immediate-type skin reactions, elevated IgE titers, and specific antiamebic IgE suggests that anaphylaxis occurs. The function of the anaphylactic reaction in pathology and resistance remains to be studied. Delayed hypersensitivity parallels healing or resistance and is retarded in human hepatic amediasis. This observation is consistent with a protective role of cell-mediated immunity." (Author's abstract)

Trnka YM, Lamont JT. Clostridium difficile colitis. Adv Intern Med 1984;29: 85-107

Before 1970s, Clostridium difficile was little more than a curiosity. This pathogen is now recognized as the most important cause of antibiotic-associated colitis, and recently, it has become one of the 'commonly diagnosed intestinal pathogens in hospital practice. This pathogen has also become one of the commonest pathogens of the lower intestinal tract. objectives of this paper are to: (1) describe the organism and its toxins. (2) outline the clinical features and treatment of C. difficile-associated enteritis, and (3) review the epidemiology and pathophysiology of infection. The organism appears unique in that infection occurs during or after antibiotic therapy, suggesting that some component of the normal microflora prevents colonization by C. difficile. Once it has overgrown in the colon, C. difficile releases, several toxins which cause tissue damage and diarrhea. Infection can range from a simple self-limited diarrheal illness to fulminant colitis with perforation and megacolon. Assay of stool filtrates reveals the presence of cytotoxin in nearly all patients with antibiotic-associated pseudomembranous colitis, and in approximately one-third to one-half of those with less severe infections. Effective therapy is available in the form of oral vancomycin, although the expense of this antibiotic has led to the use of metronidazole or bacitracin, which appear to be equally efficacious and considerably cheaper. Although a great deal about <u>C. difficile</u> in the past decade has been learned, a number of fascinating puzzles remain. Very little is known about the immune response to this organism or its toxin, or whether a vaccine might someday be feasible. Similarly, very little is also known about what effects antibodies exert on the normal colonic flora and how these effects allow C. difficile infection in a small percentage of patients. It is concluded that studies of this pathogen will undoubtedly lead to a fuller understanding of the enormously complex and still mysterious microbial ferment which lives within our gastrointestinal tract.

Trust TJ, Chipman DC. Clinical involvement of <u>Aeromonas hydrophila</u>. Can Med Assoc J 1979 Apr 21;120(8):942-6

"Aeromonas hydrophila has for some time been regarded as an opportunistic pathogen in hosts with impaired local or general defence mechanisms. Infections in such individuals are generally severe. The organism is also being isolated with increasing frequency throughout the world from a variety of focal and systemic infections of varying severity in persons who are apparently immunologically normal. Most commonly it causes acute diarrheal disease by producing an enterotoxin. Thus the organism appears to have greater clinical significance than what was hitherto suspected. The organism has been infrequently reported from humans in Canada, but its correct laboratory identification, together with increased awareness that it can contribute to illness, will undoubtedly lead to more reports of its isolation in Canada." (Authors' abstract)

Trust TJ see Walker RI

Tucker H, Schuster MM. Irritable bowel syndrome: newer pathophysiologic concepts. Adv Intern Med 1982;27:183-204

Tulloch J, Burton P. Global access to oral rehydration salts and use of oral rehydration therapy. World Health Stat Q 1987;40(2):110-5

Turkish J see Kinoti SN

Turnberg LA. Antisecretory activity of opiates in vitro and in vivo in man. Scand J Gastroenterol 1983;18(suppl 84):79-83

"Both in vitro and in vivo studies of the influence of opiates on ion transport and of their antisecretory activity are described. Morphine and the synthetic opioid, loperamide, are included particularly. It is clear that the opiates have an antisecretory effect and can inhibit secretion induced by a variety of different secretagogues. Secretion induced experimentally in the human jejunum in vivo is reduced by loperamide, indicating a potential role for such an agent in the treatment of secretory diarrhoeas. (Author's abstract)

Turnberg LA. The pathophysiology of diarrhoea. Clin Gastroenterol 1979 Sep;8 (3):551-68

Turnberg LA <u>see</u> Moriarty KJ

Turnbull PCB. Food poisoning with special reference to <u>Salmonella</u> - its epidemiology, pathogenesis and control. Clin Gastroenterol 1979 Sep;8(3): 665-714

Turner AC. Travellers' diarrhoea: prevention by chemoprophylaxis. Scand J Gastroenterol 1983;18(suppl 84):107-10

"The chemoprophylaxis of travellers' diarrhoea by iodochlorhydroxyquinoline combinations, neomycin and phthalsulphathiazole, furazolidone, bismuth salicylates and streptotriad is reviewed. Streptotriad is regarded as a highly effective prophylactic against travellers' diarrhoea. Some elementary food hygiene precautions are detailed." (Author's abstract)

Turner JA. Giardiasis and infections with $\underline{\text{Dientamoeba}}$ $\underline{\text{fragilis}}$. Pediatr Clin North Am 1985 Aug; 32(4):865-80

Infection with <u>Giardia</u> <u>lamblia</u>, a flagellated protozoan, follows the ingestion of food or <u>water</u> <u>contaminated</u> with feces containing cysts or by person-to-person contact. The worldwide prevalence of giardiasis is related to levels of sanitation and the source and management of water supplies. Infants aged under 12 months are less likely to be infected than are older children. Waterborne giardiasis is common among campers who drink untreated surface water, and epidemics result when municipal water supplies become contaminated with <u>Giardia</u> cysts. Giardiasis is a cause of traveler's diarrhea; the infection is usually acquired in areas where there is inadequate sanitation and low standards of hygiene. Achlorhydria, hypochlorhydria, and hypoglobulinemic states are often associated with severe, symptomatic giardiasis. The mechanisms involved in the production of disease by <u>G. lamblia</u> are not clearly understood. Patients with immunoglobulin deficiencies are much more likely to

have morphologic abnormalities of the intestinal mucosa than are immunologically normal individuals. Standard techniques have failed demonstrate toxin from Giardia; however, the parasites have been shown to cause a toxic effect on fibroblasts in tissue cultures. Epidemiologic studies show that the parasite appears in the stool usually within 2 to 3 weeks after exposure. In endemic giardiasis, the majority of infections are asymptomatic. Waterborne outbreaks and infections in travelers are more often associated with significant illness. Symptoms include loose, frequent, foul-smelling stools, which may be watery, pale, or greasy in appearance. Maladabdominal cramps, distention, or epigastric pain may occur. Malaise, fatigue, Flatulence. belching, anorexia, and nausea are also common. Acute giardiasis may resolve spontaneously within 4 to 6 weeks. Chronic giardiasis may contribute to protein-energy malnutrition in children. Some degree of protective immunity develops in humans following infection. Humoral antibody is an important defense mechanism against symptomatic giardiasis. Examinations of feces and duodenal contents, and duodenal biopsy are the generally available diagnostic tests. In the treatment of giardiasis, furazolidone has an efficacy of about 80% compared with 90% or better for either quinacrine or metronidazole. Quinacrine is considered the first choice based, however, on economics and the inadequate knowledge of its long-term safety. Dientamoeba fragilis seems to be transmitted by a direct fecal-oral route under conditions of poor hygiene and sanitation. Symptoms commonly associated with D. fragilis infection are abdominal pain, diarrhea, anorexia, nausea, vomiting, and flatulence. Diagnosis depends on the demonstration of the parasite in the lodoquinol appears to be the drug of choice.

Tzipori S. Cryptosporidiosis in animals and humans. Microbiol Rev 1983 Mar; 47(1):84-96

Cryptosporidium is a protozoan parasite (family Cryptospiridiidae) which completes its life cycle on intestinal and respiratory surface epitheliums of mammals, birds, and reptiles. The infection, until recently, was thought to be uncommon and the organism was thought to be opportunistic and, like other coccidia, highly host specific. Cryptosporidium was first recognized in the gastric ylands of the laboratory mouse by Tyzzer in 1907. Up to 1975, some 15 reports describing the infection in 8 species of animals were published; only 5 of them associated cryptosporidiosis with some illness, 3 of which were in calves. Since 1975, over 60 scientific publications have appeared, the majority being after 1980. During this period, the concept ofcryptosporidiosis was transformed from that of a rare and largely asymptomatic infection to an important cause of enterocolitis and diarrhea in several species, including humans. In this communication, an attempt was made to gather the available information on the organism, to examine the nature of the infection it produces, and to demonstrate that Cryptosporidium infection can, under certain circumstances, cause serious disease in some species of animals. Cryptosporidium has only recently been shown to be an important cause of enterocolitis and diarrhea in a number of mammalian species. The disease in humans was initially reported to occur in immunologically individuals, but a recent study among hospital patients with gastroenteritis indicated that the infection is common in immunologically normal patients. The organism lacks host specificity, a characteristic uncommon among other enteric coccidia, and is, therefore, a potential zoonosis. It is extremely resistant to the action of common laboratory disinfectants and to antimicrobial agents tested so far. A preliminary serological survey indicates that the infection is prevalent among and within populations of 10 mammalian species examined.

Field outbreaks of diarrhea attributed to <u>Cryptosporidium</u> infection have so far been reported in calves, lambs, deer, and goats, and the disease had been reproduced experimentally in lambs, calves, and piglets. Sporadic cases of illness in other species have also been reported, and in birds, it has been shown to cause upper respiratory tract infection. The organism infects the entire bowel but most commonly the lower small intestine, where extensive mucosal changes occur. The organism completes its life cycle on the mucosal lining by adhering to the brush border of enterocytes. <u>Cryptosporidium</u> causes partial atrophy, fusion, and distortion of villi, resulting in maldigestion in the brush border and malabsorption.

Ungar BLP see Fayer R

Urrutia JJ see Mata L

Valentini V see Giorgi PL

Valvasevi A see Rohde JE

Valvasevi A see Tontisirin K

van Den Bossche H see Janssen PAJ

van Den Hurk J see Kurstak E

van Heyningen S. Cholera toxin: review. Biosci Rep 1982;2:135-46

This review elucidates the rationale of why cholera toxin is now so widely studied; how it brings together many currently interesting areas of research, such as membrane receptors and transport, subunit interaction in proteins. and mechanism of hormone action; and how investigation of this prokaryotic protein may help in understanding the normal biochemistry of eukaryotic cells. toxin is a simple protein secreted into its growth medium by Vibrio cholerae. This toxin activates adenylate cyclase in the cells to which it binds, and is the sole cause of the disease. Empirical findings show that ganglioside GMl is the receptor for cholera toxin. Binding of the ganglioside to the toxin also produces conformational changes that are specific to ganglioside GM1. general theory of the action of the toxin is that the toxin binds to the cells throughout the interaction of ganglioside and B-subunit; this is followed by the penetration of some or all of A-subunit into the cell, where it can activate the cyclase. The mechanism of entry of the Al peptide (active part of the toxin) into the cell and the activation of adenylate cyclase by the Al pentide have been discussed. The mechanism of action of cholera toxin is similar to that of several other compounds. There are two chief areas where component action, and the inactivation true: the two ADP-ribosylation. The exotoxin, produced by some strains of Escherichia coli, is almost identical to cholera toxin. Like cholera toxin, the diphtheria toxin catalyses ADP-ribosylation. The protein structure of thyrotrophin is similar to that of cholera toxin: the subunit structure is the same and there may be However, it is clear that the toxin similarities in the amino acid sequence. and the hormone do not work in the same manner. Thyrotrophin works not by directly catalysing ADP-ribosylation as cholera toxin does, but rather by increasing the activity of an endogenous enzyme system.

Vantrappen G, Geboes K, Ponette E. <u>Yersinia</u> enteritis. Med Clin North Am 1982 May:66(3):639-53

Yersinia enterocolitica, previously called <u>Bacterium</u> enterocoliticum Pasteurella pseudotuberculosis type b or Pasteurella X, is a Gram-negative of the genus Yersinia. This genus comprises a fish pathogen, Y. ruckeri, and 3 species pathogenic for man. Y. pestis, the plague bacterium, causes adenitis, Y. pseudotuberculosis causes mesenteric adenitis, erythema nodosum, polyarthritis, and sepsis. Y. enterocolitica has been known for many years to cause zoonoses in several animal species. the past few years, it has been recognized with increasing frequency as a human pathogen in Scandinavia, Continental Europe, South Africa, Canada, Japan, Australia. Although several cases and two outbreaks have been reported in the USA, diseases commonly associated with \underline{Y} , enterocolitica infections seem to occur infrequently in the United States. The clinical manifestations of Yersinia infections in man are variable. Y. enterocolitica is a common cause of gastroenteritis in children, and in adults, it produces enterocolitis, a pseudoappendicitis syndrome, arthritis, erythema nodosum, and sepsis. \underline{Y} . enterocolitica has been shown to be a fairly common human pathogen in many countries. The clinical picture produced by Y. enterocolitica infections is quite variable. An acute abdominal disease (acute gastroenteritis or colitis, or a pseudoappendicitis due to acute terminal ileitis) and, less commonly, erythema nodosum and arthritis are the most important manifestations of the disease. On radiologic examination, mucosal lesions of the terminal ileum are found in most patients with gastrointestinal symptoms. The colon is less frequently involved. The most typical lesions consist of shallow, small, round ulcers characteristic of the disease. Microscopic examination may suggest yersiniosis, but does not show pathogenic signs. \underline{Y} . enterocolitica can be detected by stool cultures or by serologic examinations. usually mild. If specific therapy is indicated, the disease usually responds

van Zijl WJ. Studies on diarrhoeal diseases in seven countries by the WHO Diarrhoeal Diseases Advisory Team. Bull WHO 1966;35(2):249-61

Major findings from studies on diarrheal diseases done in 7 countries and the association of the ready availability of water and other related factors, like modern sanitation, with their incidence, have been demonstrated. The study was carried out by the World Health Organization's Diarrhoeal Diseases Advisory Team in Sri Lanka, Banyladesh, Iran, Mauritius, Sudan, the United Arab Republic, and Venezuela. The child population was examined in communities of various countries who differed in such respects socioeconomic standards, sanitation, personal habits and in many other ways. Sanitation systems were inadequate and remained below the level at which the incidence of diarrhea cannot be controlled in many countries. Sudan had only basic sanitation. In Sri Lanka, the water-supplied regions had better sanitation. In the water-supplied areas, the reported rates for diarrhea were always lower than those without a piped-water supply. Similar reductions in the rates of detection of <u>Shigella</u> had been observed in all countries. Lanka, Bangladesh, and Venezuela showed some similarity in the rates of infestation with helminths. Sri Lanka was free of protozoa. In Iran, 70% of the children were free of helminths. Iran had the highest infestation rate of protozoa, while the rates in Bangladesh and Venezuela were similar. spp. were the most common enteric pathogen. Shigella from Iran contained larger number of resistant strains than those reported from Venezuela. In Sri Lanka, the action of sulfa drugs and antibiotics was found to be limited within hospitalized patients only. Diarrheal diseases were more difficult to combat than other diseases among preschool children aged under 6. In Sri Lanka, 10%

of the hospital beds were occupied by diarrheal patients. Errors introduced during the survey are highlighted. Nutritional status of diarrheal patients in Venezuela was also studied. Studies on local statistics, health education surveys and surveys on pediatric aspects were also carried out.

Varis K. Psychosomatic factors in gastrointestinal disorders. Ann Clin Res 1987:19(2):135-42

Gastrointestinal functions, such as food intake, propulsive peristalsis, inhibition of reflux, secretion, digestion and defecation, are controlled by a complex autonomous neurohumoral system, which is influenced by higher cortical impulses. Life stress may modulate these impulses and in this way cause two types of gastrointestinal reactions. Psychophysiological reactions involve accentuations, inhibition or distortion of the pattern of function of gastrointestinal organs without changes in their structure. Examples of this type of reaction are often painful accentuation of bowel movements in patients with the irritable bowel syndrome, and increased gastric secretion elicited by emotional stress. Psychosomatic reactions lead to morphological changes in the e.g. activation of peptic ulcer or ulcerative Psychophysiological reactions may be important in the onset of symptoms in some functional diseases, e.g. in the irritable bowel syndrome. These patients need support from the physician, but specific psychiatric therapy is required only in cases with severe psychopathology, e.g. in patients with anorexia nervosa. The role of psychosomatic reactions in the development gastrointestinal diseases is still unclear, as is the value of specific psychiatric therapy in the treatment of diseases, such as peptic ulcer or ulcerative colitis. If this kind of therapy has some effect, it may be directed mainly towards subjective symptoms. Emotional stress psychophysiological reactions, such as elevation of blood pressure and pulse rate as well as activation of gastric secretion and bowel movements. Psychophysiological reactions, which are caused by emotional stress and modulated by psychic mechanisms, may be important in the onset of symptoms in some functional diseases, as in the irritable bowel syndrome. Accordingly, these patients need special support from the physician. However, specific psychiatric therapy is required only in cases with severe psychopathology, e.g. in patients with anorexia nervosa. The role of psychophysiological reactions in the etiology and pathogenesis of organic gastrointestinal disease is poorly understood at present. Emotional stress induces gastric hypersecretion as a psychophysiological response, but the role of the hypersecretion in the etiology of ulcer disease is unclear. There is evidence that psychotherapy may have a beneficial effect on wicer symptoms, but its effect on the healing of the ulcer crater is not known.

Vesikari T. Progress in rotavirus vaccination. Pediatr Infect Dis 1985 Nov-Dec;4(6):612-4

Vesikari T see Elzouki AY

von Graevenitz A. <u>Aeromonas and Plesiomonas</u> as agents of diarrhea. <u>In</u>: Ellner PD, ed. Infectious diarrheal diseases; current concepts and laboratory procedures. New York: Marcel, 1984:59-75

von Graevenitz A. <u>Aeromonas</u> and <u>Plesiomonas</u>. <u>In</u>: Lennette EH, Balows A, Hausler WJ, Jr., Truant JP, eds. <u>Manual of clinical microbiology</u>. 3d ed. Washington, D.C.: American Society for Microbiology, 1980:220-5

von Graevenitz A, Mensch AH. The genus <u>Aeromonas</u> in human bacteriology: report of 30 cases and review of the literature. N Engl J Med 1968 Feb;278 (5):245-9

Between July 1, 1965 and June 30, 1967, 30 strains of Aeromonas and Plesiomonas were cultured from human specimens, and 28 of the 30 strains were identified as Aeromonas hydrophila and 2 as A. (Plesiomonas) shigelloides. Fourteen of the strains were clearly of gastrointestinal origin, 8 from stools and 2 from sinus tracts. Other isolates were from bile, nose, blood, pyometra, and urine. Only 0.7% of the 4,426 children, aged under 2, were carrying A. hydrophila, only one child (among 756), aged under 6, carried A. (Plesiomonas) shigelloides A small percentage of the healthy population carried the in the intestine. organisms in the intestine. The case reports suggest a connection between diarrheal disease and the large number of aeromonads in the stool. In 19 of the present cases, they were found together with other pathogens. Their role in hospital infections is still unclear. All strains tested in vitro were sensitive to chloramphenical, kanamycin, nitrofurantoin, and nalidixic acid and were resistant to ampicillin. Most strains were also sensitive tetracycline, streptomycin, and colimycin. Sensitivity to erythromycin and cephalothin was erratic. The results of the sensitivity tests on A. hydrophila agree with those of other studies. No strains sensitive to penicillin or ampicillin have ever been found.

Wadstrom T. Aeromonas and Plesiomonas as possible causes of diarrhoea. Third Meeting of the Scientific Working Group on Bacterial Enteric Infections, Geneva, 12-14 Sep 1984. Geneva: World Health Organization, 1984. 10 p. (Unpublished document)

Wadstrom T, Baloda SB, Yuk YR. Cytotoxic and cytolytic proteins of enteropathogenic Escherichia coli and Salmonella: new concepts on possible role in intestinal colonization. Zentralbl Bakteriol Mikrobiol Hyg [A] 1986;(suppl 15):153-60

Wadstrom T, Baloda SB. Molecular aspects on small bowel colonization by enterotoxigenic Escherichia coli. Microecol Ther 1986;16:243-55

Wadstrom T see Ljungh A

Wagh MG, Ghooi RB, Shetty RK. Lactose intolerance; physiological, clinical and therapeutic considerations. Indian J Pediatr 1984 Nov-Dec;51(413):671-81

Wahed MA see Rahaman MM

Wakelin D. Genetic and other constraints on resistance to infection with gastrointestinal nematodes. Trans R Soc Trop Med Hyg 1986;80(5):742-7

"Infections with gastrointestinal (GI) nematodes are among the most prevalent infections of man. Not only are they common and widespread throughout the world, but they are frequently chronic and occur repeatedly throughout the lifetime of an individual. It is now well established that such parasites can be highly immunogenic and that their environment, the gastrointestinal tract, is well equipped to mount potent immune and inflammatory responses. The abundance and long-term survival of GI nematodes, therefore, present a paradox. This review takes the standpoint that man as a species has the capacity to produce effective and protective responses against GI infections, but that this capacity is subject to a number of powerful constraints, arising from parasite

evasion, depression of response capacity and deficiency in response capacity. These constraints are discussed in the light of evidence drawn from experimental studies with animal models." (Author's abstract)

Walker RI, Caldwell MB, Lee EC, Guerry P, Trust TJ, Ruiz-Palacios GM. Pathophysiology of <u>Campylobacter</u> enteritis. Microbiol Rev 1986 Mar;50(1): 81-94

This review presents an overview of the progress that has been made in recent years toward an understanding of the pathogenesis of Campylobacter enteritis. Campylobacter jejuni is the species most frequently isolated from patients in most geographic areas. The organism is characterized by a rapidly darting motility due to one or more amphitrichous flagella. <u>C. jejuni</u> grows best in microaerobic environments at 42°C. Various aspects of establishment of infection, e.g. infective dose, motility and chemotaxis, and adherence, have been discussed. Disease due to <u>C. Jejuni</u> infection is most gastrointestinal in nature. However, extraintestinal infections, including meningitis, cholecystitis, and urinary tract infection, have been reported. The incubation period varies (I to 7 days), and the diarrhea is usually self-limited, lasting 2 to 7 days. The recent demonstration of enterotoxin production by C. jejuni suggests that this may be the underlying mechanism in pathogenesis. In Campylobacter enteritis, clinical evidence also exists for intestinal epithelial invasion in cases with bloody diarrhea and inflammatory cells in the stool. A mechanism, termed translocation, has also been suggested. <u>C. jejuni</u> produces at least two exotoxins: a heat-labile enterotoxin and a cytotoxin. The initial process by which <u>C. jejuni</u> colonizes the intestine before appearance of disease symptoms, induced by toxins or other mechanisms, may involve flagella, lipopolysaccharide, or other structures. Immunity against C. jejuni in humans is acquired as a consequence of one or more infections. Since protective immunity against Campylobacter spp. is associated with rapid clearance of the organism from the intestine. it seems that this immunity is directed against colonization antigens. Elucidation of the importance of various components in the pathogenesis of Campylobacter infections requires the development of molecular genetic systems and simple virulence assays.

Walker WA see Cohen S

Walker WA see Lake AM

Walker WA see Lo CW

Walker-Smith JA. Nutritional management of acute gastroenteritis -- rehydration and realimentation. Hum Nutr Appl Nutr 1986;40(suppl 1):39-43

Walker-Smith JA see Elliot EJ

Walsh JA, Warren KS. Selective primary health care: an interim strategy for disease control in developing countries. Soc Sci Med 1980;14C(2):145-63

"Priorities among the infectious diseases affecting about 3 billion people in the less-developed world have been used on prevalence, morbidity, mortality and feasibility of control. With these priorities in mind a program of selective primary health care is compared with other approaches and suggested as the most cost-effective form of medical intervention in the less-developed countries. A

flexible program delivered by either fixed or mobile units might include measles and diptheria-pertussis-tetanus vaccination, treatment for febrile malaria and oral rehydration for diarrhea in children, and tetanus toxoid and encouragement of breast feeding in mothers. Other interventions might be added on the basis of regional needs and new developments. For major diseases for which control measures are inadequate, research is an inexpensive approach on the basis of cost per infected person per year." (Authors' abstract).

Walsh JA. Selective primary health care: strategies for control of disease in the developing world. IV. Measles. Rev Infect Dis 1983 Mar-Apr;5(2):330-40

Measles kills several hundred thousand infants and young children yearly. Essentially all children become infected; at least 1% of those living in developing countries die unless protected by immunization. In urban areas, peak incidence occurs in those younger than 3 years. The youngest and most undernourished children suffer the most severe complications and exhibit the highest degree of the risk of death. Diarrhea, malnutrition, pneumonia, and blindness associated with vitamin A deficiency are the worst complications. The infection is preventable by the timely administration of a potent vaccine. This endeavor requires a well-managed technical and administrative network that remains difficult to organize in many areas of the world. The vaccine is efficacious and has few adverse effects but must be provided to children during the short interval between loss of transplacentally acquired antibodies and the acquisition of natural infection. The improvements in heat stability of the vaccine has increased the likelihood of providing a potent vaccine, but a well-managed cold chain remains a prerequisite for any successful immunization program. Health education, improved management skills, publicity, and community support are all important factors for ultimately preventing the morbidity and mortality from this disease. (Modified author's abstract)

Walter M see Muller JM

Warren KS. New scientific opportunities and old obstacles in vaccine development. Proc Natl Acad Sci USA 1986 Dec;83(24):9275-7

"The present status of and priorities for vaccine development are described, and the historical conditions under which vaccines have been developed are contrasted with newer technologies for such development. Current programs, the opportunities they present, and the obstacles to their implementation are summarized." (Author's abstract)

Warren KS. Selective primary health care: strategies for control of disease in the developing world. I. Schistosomiasis. Rev Infect Dis 1982 May-Jun; 4(3):715-26

The control of schistosomiasis should be related to its unique biology and ecology. Whereas schistosomes multiply within the snail, the intermediate host, they do not replicate within the mammalian definitive host. As a consequence, a large proportion of infected humans have low or moderate worm burdens, the disease tending to occur in a small proportion of individuals harboring large numbers of worms. This situation suggests an unusual strategy: the control of schistosomal disease rather than the usual approach of control of infection and its almost invariable end point of eradication. Control of infection (transmission) requires use of mollusciciding, provision of safe water supplies and better sanitation, health education, and chemotherapy. In

spite of such measures, its total annihilation is not likely to come about. Control of disease can be achieved at far lower cost by chemotherapy alone using the newer single-dose, oral, nontoxic chemotherapeutic agents. (Modified author's abstract)

Warren KS see Chandra RK

Warren KS see Walsh JA

Wasunna A see Kinoti SN

Wauters G see Cornelis G

Weaver RE see Blake PA

Weber A. [Occurrence of Campylobacter jejuni in animals and its significance for the human]. Tierarztl Prax 1985;13(2):151-7

Weber J. Ducrotte P. Colonic motility in health and disease. Dig Dis 1987;5(1):1-12

Weinstein AJ see McHenry MC

Weiss LM see Tanowitz HB

Weiss M see Horzinek MC

Wharton BA. Food for weanling: the next priority in infant nutrition. Acta Paediatr Scand 1986; (suppl 323):96-102

Wicks J see Donowitz M

Wiess HF see Darrow DC

Williams EK, Lohr JA, Guerrant RL. Acute infectious diarrhea. II. Diagnosis, treatment and prevention. Pediatr Infect Dis 1986 Jul-Aug;5(4):458-65

Williams EK see Guerrant RL

Wittner M see Tanowitz H8

Wolever TMS see Jenkins DJA

Wolf JL, Schreiber DS. Viral gastroenteritis. Med Clin North Am 1982 May;66 (3):575-95

Wolfe MS. Diseases of travelers. Clin Symp 1984;36(2):2-32

Wolfe MS. The treatment of intestinal protozoan infections. Med Clin North Am 1982 May:66(3):707-20

Wong HB see Yip WC

Woodward WE see Pickering LK

Wood DJ. Adenovirus gastroenteritis. Br Med J 1988 Jan 23;296(6617):229-30

Wood S see Levine MM

Woode GN see Flewett TH

Woode GN see Horzinek MC

Wormann B, Hochter W, Othenjiann R. [Drug-induced colitis]. Disch Med Wochenschr 1985 Sep 27;110(39):1504-9

Wright AM see Feachem RG

Wright SG. Parasites and travellers' diarrhoea. Scand J Gastroenterol 1983; 18(suppl 84):25-9

Giardia lamblia is the most important parasitic cause of travelers' diarrhea. The parasitology of this flagellate protozoan is discussed with respect to route of infection, threshold infecting dose, and incubation period. The geographic distribution of giardiasis is widespread, being common in tropical and subtropical regions and endemic at a low level in European and North American countries. The predisposing factors are detailed together with clinical manifestations, pathogenesis, and diagnosis. Effective therapy may make use of quinacrine t.i.d. for 5-10 days, metronidazole 200 mg t.i.d. for 14 days of 2 g once daily over 3 days, or a single dose of 1.5 g of tinidazole. Although Entamoeba histolytica is distributed worldwide, it rarely causes travelers' diarrhea. The clinical features, diagnosis, and treatment of amebiasis-related travelers' diarrhea are detailed. (Modified author's abstract)

Wright V <u>see</u> Neumann V

Wu C-C <u>see</u> Chandra RK

Wu GX. [Progress of studies on yersiniosis caused by Yersinia enterocolitica]. Chung Hua Lin Hsing Ping Hsueh Tsa Chih 1984 0ct:5(5):309-13

Wyllie R see Arasu TS

Yamamoto T see Takeda Y

Yardley JH see Nime FA

Yekutiel P see Shuval HI

Yip WC, Tay JS, Ho TF, Wong HB. Total parenteral nutrition in paediatric practice. J Singapore Paediatr Soc 1984;26(1-2):59-72

Yolken RH, Leggiadro RJ. Immunoassays for the diagnosis of viral enteric pathogens. Diagn Microbiol Infect Dis 1986 Mar;4(suppl 3):S61-9

Young VR see Hoerr RA

Young CR see Levine MM

Yuk YR <u>see</u> Wadstrom T

Zhalko-Titarenko VP, Bondarenko VM, Grigoryev AV, Kupchinsky LG, Rybalko SL.

Dynamics of the interaction of <u>Shigellae</u> with the epithelium in the process of infection. Zh Mikrobiol Epidemiol Immunobiol 1986 Apr;(4):21-4.

"The study of the adhesion of Shigella flexneri to intestinal mucosal explants from human postabortion fetuses, used as an experimental model, has revealed that the process of interaction between the infective agent and the epithelium develops in accordance with Langmuir's equation of the adsorption isotherm. The specific biological feature of the adhesive interaction between bacteria and the mucous membrane is the fact that the effective adhesion of microbes is possible only in case of their high concentration on the surface of the mucous membrane. In case of their low concentration in the parietal layer, no microbial adhesion is observed, whereas epithelial villi infected with Shigellae and fixed to the mucous membrane of the explant produce a high parietal concentration of the infective agents, which leads to the increase of adhesion by more than two orders." (Authors' abstract)

Ziegler MM. Short bowel syndrome in infancy: etiology and management. Clin Perinatol 1986 Mar;13(1):163-73

Ziegler MM <u>see</u> Topalian SL

Zollinger RM. Islet cell tumors of the pancreas and the alimentary tract. Am ϑ Surg 1975 Feb;129(2):102-10

"Functioning tumors of the pancreatic islets are now recognized as the source of clinical syndromes affecting the gastrointestinal tract which have a wide variety of catastrophic symptoms. Experiences with thirty-six cases suggest at least four separate diagnostic categories in the ulcerogenic tumor syndrome. These include: a typical history, gastric analysis, and roentgenographic findings with borderline fasting serum gastrin levels; ulcerogenic tumor with evidence of hyperparathyroidism; iatrogenic ulcerogenic syndrome associated with failure of a previous operation for duodenal ulcer; and the classic ulcerogenic syndrome associated with a fulminating ulcer diathesis or diarrhea and high serum gastrin levels. The problems presented at operation include: decisions to be made in the presence of a negative exploration; the finding of a solitary tumor in the wall of the duodenum; solitary pancreatic tumors particularly in the body and tail; ulcerogenic tumors in the very young; liver metastases in the elderly; and the wisdom of removing gross metastases in combination with total gastrectomy. The long-term survival in the ulcerogenic tumor syndrome approximated 50 per cent, with 40 per cent of those having proven malignancy living 5 years. Evidence of hyperparathyroidism relatively common in association with both the ulcerogenic and diarrheagenic tumor syndromes. The association may be a result of a congenital abnormality, metabolic alkalosis, or a direct effect of the islet cell tumor. Parathyroidectomy may be indicated when both the serum calcium and parathormone levels are elevated in the presence of borderline fasting gastrin levels. latter may return to normal after parathyroidectomy. The evidence hyperparathyroidism closely parallels the episodes of diarrhea in diarrheagenic syndrome, and hyperparathyroidism may regress spontaneously after total removal of the pancreatic tumor. Just as routine calcium determinations have made the diagnosis of hyperparathyroidism more commonplace, it is suggested that the gastrointestinal syndromes associated with islet cell tumor would receive wider recognition if radioimmunoassays for gastrin as well as secretion, and the other routinely." (Author's abstr secretin-like polypeptides, were carried (Author's abstract)