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SYMPOSIUM ON 'PROTEIN–ENERGY MALNUTRITION'

Childhood malnutrition—the global problem

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The brief given to me for this paper suggests that 'after a short introduction on the way malnutrition is measured in children there could follow a review of what is known of the international pattern of prevalence rates and how these relate to indicators of social and economic development'. I do not propose to stick to this brief exactly, because I think that at the present time it is more important to understand what we are measuring than to determine the prevalence of what is measured. Some years ago the World Health Organization published a series of articles on malnutrition in different regions of the world, with maps illustrating the prevalence of PEM, vitamin A deficiency, etc. (Someswara Rao, 1974; Bailey, 1975; Burgess & Burgess, 1976). My terms of reference suggest that my first job should be to collect figures to bring this information up to date, but I do not intend to do that, because WHO can do it more thoroughly.

It is more useful, in the context of this meeting, to consider in a general way what kind of information is available, what it means and what use can or should be made of it.

The first point is a very obvious one, but still not always appreciated, that information about matters such as prevalence is of no value unless related to action, either as a stimulus or as a guide. I use the word stimulus because at the World Health Assembly last year, in the general discussion on nutritional surveillance, the point was made that the health sector has a moral duty to take the lead in drawing the attention of governments to the prevalence of malnutrition. The point that information is gathered to be used was clearly in the mind of John Graunt, the founder of the subject of vital statistics, when he wrote: 'Having been born and bred in the City of London, and having always observed that most of them who constantly took in the weekly Bills of mortality, made little other use of them, than to look at the foot, how the Burials increased or decreased; And, among the Casualties, what had happened rare and extraordinary in the week current: so as they might take the same as a Text to talk upon in the next Company . . . Now I

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thought that the Wisdom of our City had certainly designed the laudable practice of takeing, and distributing, these Accompts, for other and greater uses than those mentioned above, or at least, that some other uses might be made of them. . . .' (Graunt, 1662).

We come, then, to the kinds of information available on the prevalence of malnutrition and food deficiency and the uses which can be made of it. Figures on food production provide a background. The Food and Agriculture Organization (1977) has recently summarized information for the whole world in their 4th World Food Survey, which shows that in developing countries in the last ten years or so total food production has increased by about 40% and food production per head by about 8%. These average figures of course conceal great differences in distribution between different countries, different groups in the same country and individuals in a group. Table 1 shows the estimated average energy intake per head in different categories of countries. The situation in the so-called MSA countries (most seriously affected countries) has deteriorated over the decade, whereas in the others it has improved. It seems a remarkable coincidence that in the world as a whole the average energy supply is just about 100% of the requirement.

Table 1. *Daily energy supply/head (% of requirement)*•

	1961–1963	1964–1966	1969–1971	1972–1974
Developed market economies	123	124	129	131
Eastern Europe and USSR	126	127	133	135
Developing market economies:				
Most seriously affected (MSA)	91	90	92	90
Non-MSA	95	96	100	101
World	101	103	106	107

•From 4th World Food Survey, Food and Agriculture Organization, 1977.

FAO has also attempted to calculate the proportion of people whose energy intakes are inadequate. The assumptions are that the mean and range of intakes are known, that the distribution is skewed and that an intake of less than 1·2×BMR is definitely inadequate for normal health and activity. Results of these calculations are summarized in Table 2. If a quarter of the people in developing countries are really living on an average intake of 1·2×BMR or less, there must be a remarkable degree of adaptation, as has been suggested by Miller *et al.* (1976) and by Norgan *et al.* (1974) on the basis of their work in Ethiopia and New Guinea. This is certainly a subject which demands further study.

If the information provided by FAO is correct, we can presumably conclude that at present it is not the total supply of food in the world which is the limiting factor, but its distribution—an obvious point which many others have emphasized, but which still needs to be made.

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Table 2. *Estimated proportion of subjects (%) with energy intake below 1·2×BMR***

	1972–1974
Africa	28
Far East	29
Near East	16
Latin America	15
Most seriously affected (MSA) countries	30
Non-MSA	18
All developing countries	25

*From 4th World Food Survey, Food and Agriculture Organization, 1977.

The prevalence of malnutrition in children

As with other diseases, the indicators of prevalence are mortality and morbidity.

Mortality. Twenty years ago Dr Verity Wills and I proposed that whereas the infant mortality rate (deaths from birth to 1 year) has been regarded as a general indicator of the effectiveness of public health services, the death rate from 1 to 4 years might serve as a rough index of the prevalence of malnutrition (Wills & Waterlow, 1958). This idea has been quite widely accepted, and by and large it has been justified, at least for one group of countries, by the epic study of Puffer & Serrano (1973) on preschool child mortality in Latin America and the Caribbean. In this work every death of a child below 5 years old in the designated population (some 30 000 deaths in all) was scrupulously followed up, and it was shown that malnutrition contributed to death in about half the cases. Table 3 suggests that if reduction in mortality is a criterion there may be some cause for optimism. Unfortunately, there are very few developing countries which provide statistics of this kind.

Table 3. *Annual rate of decrease of child mortality in selected countries**

Country	Period	Decrease /year (%)
Egypt	1960–1972	3·8
Jordan	1966–1973	12·5
Sri Lanka	1951–1969	7·9
Thailand	1969–1973	6·9
Colombia	1954–1972	4·5
Mexico	1970–1973	13·8
Venezuela	1950–1973	6·2

*Dyson, 1977.

As with per caput food production, national mortality statistics disguise differences between regions and groups. Developing countries are more heterogeneous than developed ones. Table 4, from Brazil, illustrates the very large differences which may be found. They would probably be even greater if rural communities had been included.

Table 4. *Infant and preschool child mortality rates in three cities in Brazil**

	Deaths/1000 live births	Deaths/1000 inhabitants
Age, years	0–1	1–4
Recife	91	9·0
Ribeirao Preto	53	2·6
São Paulo	61	2·8

*Laurenti, 1977.

Perhaps even more valuable than national statistics are longitudinal studies of particular communities. A classic example is the village of Keneba in The Gambia. McGregor (1976) states that of the cohort of children born between 1965 and 1969 only 44% were still alive in 1974. Table 5 shows the death rates in the first 5 years of life in three highland villages in Guatemala. These rates, high though they are, are not as appalling as that in The Gambia.

Table 5. *Infant and preschool child mortality rates in three Guatemalan villages, 1959–1964**

	Deaths/1000 live births	Deaths/1000 in age-group			
Age (years)	0–1	1–2	2–3	3–4	4–5
Village A	88	88	58	33	14
B	146	77	28	72	42
C	191	166	80	42	28

*Ascoli *et al.* 1967.

The simplest method of all of obtaining information about mortality rates is by enquiry from a cross-section of mothers, because they usually seem to be able to recall how many children they have had and roughly at what ages they died. Thus Omolulu (1972) in a 4-year study of the village of Osegera in Western Nigeria found that 36·6/1000 of 1 to 4-year-old children died each year. The infant mortality was 135/1000, so that almost 30% of children would fail to reach the age of five. Similarly, Burgess *et al.* (1969) calculated that in the Kisewara district of Tanzania between 22 and 33% of children died before they reached 5 years.

I make no claim that in all these situations malnutrition is the main cause of the high death rates. We would need a Puffer & Serrano (1973) type of study to establish that. Gastroenteritis is most commonly given as the cause of death, but its relationship to malnutrition is well known. I think that in these African countries, with their extraordinarily high death rates, the role of holoendemic malaria could be very important, because, like malnutrition, it depresses cell-mediated immunity (Williamson & Greenwood, 1978).

Morbidity. The indicator which WHO has used up to now is a deficit in weight for age. I have argued repeatedly (Waterlow, 1973, 1976) that this is a bad indicator because it confounds two physiologically distinct effects, deficit in weight

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for height (wasting) and deficit in height for age (stunting). However, I have to admit that there is a certain logic about using weight for age from a crude public health point of view. The general pattern (to which there may be many exceptions) seems to be that the rates of gain in weight and height both begin to fall off at about the same time, at about 3 months of age in many countries (Waterlow & Rutishauser, 1974). By 2 to 3 years normal weight for height is regained, but catch-up in height occurs more slowly, so that by this time we are left with a group of stunted or retarded children who, because of being stunted, still have a low weight for age. It is therefore reasonable to argue the point that prevalence of deficit in weight for age correctly indicates the incidence of malnutrition. By including both children who are currently malnourished and those who were malnourished at some previous time, it gives the proportion of children who have passed through a period of malnutrition. It has also been argued that since the prevalence of low weight for age is always much greater than that of low weight for height, the former makes a much greater impression on authorities responsible for health and nutrition and provides a better stimulus for action. This argument may sound cynical, but I think it is reasonable if we accept that the only point in collecting figures on prevalence is to promote action.

The relation between morbidity, as judged by weight deficit, and mortality is of interest, but there is very little information about it. Sommer & Loewenstein (1975) in Bangladesh used arm circumference, which correlates quite well with weight for age (Rutishauser, 1969; Shakir, 1975) as an index of malnutrition. They found that in children below the 9th centile the risk of death was three to four times greater than that of children above the 50th centile. Kielmann & McCord (1978) in the Punjab found that the mortality of children between 1 and 36 months old doubled with each 10% drop below 80% standard weight for age. At all weight deficits the mortality was lower in the older children. This suggests that it might have been useful to distinguish between wasting and stunting, because wasting is commoner in younger children, stunting in older ones. It is very important to know whether a stunted child who is of normal weight for height is in fact at increased risk. In Jamaica we used to discharge children from the ward as 'recovered' when they had reached normal weight for height, even though they were still small in stature. The rates of recurrence and of death after discharge were very low.

The relation between severity of malnutrition and mortality is not fixed. Action may affect one but not the other. Cravioto & Delicardie (1976) mention that in their longitudinal study in a Mexican village the provision of medical care halved the infant and preschool mortality rate with no effect on the incidence of severe PEM. To use the old analogy of the iceberg, the medical services can cut off the tip, but they leave the main part untouched. This raises the key question, to which I shall return: does it matter passing through an episode of malnutrition if you do not die of it?

Having discussed the crude indicators of the prevalence of PEM, it may be useful at this point if I try to define what seem to be the logical steps in any enquiry:

1. Description of the over-all problem; assessment of prevalence by crude indicators.
   Purpose: to stimulate further enquiry and action.
2. Identification of those people who are malnourished.
   Purpose: to focus action where it is most needed.
3. Definition of the physiological problem.
   Purpose: to ensure that action is appropriate to the needs of the people and the possibilities of achieving anything.
4. Examination of causal factors.
   Purpose: to assess priorities for different kinds of intervention.

Obviously these steps are inter-related. As regards step 2, identification, Payne's work has shifted the emphasis from the traditional physiologically vulnerable groups to a functional classification in terms of ecological and socio-economic factors, what Cravioto and his colleagues call the macro-environment. It may be a difficult matter of judgment to know in how much detail to pursue the functional classification. Cravioto & Delicardie (1976) in Mexico and Grantham-McGregor *et al.* (1977) in Jamaica have shown that in groups where the macro-environment is the same some families have malnourished children while others do not. The Mexican workers found that the significant factors seemed to be the mother's attitude to the child and her interest in the outside world, as judged, for example, by the extent to which she listened to the radio. I expect it would be dangerous to generalize from this experience; there are probably other situations in which the critical factor is the incidence of infection, and others again in which it is the availability of food. The point is an obvious one, that detailed examination of who is malnourished will provide a great many clues as to why they are malnourished.

What I have called the third step, definition of the physiological problem, is perhaps the most difficult. Under this heading comes the whole question of cut-off points. Where is the appropriate dividing line, from the point of view of action, between malnourished and not malnourished? The results of Kielmann & McCord (1978) referred to previously show that if death is the criterion children less than 1-year-old have an increased risk if they are below 80% of standard weight for age, but above 1 year there is no increased risk until they are below 70% of standard weight. Thus by this criterion what is significant malnutrition at one age is not significant at another age.

This leads on to the problem of the importance of stunting, which in most populations is the commonest manifestation of malnutrition after the age of 1 to 2 years. The first question is whether stunting is simply the end result of an acute episode of malnutrition, like, say, healed tuberculosis, or whether it represents a process which is different in kind from that which causes wasting. I have the impression that most people tacitly incline to the first view, but I am not sure that this is correct. There are great differences in the proportion of wasted to stunted children in different population groups. A good example is the classic paper of Rutishauser & Whitehead (1969) on the Karamoja and Baganda tribes of Uganda. The Karamoja children, who live on milk and meat, are tall and thin, while the

Baganda, whose staple is plantains, are short and fat. Another example, from two regions in Central America, is shown in Table 6. It would be surprising if such differences could all be explained on genetic grounds.

Table 6. *Prevalence of wasting and stunting of children* (%) *in two districts of Salvador*•

Age (months)	District	Wasted †		Stunted ‡	
		A	B	A	B
6–11		17	21·5	29·5	21
12–23		20·5	32	40·5	36
24–35		17·5	26·5	54	38
36–47		10	20·5	65	45
48–59		10·5	24·5	59	51

•Valverde (unpublished results).
†Wasted, <80% expected weight for height.
‡Stunted, <90% expected height for age.

If stunting results from a different kind of malnutrition from wasting, what kind is it? It is tempting to suggest that retardation of linear growth is caused by protein deficiency, but dietary surveys do not bear this out (Waterlow & Rutishauser, 1974). Moreover, if the energy supply is inadequate, as is commonly the case, protein cannot be utilized, so that according to current teaching it is impossible to achieve adequate protein nutrition in the presence of insufficient energy. This seems to me a subject which urgently needs further research.

The second question is whether stunting is simply an end result, or whether it is a continuous process. In this connection the results of Martorell and co-workers (unpublished results) in Guatemala are of great interest. They compared linear growth and the rate of skeletal maturation in children up to 3-years-old, with and without an energy supplement. In the unsupplemented children the rate of growth was less, but the rate of maturation was not much affected. Similar results on older children were obtained by Lampl *et al.* (1978) in New Guinea. Martorell concluded that 80% of the effect of the supplement on body size was due to an effect on what they call the intensity of growth. If that is so, we cannot look upon stunting simply as an end result of an earlier injury. On the contrary, it represents an on-going process which apparently can be reversed, or at least prevented from getting worse, by dietary means. This has obvious implications for action, provided we accept that it matters to be stunted. On this point I think we have to accept Cravioto's dictum: 'Survivors of early severe malnutrition are different from other children' (Cravioto & Delicardie, 1971). They are different because they are mentally and behaviourally handicapped. Cravioto's work, and that of Richardson (1976) in Jamaica suggest very strongly that a main cause of this handicap is not so much malnutrition *per se*, but the environment in which malnutrition develops, particularly the lack of social stimulation in the home.

The argument, then, is as follows:

(a) The commonest diagnosed form of malnutrition is stunting.
(b) Smallness in physical size is, in itself, of little importance.
(c) Smallness is accompanied by mental and behavioural handicap.
(d) This handicap is in large degree the result not of malnutrition but of social deprivation.

If this argument is correct, measures to combat malnutrition, such as feeding programmes, will not have much effect unless at the same time they reduce the extent of social deprivation.

At the beginning of this paper I made the point that studies of prevalence have no value unless they are linked to action. However, the most effective action is not necessarily that directed specifically to the improvement of nutrition. Mata & Mohs (1976) have given a good example from Costa Rica. Between 1966 and 1975 the proportion of stunted children (less than 91% of standard height for age) fell from 19·2 to 8·4%. Mata comments: 'The important consideration is that the changes in nutrition and health have been based on education, on reforms and social justice, on a substantial improvement in environmental sanitation and in the standard of life. Apparently no relation exists between these improvements and the scientific development of nutrition'.

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