THE EFFECTS OF NUTRITION ON NATURAL FERTILITY

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PREFACE

The Cholera Research Laboratory (CRL) operates under a bilateral project agreement between the government of Bangladesh and the United States of America. Research activities of CRL center on the inter-relationships between diarrheal disease, nutrition, fertility and their environmental determinants. CRL issues two types of papers: scientific reports and working papers which demonstrate the type of research activity currently in progress at CRL. The views expressed in these papers are those of authors and do not necessarily represent views of Cholera Research Laboratory. They should not be quoted without the permission of the authors.

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ABSTRACT

This paper presents an analysis of malnutrition as it effects those biological mechanisms directly related to fertility performance. Only general protein-calorie malnutrition has been considered. The evidence seems clear that malnutrition retards menarche and through this mechanism can indirectly effect the age of marriage in some societies. Malnutrition may result in earlier menopause although data is limited. Acute famine conditions, with or without frank starvation, leads to severely depressed fertility through both biological and behaviour mechanisms. Studies specifically designed to establish the relationship of chronic malnutrition to depressed fecundability or prolonged lactational amenorrhea found essentially no relationship. Fecundability seems to be primarily a function of coital frequency while lactational amenorrhea is a function of breastfeeding practices and is mediated through neurohormonal mechanisms related frequency intensity and duration of suckling. Data on malnutrition and fetal wastage is conflicting. If there is an effect it must not be great. Maternal malnutrition is clearly associated to low birth weight and thus poor infant survival. This however can result in a rise in fertility in breastfeeding populations. Overall the data available indicate that the biological effects of malnutrition cannot account for the major variations in natural fertility between different non-contraceptive population group.

Introduction

Natural fertility is a concept which has been developed to include any patterns of childbearing in a population that exist in the absence of a conscious effort to control the number of offspring by a couple.[1] This concept recognizes that there may be both behavioural and biological factors which contribute to the pattern of natural fertility. In considering the effects of nutrition on fertility, while malnutrition might be considered as simply a biological state, in fact variations in the nutritional status of a population may effect fertility through behavioural as well as biological mechanisms.

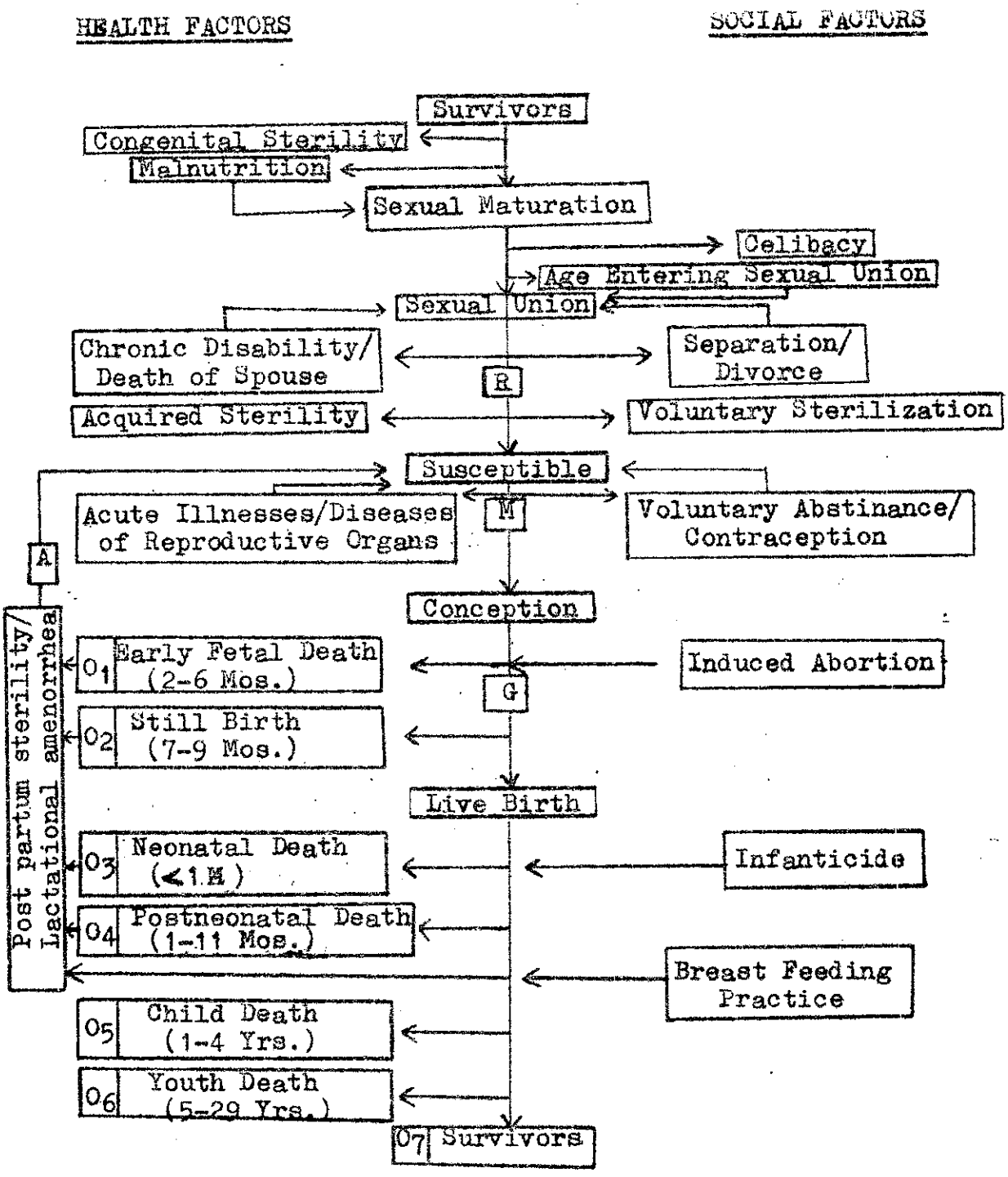
A Model of Human Reproduction

A general conceptual framework[2] useful for analyzing the effects of nutrition on fertility is depicted in Figure 1. This figure illustrates the model for reproduction in the female; the relevant factors affecting male reproduction will be discussed where appropriate.

The central line in Figure 1 identifies the successive biological steps required for a woman to produce surviving offspring. She must reach sexual maturation and enter into a sexual union. Both of these factors are required, and these determine the duration of effective reproductive life (R). With menstruation, she will presumably be susceptible to pregnancy at monthly intervals. The duration of the susceptible period, or menstruating interval (M) is determined by the number of months until a recognized conception. The period of gestation (G), if successful, terminates in a live birth which can result in surviving offspring. After a period of post-partum sterility and/or lactational amenorrhea (A), the woman re-enters the susceptible period and the reproduction cycle is maintained until the sexual union is broken or sterility intervenes. The left side of Figure 1 identifies the health factors that can affect reproductive performance; the right side identifies the social factors.

This framework is useful in a discussion of the effects of nutrition on reproduction since it forces one to specifically identify where and how in the reproductive cycle various states of malnutrition actually affect the reproductive process in a given population. More significantly, by identifying how health and social factors may interfere with the reproductive process

Figure 1



(See page 4 for caption)

CAPTION FOR FIGURE 1 (preceding page)

A model of human reproduction. The relevant biological variables are: R - duration of effective reproductive life; M - duration of the menstruating interval; G - duration of gestation; A - duration of postpartum and lactational amenorrhea; O - proportion of pregnancies with a given outcome. The health factors affecting reproduction are shown on the left side, the social/behavioural factors on the right.

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through many of the same biological mechanisms, it highlights some of the problems that are encountered in population-based studies which are attempting to determine whether variations in fertility levels are actually due to biological factors affecting the reproductive process or concomitant behavioural changes in populations with various states of malnutrition.[2]

Scope of this report

This paper will not present a comprehensive review of all work related to nutrition and fertility; rather, it will outline the areas where these are important relationships and then highlight the information and issues developed at the Conference on Nutrition and Human Reproduction organized by the Center for Population Research, National Institutes of Health in Bethesda, Maryland February 13-17 1977. The discussions will be augmented by data currently being developed by the Cholera Research Laboratory which is conducting a longitudinal population studies on the interrelationships of nutrition and fertility in rural Bangladesh.

Menarche and Menopause

Menarche and menopause demarcate the limits of potential reproductive life span in the female. Dr. Gray elsewhere in this Seminar presents a comprehensive review of many of the biological factors related to the timing of menarche and menopause.[57] The relationship of nutritional status to menarche has been postulated on the basis of three general types of observations. First there is the documented secular decline in the age of menarche in many western countries. Tanner, for example, has reviewed the literature on changing age at menarche in a number of European countries and noted that over the past century there has been a correlation of the decline in the age of onset of menarche with an increase in height and weight by age.[3]

The second line of evidence comes from observations on variations in menarche associated with socio-economic status. For example, higher ages of menarche are reported in impoverished nations where nutritional status has remained inadequate. In rural Bangladesh, Chowdhury et al reported the median age of menarche at 15.8 for Muslims and 16.0 for Hindus.[4] These data may be compared with the present age of menarche in the U.S. which is estimated at between 12 to 13 years.[5] Since socio-economic conditions are more closely related to nutritional status in developing countries, socio-economic or urban-rural differentials in menarche may also be observed. Burrell et al reported the average age of menarche for South African Bantus of 15.02 years for those classified as not poor and 15.42 for those classified as poor.[6] Madhavan estimated the mean age of menarche in Madras, India, at 12.76 years for urban areas and 14.16 years for rural areas, and similarly, in Kerala, the differentials were 13.24 for urban areas and 14.42 for rural areas.[7]

The third line of evidence relating nutritional status to menarche come from studies which have sought direct correlations. A study in Uttar Pradesh, India, reported that girls of better nutritional status who received diets higher in calories and proteins showed earlier menarche.[8] Similarly, a study of Slovian girls found that those with diets rich in protein reached menarche at 12.65 years compared to 14.1 years for those with diets composed mostly of carbohydrates.[9] Hillman reported a high correlation between present nutritional status and age of menarche for U.S. women aged 18 to 25 years.[10] In a ten year prospective study of sexual development in American girls aged 8 to 10, Zacharias et al related onset of menarche to body build.[5] They reported a significant, positive correlation between the ponderal index, (a function of height and weight) and age of menarche. Late menarche was associated with thinness and early menarche with heaviness. Dreizen et al also observed in a U.S. study that well nourished girls had an average age of menarche of 12.43 compared to 14.45 among poorly nourished girls.[11]

Chowdhury et al has recently completed a study in Bangladesh which clearly illustrates the relationships between weight for age and the onset of menarche among girls in rural Bangladesh.[4] These data, presented in Table 1, indicate that within any age-group the proportion of girls having attained menarche is strongly influenced by body weight.

Table 1

Percentage of Rural Bangladeshi Females Who Have Had Menarche By Age and Body Weight.[a]

Age	Weight (Kg.) Under 30	30-34	35-39	40+
10-13	0.0 (457)[b]	4.3 (23)	27.3 (11)	
14-15	2.9 (105)	16.9 (59)	51.7 (60)	96.0 (25)
16-17	13.5 (18)	45.5 (44)	86.6 (82)	97.6 (83)
18-20	40.0 (20)		100.0 (46)	100.0 (122)

a/Data are from a cross-section survey in Matlab Thana, Comilla District, obtained in March 1976. Source: Derived from Table 2 of Chowdhury, et al.[4]

b/Numbers in parenthesis are number of women.

Frisch has undertaken the most extensive analyses of nutrition and menarche, and has developed the hypothesis that menarche is closely related to the attainment of a critical body weight, which represents a critical fat-lean ratio or fat-body weight percentage.[12,13] The biological mechanism suggested is that the critical weight represents a particular metabolic rate which would be signalled to the hypothalamus. This alters the hypothalamic sensitivity to the gonadotropins and results in increased gonadotropin production and the initiation of menarche.[14]

Because of a less precise indicator of sexual maturation, there is little information on the effects of undernutrition in the male. Indirect evidence suggests that undernutrition probably delays sexual maturation in boys as it does in the female.[15]

With reference to the age of menopause, as Gray notes elsewhere in this Seminar, data are meager and subject to methodological limitations.[57] The lines of evidence relating nutritional status to menopause are similar to those relating nutritional status to menarche. For example, Wolanski reported a secular rise in the age of menarche among Polish women in the last century.[16] Wyon et al reported a median age of 44 years among women in the Punjab in India, in 1966.[17] This poorer population could be contrasted with the median age of menopause among American women of 49.8 years.[18]

The only study available directly relating nutritional status to menopause was reported from New Guinea where the data indicated that among malnourished women the median age of menopause was 43.6 years while among better nourished women it was 47.3 years.[19]

Malnutrition and the age of marriage

If menarche is a signal for marriage in a society, then it is reasonable to relate malnutrition to the age of marriage. Recent studies in rural Bangladesh reveal that this is the case. Table 2, from the report by Chowdhury et al, indicates that the proportion of young women married within any age group is strongly related to the attainment of menarche.[4] This relationship between malnutrition and marriage takes on added significance in the Bangladesh situation when it is noted that the 1976 survey suggests that the cohort of girls born in 1961 or later will attain menarche at least one year older than earlier cohorts apparently due to recurrent famine conditions between 1971 and 1975.

Table 2

Percentage of Rural Bangladeshi Females Who Are Married By Age And Menstrual Status[a]

	Premenarche		Postmenarche	
Age	Number	% Married	Number	% Married
10-13	486	0.2	5	27.4
14-15	181	3.3	68	
16-17	52	5.0	175	44.6
18-20	8		180	66.1

a/ Data from Chowdhury, et al.[4]

Starvation and Famine

Starvation and famine are exceptional conditions which should be considered separately from chronic states of malnutrition that are more widespread in human populations. Starvation causes drastic alterations in reproductive function. In the classical experiments by Keys, et al, starved males demonstrated successively the loss of libido, decreased sperm number, loss of sperm motility and eventually the cessation of sperm production.[20] Similarly among females under conditions of starvation, loss of libido and amenorrhea, are the distinguishing features.[21,22]

Famine is a much more complex situation.[23] While the hallmark of famine is an overall lack of food in the population, the consequences on the patterns of fertility reflect the complex interaction of both biological and social disruptions in the population. Two well documented famines in recent years highlight these complex interactions.

Stein and Susser document in some detail the Dutch famine of 1944/45.[24,25] This famine occurred acutely in an otherwise well-nourished population because of war-time conditions. There was severe food shortage from November 1944 to April 1945. There was no detectable effect of the birth-rate during the nutritional crisis, however, after a nine month lag there was a sharp decline in fertility by 50% indicating a severe depression in the number of conceptions. The famine was relieved quite quickly by the liberating armies and this was associated (after a nine month lag), with a remarkable spurt in the birth rate to almost double the pre-famine levels.

In discussing the effects of famine on fertility Stein and Susser suggest that one major effect was clearly loss of libido in the male and amenorrhea in the women.[25] There was no clear evidence of a rise in fetal wastage.

Less well explained were distinctive differences in the impact of the famine on fertility between social classes as well as by age and parity. The higher social classes had less of a depression of fertility than the lower classes. The authors speculate that this might have been because the higher classes, with their resources, money, and influence, could have obtained more food. Fertility was also less depressed in younger women and/or primiparae. While this may relate to older women being more severely

affected by malnutrition, it is also suggested that this may relate to differential changes in mating behaviour among older, higher parity women in response to famine conditions.

The Bangladesh famine of 1974/75 also had profound effects on fertility. Because of extensive severe flooding throughout Bangladesh destroying a substantial fraction of the rice crop, the price of rice began to rise in the second quarter of 1974 and peaked at over 200% of earlier levels by the first quarter of 1975. The vital registration project of the Cholera Research Laboratory documented in Matlab Thana a 50% rise in the crude death rate with deaths concentrated largely in infants and children.[26] Much more striking was the decline in fertility. The crude birth rate which had ranged from 41.8 to 47.8 per 1000 over the period 1966 to 1973 declined to 27.6 between April 1975 and March 1976.

A detailed analysis of the available data by Chowdhury and Chen suggests that there were multiple factors contributing to the fertility decline, although precise quantitation of the relative effects is not yet available.[27] The major factor appeared to be a reduction in the conception rate, due in part, no doubt, to the fear and anxiety associated with the crisis conditions. Physiological loss of libido and amenorrhea could have contributed though this is not documented. A significant contributing factor was the temporary out migration of husbands searching for work; marriages were also posponed because of the economic conditions.

There was a significant increase in induced abortions, however, there was no clear evidence of a rise in spontaneous fetal wastage. A cross-sectional sample survey of the study population near the height of the famine suggested that the duration of lactational amenorrhea was somewhat prolonged in late 1975 from levels found in earlier studies in 1968/69 (around 21 to 22 months as compared to about 18 months).[28]

Because of these social and biological interactions under famine conditions, it is difficult to know if the situation actually fits the definition of "natural" fertility. There are clearly voluntary efforts to limit fertility, however, this is because of the crisis conditions and does not relate to "the number of children already born" or the number "which the couple does not want to exceed".

Malnutrition and Birth Intervals

The effect of chronic malnutrition on birth interval dynamics is of considerable interest, not only theoretically, but also because it has policy significance for developing countries where health conditions are improving.[2] The two segments of the birth interval that have the largest effect on overall reproductive performance are the duration of lactational amenorrhea and the duration of the menstruating interval.

a. Lactational amenorrhea

Physiologists and clinicians are beginning to study intensively the biological mechanisms relating to milk production and fertility suppression during lactation. It is now well-established that lactation and post-partum amenorrhea rely heavily upon the effectiveness of the nursing stimulus, which in turn causes secretion of the pituitary hormone prolactin.[29] This hormonal reflex system has even been demonstrated in normally menstruating women who induce lactation simply by nipple stimulation.[30] The effects of prolactin on fertility are poorly understood, but evidence suggests that is modulates hypothalamic and ovarian function,[31] including suppression of luteinizing hormone releasing hormone from the hypothalamus[32] and inhibition of ovarian steroid production.[33] Further, prolactin may actually block follicle stimulating hormone activity at the ovary.[34]

There are wide variations in the duration of lactational amenorrhea among various populations which can be strongly correlated either within populations or between populations to the duration of breast-feeding.[35,36] Frisch and others have suggested that malnutrition may also be a major determinant of the prolonged states of lactational amenorrhea frequently observed among poorly nourished populations, particularly in developing countries.[37,38]

It should be stressed that research in this area is complex because of the multiple interacting factors relating maternal and child nutrition to breast feeding practices and thus to amenorrhea. For example, well-nourished mothers in the upper economic classes may tend to supplement their child earlier, thus reducing the frequency of breast-feeding.[39] Nutritional supplementation programs for mothers and children can also reduce breast-feeding practice.[56] On the other side of the picture, there is evidence that the volume of breast milk produced is related to the level of maternal nutrition;[40] thus, it may be that poorly-nourished mothers who produce less breast milk experience more prolonged and intense suckling by the hungry infant.[30] Because of these interactions, it is essential that studies seeking to relate nutritional status to lacta-

tional amenorrhea control carefully for the frequency, duration and intensity of breast feeding.

Three studies reported at the NIH Conference on Nutrition and Human Reproduction bear directly on this question. Carael contrasted the duration of lactational amenorrhea in three central African communities.[41] Two tropical forest communities with diets relatively balanced in proteins, carbohydrates and fats practiced breast feeding for an average of 16.9 and 18.7 months and reported lactational amenorrhea of 10.7 and 10.4 months respectively. The third community, in the highlands, with a high carbohydrate diet deficient in proteins, had a mean duration of nursing of 22 months and a post-partum amenorrhea of 18.7 months. Carael reported that for women with approximately equivalent durations of breast feeding the highland community had a more prolonged duration of amenorrhea. There were substantial differences in breast feeding patterns and types of infant supplementations between the three communities, however, making it impossible to draw definitive conclusions.

Delgado et al. examined the relationships of maternal nutritional status to duration of lactational amenorrhea among a group of women in rural Guatemala who were participants in a longitudinal nutritional study.[42] They were able to demonstrate a slight negative correlation ($r = -0.14$) between mother's weight and length of post-partum amenorrhea although the result was not significant for 160 cases. There was also evidence of a small reduction in the length of post-partum amenorrhea among mothers who took the largest nutritional supplementation during pregnancy. This result, however, is difficult to interpret since mothers were self-selected in use of the nutritional supplsmentation and there is no data on maternal or infant supplementation during the period of lactation.

Chowdhury presented the preliminary results of an intensive longitudinal study of 2,000 women in rural Bangladesh who had been followed for one year in 1975/76.[43] 502 women who terminated lactational amenorrhea were classfied into three nutritional categories by body weight in the month of termination: less than 38.5 kg; 38.5 to 42.4 kg; and greater than 42.4 kg. The mean months of post-partum amenorrhea for each group were 17.9, 17.5, and 16.8 respectively. This suggests that post-partum amenorrhea may have been prolonged by only about 6% among women who are poorly nourished as compared to those who are well nourished.

A similar longitudinal study is currently in progress by Hull in Indonesia.[44] Although detailed results are not yet

available, she has reported variations in lactational amenorrhea between social classes with much shorter duration among the better nourished upper classes. However, most of this variation appears to be attributed to differences in breast feeding practices.

While the results suggest that malnutrition may be a variable in prolonging the duration of lactational amenorrhea, it seems clear from the data currently available that it could not account for the major variations in fertility reported between different societies or between different social and economic groups. These variations seem to be primarily related to differences in breast feeding practices.

b. Fecundability

The effects of famine conditions and gross starvation on reproductive potential and performance are profound as noted earlier. The more fundamental question is what is the effect of chronic states of moderate protein-calorie malnutrition on fecundity.

Frisch has suggested that the effects of chronic malnutrition on fecundity is one of the important factors accounting for poor reproductive performance in many historical populations as well as in poorer areas of the developing world.[37]

There is some clinical evidence that malnutrition in women is associated with irregular menstrual cycles and a higher frequency of anovulatory cycles.[45] Until recent years however no population based data has been available selectively examining the effects of malnutrition on fecundity. Recently population studies in Bangladesh and in Indonesia have been designed to specifically examine this question. Preliminary results are currently available from Bangladesh.

In a two year prospective study of 200 women in rural Bangladesh in 1968-69 Chen et al[38] estimated that the average fecundability was 0.10. This is considerably lower than estimates reported from Western populations[48] and implies an average waiting time to conception of about ten months. In attempting to disaggregate the factors accounting for this low fecundability, two factors were found to be important. First, husbands were absent for temporary or seasonal work. Correcting for this factor Chen

et al found the average fecundability to be 0.13. Second, there was a striking seasonal variation in fecundability. During the peak months of conception, in the cool season, fecundability was 0.18 to 0.24 while during the monsoon season fecundability was 0.04 to 0.11.

If this striking seasonal variation in conception rates is primarily due to variations in coital frequency related to climatic conditions, then the high rates, which are comparable to rates well nourished populations such as the Hullerites,[46] suggest that chronic malnutrition does not impair fecundability. It is possible that seasonal variations in nutritional status are a factor in the fluctuation in fecundability. Menken, however, has examined the regularity of menstruation in this population and found no systematic monthly fluctuations.[47]

Chowdhury has conducted a preliminary analysis of the waiting time to conception by nutritional status in the 1975-76 prospective study in Bangladesh cited earlier.[43] The 419 women who conceived during the one year study period were weighed in the month of conception, and grouped in the same three weight categories noted above. The average months from first post-partum menstruation to conception were 10.0, 10.7 and 11.3 for women in the highest, middle, and lowest weight groups respectively. This apparent small effect of malnutrition on fecundability did not prove significant in a multiple regression analysis controlling height, age, parity, and husband's age.

These findings in rural Bangladesh are consistent with the results from rural Guatemala reported by Bongaarts and Delgado in this Seminar, which also showed that the live birth conception wait interval was not related to the mothers nutritional status.[58] While the data are inadequate to say definitively that chronic malnutrition does not effect fecundability, it does begin to appear that direct biological factors related to nutrition may play only a very minor role in the fertility of a population. Much more important may be the behavioural responses of the population which can be conditioned by chronic food shortage.

Nutrition and Pregnancy Outcome

Pregnancy makes major nutritional demands upon the mother; thus it might be expected that malnutrition could have major effects on fetal survival. In fact, there is little evidence for a significant effect. This may be due to methodological

problems, particularly in detecting early fetal wastage. Where comparable data is available on early fetal wastage, the rates in well nourished populations actually appear higher than in poorly nourished populations: 126 and 227 per thousand pregnancies in New York[49] and Hawaii[50] respectively may be contrasted with 107 and 134 for the Punjab and Bangladesh.[38]

Even under conditions of acute starvation, the data on fetal wastage are not clear. Anatov[52] reports a doubling of the fetal wastage in the 1942 siege of Leningrad, while Stein and Susser could not find clear evidence that an increase in fetal wastage accounted for the fall in fertility in the 1944-45 Dutch famine.[25] Also, no increase in fetal deaths were observed in the 1974-75 Bangladesh famine.[27]

Maternal malnutrition can be directly related to poor fetal development and low birth weight.[53] Since low birth weight is associated with poor infant survival,[54] it is possible to envision a mechanism whereby malnutrition results in a rise in natural fertility rates. In fact, this sequence of accelerated childbearing has been observed in Bangladesh, and is a factor in the high rate of maternal mortality.[55]

Nutrition, Infant Survival and Fertility

If one considered net reproductive performance it would be clear that the most profound effect of malnutrition in a population is on the survival of the infant.[59] Infant mortality can affect fertility through either a biological or a behavioural mechanism.[60,61] The biological effect relates to the fact that women with early infant death will naturally resume ovulation earlier than with women with surviving breastfed children.[2] It should be noted that this biological effect due directly to infant malnutrition is not likely to be great except in populations with very prolonged lactational amenorrhea. This is because malnutrition would ordinarily not appear in a fully breastfeeding infant before the age of 6 months unless there were intervening diseases such as diarrheal illness.[62]

The behavioural effect relates to what is termed the "child survival hypothesis".[63] This posits that couples in populations experiencing high infant mortality will seek to have additional children either to replace those that are lost, or as insurance against perceived potential losses.[61]

Both the behavioural and the biological mechanisms would link high levels of infant mortality seen in malnourished populations to higher levels of fertility, although only the biological mechanism could qualify under the definition of natural fertility.

Discussion

This report has focused on an analysis of malnutrition as it is likely to affect those biological mechanisms that are directly related to fertility performance, that is the reproductive lifespan, post-partum amenorrhea, fecundability and pregnancy outcome. Further, only general protein calorie malnutrition has been considered rather than the possible effects of isolated nutritional deficiencies, primarily because the focus has been on fertility effects which are likely to have significant demographic impact.

The relationship of nutrition to fertility levels in human populations has been a subject of considerable discussion. Generally the frame of reference for the level of natural fertility that approaches the biological limits has been the Hutterite women who average about ten live births over a reproductive live beginning at age 20. Essentially every other non-contracepting population, both historically and in modern times, has had lower fertility levels. Since these populations are generally poorly nourished in comparison to the Hutterites it has often been postulated that their depressed fertility is directly related to the biological effects of malnutrition.[14] The analysis in this report indicates that when one searches for direct causal relationships between nutritional status and fertility this hypothesis is not well supported.

It is quite clear that gross starvation results in the cessation of all reproductive functions in both the male and the female. Famine conditions even when not associated with frank starvation can also lead to severely depressed fertility through both biological (including psychological) and behavioural mechanisms. Regarding the more common chronic low grade malnutrition, the only clear effects on reproductive performance relates to the reproductive life spans; menarche can be delayed and it is probable that there is an earlier onset of menopause. Since these effects are at the extremes of reproductive life, where behavioural changes relating to age of marriage and conscious desires to terminate of child bearing are the strongest in most societies, it is unlikely that this has a great effect on overall reproductive performance.

Within the fecundable years there is no conclusive evidence that chronic malnutrition per se has a major effect on fertility at the population level. The two components of the reproductive cycle of greatest interest, that is lactational amenorrhea and fecundity are apparently far more influenced by social and behavioural factors. The first relates to breastfeeding practices and the second to patterns of cohabitation. As physiologists are beginning to dissect the precise neuro-hormonal control mechanisms related to lactation and its exquisite sensitivity to nipple stimulation by suckling it appears that most of the variability in amenorrhea will relate to patterns of breastfeeding.

Since fecundability is so closely related to coital frequency, if obviously would be strongly influenced by life style. One could visualize chronic malnutrition causing some reduction in coital frequency due to fatigue in a hardworking rural agrarian population.

In summary, the data available would suggest that the biological effects of nutrition probably cannot account for the major variations in fertility between different non-contracepting population groups. Most of the evidence relating nutrition to fertility is very limited except under extreme conditions of malnutrition. Now that the physiological mechanisms relating to fecundity and to lactational amenorrhea are better defined, more research should begin to provide answers to this important question which has policy significance to developing countries worldwide.

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