TABLE B—LOW BIRTHWEIGHT IN 1973

Country	% of live births: Less than 2500 g	% of live births: Less than mean−2 SD (estimated from graph)	% of live births with unknown birthweight	No. of live births in study
Austria	6·2	4·5	0·002	49 590
Cuba	10·7	3·7	7·94	233 366
Hungary	11·6	6·5	0·0006	156 224
Japan	5·3	3·5	0·02	209 023
New Zealand	5·4	3·9	0·17	121 454
Sweden	4·2	3·5	1·02	109 560
U.S.A. (6 states)	6·6	4·3	0·27	146 259

column of Rooth's table III does not tally with the data in table B because Rooth's percentages appear to be taken from a table from which multiple births were excluded, whereas mine includes all livebirths, as this was the definition given in his paper.

Apart from his claims about the selection of weight groups, the computational errors do not affect Rooth's basic argument, although the data for Cuba should be interpreted with some caution as birthweight was missing for 7·9% of live-births in 1973.

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SIR,—Professor Rooth describes the use of cumulative birthweight distribution curves plotted on a probit scale to indicate the proportion of low birthweight, as the percentage of births more than 2 SD below the mean for different populations. In this way the definition of low birthweight changes with the mean birthweight and with the slope of the curve, if we assume that one gaussian distribution describes the bulk of the population while another describes the births belonging to the minor fraction. Two gaussian distributions are indeed present in populations of different geographical areas,[1,2] and W.H.O. data[3] conform to this pattern.

At a course on perinatal mortality held in Erice, Sicily, last September, J. G. Fryer and his colleagues showed that the cumulative distribution curve for birthweight in Palermo, Italy (1972) and in Gothenburg, Sweden (1972–73) is described by a mixture of two gaussian distributions, superimposed with different means and SDs. In Palermo 80% of the live births belong to a "primary" gaussian component with a mean ±SD of 3570±440 while the remaining 20% belong to a "secondary" component (3460±1025). In Gothenburg the primary component covers 93% of the population (3490±470); only 7% belong to the secondary component (2870±1030).

Birthweight-specific neonatal mortality rates computed separately for the two components show a wide gap, being much lower for the primary component than for the secondary component. Low birthweight (<2500 g) does contribute heavily to mortality, but this effect is very much stronger in the secondary component, where mortality is high throughout the birthweight range, especially in Palermo. To work out health policies the fraction of the population belonging to this secondary component needs to be known for defined geographical areas.

The exclusion of early neonatal deaths from all livebirths, to build-up a correct estimate of the birthweight distribution, may hide some health needs, especially where such deaths are more common. Plotting mortality rates by birthweight group on the same graph that shows the overall birthweight distribution for all live births strengthens the information that can be had from this kind of analysis.[4] On the other hand it seems very sensible to exclude late fetal deaths when constructing the distribution curve since the time elapsed between death and birth is unknown (except for intra-partum deaths), and during this time the weight of the fetus may change.

An approach that compensates only in part for the lack of a proper descriptive population distribution may be the analysis of the specific mortality rate, which is based on the number of fetal deaths in each birthweight group expressed as a percentage of the number of all fetuses that attain or pass through the birthweight range—i.e., those born alive or dead with a birthweight within the range in question or born later with a higher birthweight (in other words, the number of pregnancies entering the risk factor group[4,5]).

The inference that redefining low birthweight by the mean−2 SD criteria will mean a reduction in the frequency of the light-for-dates babies does not seem to fit-in with population intrauterine growth patterns. This aspect can be evaluated by analysing the distribution of birthweight at different levels of gestational age throughout the whole gestational period in a birthweight/gestational age diagram or, according to Prof. J. R. Ashford (personal communication), in a system of isobols corresponding to loci of equal frequency.

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MATERNAL CHOLERA IMMUNISATION AND SECRETORY IgA IN BREAST MILK

SIR,—It seems that lymphoid cells from the Peyer's patches of the small intestine committed to secretory IgA (SIgA) production can migrate to the mammary gland.[1] As a result, breast-fed infants ingest ready-made SIgA antibodies against intestinal pathogens that have infected their mothers. Antibodies against *Vibrio cholerae* and *Escherichia coli* enterotoxins, and against *Shigella*, *Salmonella*, enteropathogenic *E. coli*, and *V. cholerae* have been found in breast milk.[1] Parenteral vaccination of lactating women in one endemic area with killed cholera vaccine has produced a booster effect on specific milk SIgA antibody levels.[2]

We recently administered parenterally a 0·5 ml dose of either an aluminium-adjuvanted, formalinised cholera toxoid vaccine or aluminium-adjuvanted vaccine containing the formalinised cholera toxoid and cholera whole cell vaccine to volunteers in Bangladesh. The vaccines used were produced by the Wellcome Research Laboratories, U.K. Using an ELISA test[3] we found a significant increase in anti-cholera toxin (anti-CT) IgA titres in the milk of 5 of 6 lactating mothers; 3 of the 5 mothers also had a significant increase in milk anti-CT IgG titres. In 1 woman the increased anti-CT IgA titre persisted for

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3. Holmgren J, Svennerholm A-M. Enzyme-linked immunological assays for cholera serology. *Infect Immunol* 1973; 7: 759–63.

ANTIBODY TITRES IN A LACTATING WOMAN RECEIVING A COMBINED WHOLE CELL-TOXOID CHOLERA VACCINE

Day after vaccination	Anti-CT titre*			Anti-LPS titre*		
	IgG	IgM	IgA	IgG	IgM	IgA
Serum						
0	15	200	<54	23	166	2·2
14	125	270	456	181	152	10·4
Milk						
0	<2·5	<4·3	<11	<1	<0·7	1·0
5	<2·5	<4·3	<11	<1	<0·7	1·1
7	<2·5	<4·3	500	<1	<0·7	1·8
8 mo	<2·5	<4·3	100	<1	<0·7	0·6

* Results are expressed as ELISA units with a standard control serum (National Institutes of Health) having a value of 100 units.

8 months; this woman also had a significant rise in serum anti-CT and serum anti-lipopolysaccharide (anti-LPS) IgG and IgA titres (table).

Using the ELISA test we also measured the specific milk anti-CT IgA response to the A and B subunits of cholera toxin in 3 of the mothers. In 2 only a significant anti-B response was found while in the third both a significant anti-A and anti-B response were observed.

In contrast to these results no rises were seen in milk or serum anti-CT or anti-LPS titres in 3 lactating women who received tetanus toxoid.

We conclude that parenteral immunisation with cholera toxoid can significantly increase anti-CT IgA and anti-CT IgG titres in breast milk in this population living in a cholera endemic area and that the anti-CT IgA response can occur against both the A and B components of cholera toxin.

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ATHEROGENICITY AND THE SUPERMARKET SHELF

SIR,—We cannot agree with Dr Smith's statement (March 8, p. 534) that, in the debate on dietary change and coronary heart disease (CHD), emphasis is only placed on lowering serum cholesterol. On the contrary in recent years discussion on diet has almost always included its fat content as well as its fatty-acid composition in relation not only to blood lipids but also to platelet function and, in recent years, to prostaglandin activity.

Physicians advise restricted consumption of *all* saturated fat whether of dairy, meat, or food industry origin. The latter includes hydrogenated margarines as well as coconut and palm oils widely used for cakes, biscuits, non-dairy creamers, and so on. Their constituent fatty acids have different effects in relation to atherogenesis and thrombogenesis. It is not always appreciated that butter is also, in effect, an hydrogenated product resulting from bacterial action on polyunsaturated vegetable oils in the rumen of the cow.[1]

Wissler and his colleagues in Chicago, to whom Dr Smith refers, have not only used coconut oil and butter to produce pathological changes in the coronary arteries of non-human primates but, using ordinary Western diets, have produced similar changes which cannot be distinguished under the microscope. Considerable regression may occur on changing back to their natural diet. These changes did not occur or were only mild with the "prudent" diet now widely recommended for coronary prevention. Wissler also used peanut oil but in large amounts which far exceeded those consumed by human beings. The lesions were more fibrotic with much less lipid than in human atherosclerosis.[2] Consumption of peanut oil is not usually recommended, because it is relatively low in PUFA; it is not considered likely to be harmful in amounts most people use.

In the U.K. with 20% of the E.E.C. population we consume more than 40% of the milk. Unfortunately the Milk Marketing Board refuses to lower the fat content of standard milk from 3·8% to, say, 2·3% at which level the change would hardly be noticed, as those who "pour off the top" know. They also obstruct the import of low-fat milks from E.E.C. countries. As a result low fat milks and milk products are not readily available here as in other countries. It is only excess fat in milk which is considered harmful. The nutrients are in the skim. The welfare of the dairy industry would be greatly assisted if we stopped importing 75% of our butter and 40% of the cheese as at present.

The fat switch from butter and other saturated fats to polyunsaturated oils and margarines has a sound nutritional basis. Restriction of dairy fat is emphasised because it makes the largest single contribution and accounts for about half the consumption of saturated fat in the U.K. Butter has no nutritional value apart from its vitamins A and D content of which there is no lack in this country. The vitamins are in any case added to margarine by law.[3]

The dairy industry's ethics have also been called into question on the international scene by Joossens[4] as they have on the domestic scene. Recent advertising campaigns have been aimed at children and young people whose developing tissues are particularly susceptible to injury. The recent Butter Information Council's advertisement in *The Lancet* (London edition, March 1) is misleading and gives erroneous data on fat consumption in the U.K. and omits all mention of fatty-acid composition. The taste for butter and cream induced at this age is undesirable. Trenchard extrapolates from a slight underestimation of carcass fat early in this century and, despite his criticism, the official figures of consumption are more likely to be correct. It is agreed that our level of dietary protein has remained much the same in the past 30 years and all accept that there has been a reduction of carbohydrates (mainly from cereals and potatoes). Hence fat consumption must have increased. Consumption of dairy fat alone has increased by 25% in recent years.[5]

Trenchard fails to state that the greater consumption of vegetable oils was almost entirely in hydrogenated (saturated) form and we agree the majority of margarines which are hardened are likely to be injurious. Although lard used to be a good source of polyunsaturated fat, because of altered animal feeding it is now more saturated (44% and not 30% as stated by Dr Smith[6]) and it is then further hardened, by hydrogenation before packing, to increase shelf life. In striking contrast the soft polyunsaturated margarines and oils reduce plasma lipid levels and improve platelet function. Experimentally they are not atherogenic or thrombogenic, as are saturated fats, but harmless and protective.

Advice to the housewife was not spelled out by the D.H.S.S. in their 1974 COMA report, which in consequence made little

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