ARCHIVOS LATINOAMERICANOS DE NUTRICION
Organo Oficial de la Sociedad Latinoamericana de Nutrición

Vol. 45 N° 1-S, 1995

Small but healthy?

George H. Beaton

It is a great pleasure to participate in this Congress which honours Jose Maria Bengoa. It was Dr. Bengoa who first prodded me into an interest in the interpretation and application of anthropometric data. When I knew him 20-25 years ago, one of his strong passions was promotion of the 'nutrition rehabilitation clinics'. You will all appreciate that a main criterion for admission to such programs, and the main criterion for clinical effect was indeed the measurement of weight and height. A second passion of Bengoa was the establishment of a monitoring system that might use school admission anthropometric measures as an indicator. His dream of networks of rehabilitation centres did not materialise in the form he originally hoped but they have appeared in other forms. Nutrition monitoring systems, perhaps more comprehensive than he envisaged, have emerged and are well displayed in the series of SCN (United Nations` ACC subcommittee on Nutrition) reports on the World Nutrition Situation (1-5). Dr. Bengoa has had important impact on the development of international nutrition activities and has influenced many people, including myself. It is a pleasure to be able to join this appreciation of that contribution.

The objective of this paper is provide a personal perspective of some changes in thinking about the interpretation and use of anthropometric measures in children. For the most part, the material I will present is taken from three publications (6-8) spaced over a four year period. They reflect a gradual evolution of the author's own thinking and understanding in this area though, of course, they reflect a gradual evolution of the thinking of many others on whose work and writings these papers were based.

Not mentioned is an earlier study undertaken for UNICEF and the SCN. Beaton and Ghassemi (9) published a review of pre-school child feeding programs around the world. That review suggested that while anthropometric effects of supplementary feeding programs could be seen in research programs and well controlled pilot studies, in operational programs it was difficult to see an effect. An exception was in the nutrition rehabilitation programs which selected children with major anthropometric deficits. At the time, the authors were unable to offer a convincing explanation. Was the failure to see an effect simply the result of poor quality of data collection and analysis in operational programs as contrasted with research projects, as many critics have since suggested? Did the absence of detectable effect really reflect failure of the program infrastructures, inadequate levels of feeding or failure of specific targeting, as other critics of the programs have suggested? Beaton and Ghassemi raised another distinct possibility - that the programs did have effects but that 'growth' was the wrong response to have expected? Today, I think I personally understand much more clearly what we were seeing 15 years ago when we did the pre-school child feeding review.

Department of Nutritional Sciences. University of Toronto, Toronto, Canada.

A description of the evolution of thinking may be informative - perhaps it will remind all of us that the evolution continues and that today's 'truths' may have to be questioned tomorrow. The overall conclusion that arises from this evolution of thinking, and the point that was not appreciated in general thinking when the Beaton and Ghassemi review was conducted was that:

Being small is not the problem
It is becoming small that is a problem

In 1982 the perceived goal of feeding programs was to help small children become bigger. The programs were not really designed to prevent children from becoming small. The programs reviewed in 1982 reached children who had already become small and who should not have been expected to show much, if any, anthropometric response except among those who were thin or severely and currently malnourished.

It has long been accepted in paediatric practice that growth faltering is a typical finding in almost all serious clinical illnesses in young children. The desirability of monitoring weight gains in young children was a well established part of paediatric practice early in this century. Of course it was also known from animal studies that changes in growth rate were uniformly seen as a manifestation of food restriction or of specific nutrient deficiencies. Thus there was a long standing biological basis for linking anthropometry with the assessment of nutritional status.

In the 1950's there was a new awareness that Kwashiorkor and its sister syndrome Marasmus, were highly responsive to dietary management and hence represented major nutritional deficiency diseases. It was gradually noted that in addition to the clinical manifestations of these conditions, the children were small by Western standards. Further, it was recognised that in general children in the communities where the clinical syndromes appeared were also small. This observation had profound impact in the nutrition community. What we were suddenly saying was that the 'usual' or 'normal' condition of children in these communities was indeed abnormal. We went much further, we concluded that the children were small because they were malnourished. By 1960 the notion that smallness = malnutrition was firmly established. The famous Mexican paediatrician, Dr. Gomez, had earlier recognised that body size was a clinically useful predictor of outcome for children entering the clinics of his hospital. On empiric evidence he developed a classification scheme based on the observed weight relative to that expected for the individual's age and gender - the now well-known «Gomez Classification». It seemed logical to use this classification scheme to describe children in the communities where kwashiorkor and Marasmus were being found. For the record, during the meetings of an expert committee (10), Dr. Gomez made the strong assertion that he was upset about that application of his classification scheme.

He emphasised that it had been developed to apply to children who had already been judged to be ill and had never been intended as a screening tool for children in the community or whole populations as later developed.

Two things evolved from this beginning. First, the Gomez classification and its subsequent iterations became internationally established and continue in wide use today. This locked a conceptual framework that equated smallness with malnutrition. Second, and perhaps more important, arising from the early work of Gomez, many many epidemiologic studies have shown, very convincingly, that within every population studied, those children with the greatest deviation in anthropometric measures exhibit the greatest risk of subsequent morbidity, mortality and signs of impaired functional development (11). This, is consistent with Dr. Gomez' original observations - the smallest children (in respect to expected size) presented the greatest clinical risk and required the most intensive treatment.

However, there was backlash to the equating of smallness and malnutrition. Indeed there were two types of backlash. First, it was noted that not all small children became ill or died. Clearly the adult population existed and appeared to function adequately within their societies. This became the basis for arguments about «adaptation» to chronic malnutrition. One widely publicised critic of conventional thinking was Seckler who coined the now famous rhetorical question «Small but healthy?» (12-13). A second type of backlash arose from papers such as our 1982 review of pre-school feeding. If indeed smallness was equated with malnutrition and was extremely prevalent in most developing country rural communities, why was it so difficult to show substantial response to diet. Conventional medical thinking of the time would argue that the ultimate proof of a nutritional deficiency was clinical response to specific nutritional therapy. Many saw our paper as a fundamental criticism of then-current concepts. Looking back, I agree - the findings of that paper were indeed a challenge to conventional wisdom but it was not recognised at that time - the literature and general thinking had to evolve still further.

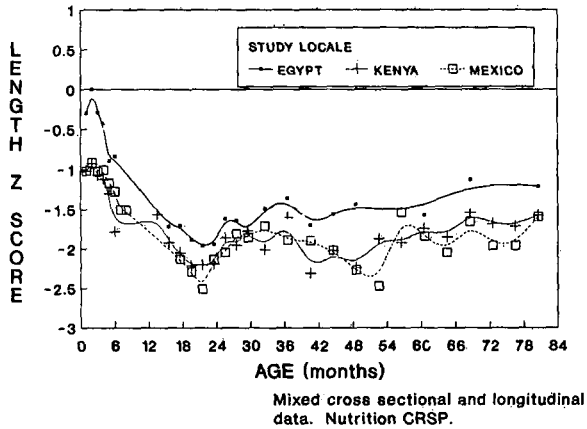
By 1989 when I presented my paper «Small but Health? Are We Asking the Right Question?», I had been exposed to the preliminary results of the Collaborative Research Program in Nutrition (Nutrition CRSP) (14). The Mexico project of that program (led by Adolfo Chavez, Lindsay Allen and Gretel Pelto) had made an extremely important observation, subsequently confirmed in the data from Egypt and Kenya. Deviations from reference growth patterns of young children were evident as early as 3 or 4 months of age - much earlier than had been generally recognised. By the end of the CRSP data collection, it could be seen (Fig 1) that the process of growth faltering began well before 5 months and appeared to have ended by 18-24 months. That is, there appeared to be an age window during which failing to grow was an active process and an age at which this was transformed to a state of «having failed to grow».

These observations provided a possible explanation of why the 1982 review of pre-school feeding programs by Beaton and Ghassemi, and a subsequent review of programs in Latin America (15-16) had failed to see major effects of food supplementation on growth and achieved size. Very few operational programs reached children below the age of 30-36 months. Most were in the 3-5 year old range. Is it not reasonable to suggest that most of the children were too old to respond anthropometrically?.

This set in motion a personal campaign to sound the alarm with regard to our then-current mode of thinking about anthropometry. Smallness (in older children) does NOT mark [current] malnutrition. Generally it DOES mark past growth faltering. I was, and am, concerned that we have mislead ourselves by continuing to use the term «malnutrition» when we mean «smallness». Many have very carelessly assumed that anthropometry is a good proxy for dietary intake and that associations between anthropometric measures and various functional outcomes is evidence of relationship between nutrition (meaning dietary adequacy) and function.

FIGURE 1

Observed lengths of young children in villages in Egypt, Kenya and Mexico. Data expressed as Z scores using the CDC reference data set.



Taken from the Nutrition CRSP (Calloway et al, 1988)
Mixed cros sectional and longitudinal data. Nutrition CRSP

It was the critics, like Seckler (12-13), that challenged these assumptions and made us rethink the relationships. Perhaps we should really thank him while at the same time continuing to disagree with some of the conclusions he drew about relative unimportance of food intake.

An extremely important part of the evolution of my own thinking can be attributed to participation in an SCN workshop on the Appropriate Use of Anthropometric Indices. Rey Martorell and I spent many hours gradually untangling old concepts and rebuilding a coherent framework that seemed reasonably consistent with existing knowledge. The report of that meeting includes the diagram portrayed in Fig 2.

Two important features of this schematic are emphasised. First, it recognises that growth (and hence achieved size) is an outcome that can be influenced by genetic factors, by the impact of disease processes, and by dietary intake. That is entirely consistent with the evidence. We owe a great deal to people like Rey Martorell and Jean-Pierre Habicht for the many analyses and population comparisons that have convincingly established that, between populations, it is environmental more than genetic differences that produce the typical deviations growth curves for young children.

The second critical feature of this model is that it suggests that other functional outcomes may be influenced by the same environment that influenced physical growth - but not necessarily by the same path or by the same specific environmental factors. Growth failure may be a very effective marker of these other outcomes without being in the direct causal path. Note, for example, the suggestion that impairment of psychological development may arise from the early environment of the child and its interactional experience - a point argued for many years by Craviotto and by Monkeberg. Such a path of effect would

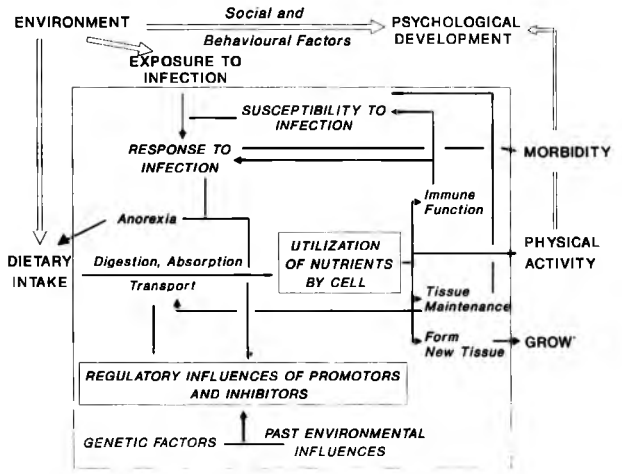
not necessarily involve the mechanisms of growth but the diagram does recognise the possibility of a link between diet and psychological development mediated through physical activity of the infant and care-giver as a response to level of total food intake.

The important realisation that comes from this type of schematic is that after growth faltering has occurred, small size continues to be a marker of the environment in which growth faltered. Because that environment really involved multiple biological and social insults, deviant anthropometry remains a powerful indicator of the disadvantaged child. For that purpose it is not important whether or not it marks dietary inadequacies in that early period - only that it continues to mark a child at risk.

It was this type of thinking that set in place the major conclusion of this paper, presented earlier.

FIGURE 2

Schematic portrayal of possible linkages between environmental and genetic factors affecting human growth and development.



Taken form an SCN report (Beaton et al, 1990)

Being small is not the problem
It is becoming small that is the problem

Clearly genetically small children do not present the same cluster of risks that are associated with environmentally determined smallness.

The SCN report (8) made a strong assertion that a clear distinction must be made between smallness, meaning short stature, and thinness. While the report argued that in older children, short stature marked a disadvantaged history and as such really said little about current nutritional status, weight for length is a marker of current state of chronic energy balance. That was not a new observation but it was one that needed to be emphasised.

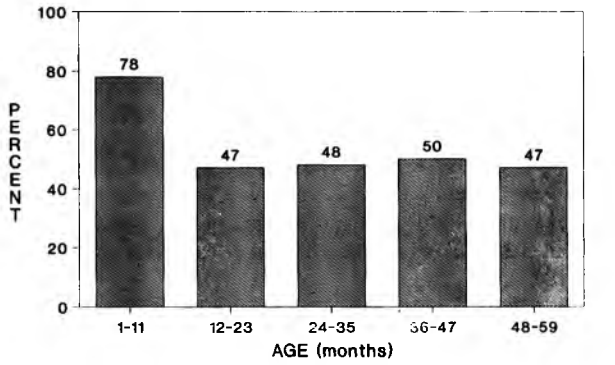
Having become relatively satisfied with the conceptual framework that had emerged, this author was left with a question that John Waterlow had been attempting to address for many years (17) If indeed there is a period when failing to grow is an active process, followed by conversion to a state of having failed to grow, what is the vulnerable period? What is the age of transition? There is no clear answer to the question but there are some strong empirical hints.

In the 1993 paper on targeting of supplementary feeding, an attempt was made to address this question with the fragmentary data then available.

The Nutrition CRSP data (Fig 1) suggested that growth faltering started before 5 months, was very active in the 6-12 month period and appeared to be essentially complete by 18-20 months. By the time of the SCN workshop, Zerfas and Teller (18) provided access to an unpublished analytical review. They had reviewed anthropometric data from 22 countries. The data was presented in narrower age intervals than had been the custom. By looking at the data this way, Zerfas and Teller documented a recurring pattern. Fig 3 presents data for one of the countries, Uganda, reviewed in their report. What was common to most of the countries was that the prevalence of non-deviant body weights (the reverse portrayal to that usually presented) begins to fall very early in life and hits bottom, in most cases, around two years of age and in all cases by three years or so. The deviation, in Z score units, then remains relatively constant across increasing age. The implication is that growth faltering occurred early and then the state of having failed to grow, continued.

FIGURE 3

Observed weights in surveys in Uganda. Data expressed as proportion of children with weights above the - 2 SD cut-off for «malnutrition».



"Adequate" = over - 2 SD of reference

Based on data presented by Zerfas and Teller (1990)

If one then looks at expected growth rates, estimated, for example, as the rate of change in median lengths in the NCHS reference data sets, one sees that the hypothesised period of active growth failure coincides with the period of very active growth in industrialised countries. The delay in detection of onset of apparent faltering to 4+ months may reflect a protective effect of breast feeding for those early months in developing countries. It may also reflect the fact that faltering begins earlier but there is a delay before the cumulative effects can be detected with statistical significance.

There is another type of suggestive evidence. That comes from a re-examination of the data from supplementary feeding studies. Gopalan, in India, reported a study (Gopalan et al, 1975) of supplementary feeding of older children. Some results are presented in Table 1. Three features of that study are very interesting today. First, the positive effect of supplementary feeding was seen primarily among children who developed measles - the extra food seemed to compensate for the otherwise negative impact of disease. This observation is consistent with a much more recent report by (19-20) that breast feeding seemed to have its greatest positive impact on early growth in the situation of high exposure to an adverse environment favouring disease. They suggested that nutrition (diet) was compensating for the effect of disease.

TABLE 1
Gain in Length During Supplementary Feeding, Hyderabad, India (treated - control)

Age (years)	Difference in Rate of Height Gain (cm / 14 months)
1 - 2	2.8
2 - 3	1.7
3 - 4	1.7
4 - 5	1.1

Based on Gopalan et al (1975)

The second interesting aspect of the Gopalan study was that the supplementary feeding managed to maintain growth rates at the median expected for that age in the NCHS data. That is, while the study demonstrated that diet was important in maintaining normal growth, there was no evidence of catch-up from past growth faltering the children were small and remained small but dietary intervention prevented them from becoming smaller..

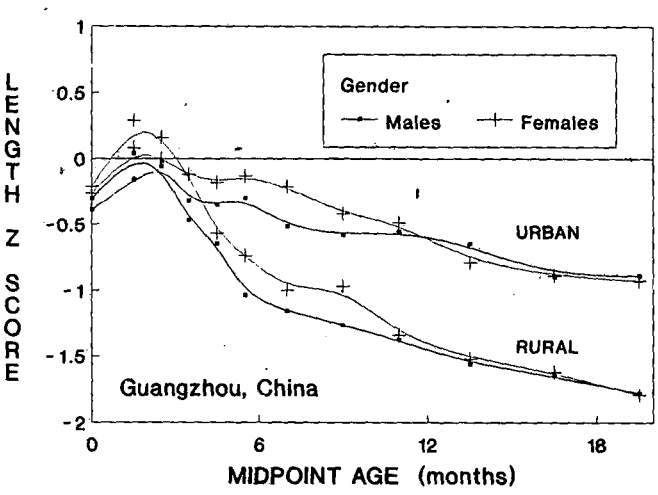
The third aspect of the Gopalan study warranting attention is that in this circumstance (effect of measles on linear growth), the effect of dietary intervention was seen long after the end of the period of active growth failure suggested above. This implies that there is no absolute end to environmentally determined growth failure and responsiveness to diet, at least as long as the bone epiphyses have not closed.

Coming closer to home, one can look at the data from the studies conducted by Mora and colleagues in Colombia and analysed by Lutter (20). While an effect of supplementary feeding was well documented, the intervention did not raise growth rates above the NCHS median rate and did not appear to provide catch up from past failures. Further, since the median expected growth rate is a function of age, the absolute effect of supplementary feeding was also a function of age - just as the SCN report predicted when it proposed an age range for an active process of failing to grow.

Another, very important, piece of evidence arises from the INCAP long term follow-up of children in communities that had been the site of nutrition intervention trials. It has been shown that anthropometric data collected at three years of age was predictive of body size, and of some functional parameters when these children were young adults. Perhaps the most important finding was that early growth faltering from whatever cause, predicted function much later in life - becoming small WAS important. The second important contribution of this study was its provision of indirect evidence that after three years of age, environmental factors (including some continuation of supplementary feeding) appeared to have had minimal impact on growth and did not overcome the predictive power of the anthropometric status at three years of age. Note however that supplementation did not continue throughout life. The absence of «catch-up» could mean simply that the children remained in equilibrium with an environment of deprivation. Martorell (21), reviewed the literature and concluded that there was very little evidence that catch-up growth occurs in later childhood or adolescence among children remaining in the same environment. Golden (22) expressed the view that reversibility was biologically possible and could be seen when the environment was changed. He pointed to a possible role of continuing micronutrient inadequacies as factors preventing catch-up growth in the developing country situation. There was little or no direct evidence to support his optimistic view.

As a third line of evidence only one example is presented, but it is a very interesting one. It has long been questioned whether the typical small body size of Chinese populations represent a true genetic determination of growth and achieved size rather than an environmental influence. When data from Guangzhou China are expressed as Z scores (Fig 4), the picture remarkably similar to that seen in other parts of the world. It appears that infants in Guangzhou show 'growth faltering' in the same age window as other regions of the world.

FIGURE 4
Observed lengths of infants and children in Guangzhou, China. Data were collected as part of a national survey and were made available by Ho Ping



Data provided by Ho Ping, 1990

In China, two major anthropometric surveys were carried out in 1975 and 1985, ten years apart (Zhang and Huang, 1988). If one then examines the changes seen in China between these two surveys, age-specific changes in anthropometric measures can be estimated. It appears that a major change occurred in the 6-12 month age group and that this change was then carried through subsequent ages. That is, both lines of evidence seem to suggest that in mainland China there is a major change underway and that something in the environment of early physical growth is reducing the traditional growth faltering. This cannot be a genetic factor. The data suggest that the original constraint to growth was in the same age interval as in other settings and that the impact of change was to relieve the growth constraint in this specific age interval. It was a preventable growth faltering. Larger sizes of older children in the second survey would seen to reflect progressively increasing relief from the early growth failure (23).

We then have three lines of evidence all suggesting the same thing.

There is an age interval, probably beginning by 4 months or even earlier and perhaps ending by two years, and certainly by three years, when growth is subject to important influence by environmental variables including both infection and diet. After that time, it is only

in severe situations that growth is affected although weight for length can be affected at any age. It is unlikely that significant catch-up growth (recovery from earlier growth faltering) occurs after about 2-3 years. Body composition and undoubtedly many body functions remain sensitive to dietary adequacy but stature is no longer a good index of current nutritional state - or of response to dietary intervention!

This has many implications, both theoretical and programmatic. One lies in the interpretation of secular trends. I suggest that secular trends in achieved stature reflect conditions that were present when the child was 6-12 months old. It follows that there is an expected lag between improvement of conditions and observation of changes in achieved stature in older children. This suggests that we might gain additional insights if we re-examined secular trends, building in expected lags and then began linking that to known historical events in the community or country.

Another obvious implication relates to growth monitoring. If you accept the model presented in this paper, then you will be compelled to accept also the inference that monitoring change in length will have its greatest potential power during the first year or two of life. After that time monitoring weight or weight for length will continue to serve a useful purpose in marking children who are getting into trouble.

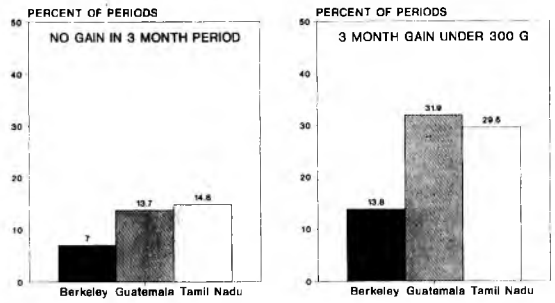
In turn this raises some considerable practical difficulty. The arguments presented in this paper rest on looking at aggregated data - mean anthropometric measures for groups. It also involves 'after the fact' observation. Conversely growth monitoring involves the use of recent history to predict the future - it is very very difficult to say what marks the beginning of a true faltering of growth and what is simply normal variability of growth rates.

Early growth and physical development has been studied intensively in paediatric centres in industrialised countries. There is considerable variation in growth rates both between and within individual infants. Normal healthy infants exhibit spurts of linear growth and periods of little or no change in length, although weight may increase. It may be extremely difficult to distinguish between this normal phenomenon and the early stage of a true faltering of growth.

The practical implication of these fluctuations is beautifully illustrated in a very recent publication by Martorell and Shekar (24). Taking the criteria of growth faltering developed for the large Tamil Nadu, India, intervention project, they applied them to historical data from three populations of children: one in Tamil Nadu, one in Guatemala and one in Berkeley, California. Shown in Fig 5 (right side) are the proportions of all three month weight increments that were less than 300 g. As would be expected, there was a greater incidence of 'growth faltering' in Tamil Nadu and Guatemala than in Berkeley. But even in the Berkeley middle-upper income group which, as adults, was above the average national size, the apparent prevalence of growth faltering in single three-month periods was 13.8%. If the proportions of periods with no net change in weight are compared (Fig 5, left), a similar picture emerges. The apparently healthy Berkeley infants showed 7% of periods with no net change in weight. Seen as a problem of sensitivity and specificity, the authors point to the very real difficulty of establishing appropriate cut-offs. Unfortunately they do not present analyses for the 6-12 month age group when growth faltering might be more serious and perhaps more evident. Not shown in the figure are the proportions of infants showing high rates of gain (over 500 g) in the same three month periods - 74.2% in Berkeley vs 49.2% in Guatemala and 38.2% in Tamil Nadu. It may be that the important difference was in the proportions of infants that could achieve the peak short term growth spurts.

FIGURE 5

Weight changes seen in 12-24 month old children (proportions of all periods)



Drawn from data in Martorell and Shekar (1994)

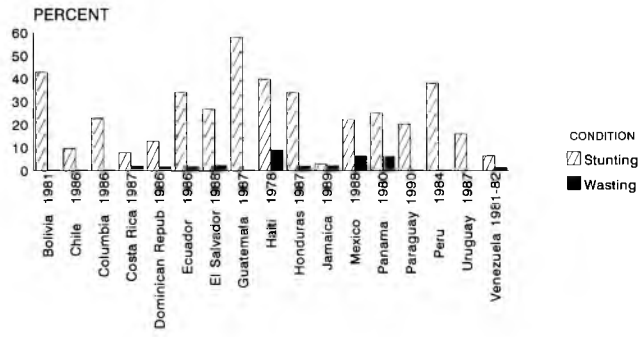
Based on data presented by Martorell and Shekar (1994).
Drawn from data in Martorell and Shekar (1994)

The major point, however, remains the same. It is much easier to see the phenomenon of growth faltering in retrospect than to see it prospectively.

When we look at data such as those shown in Fig 6, we are left with an unavoidable conclusion: the problem of 'failing to grow' and the deleterious environment that it marks is prevalent in many countries represented at this meeting. We cannot - we must not - assume that the phenomenon is related solely or even primarily to inadequate dietary intakes. However we must accept that the existence of growth faltering marks a public health problem of the first order and that it demands concerted attention and action.

FIGURE 6

Reported prevalence of wasting and stunting in young children in Latin America and the Caribbean. Plotted from data summarised by Musgrove (1993)



Taken from Musgrove, 1993

CONCLUSIONS

Although this paper emphasises some recent changes in approaches to interpretation of anthropometric data, certain truths remain unchallenged:

1. unusual smallness marks multiple types of risk to the individual and also marks a household at risk
2. after 2-3 years, short stature is a marker of history and not current nutritional status
3. in younger ages, say 6-18 months, diet might be expected to have a greater and more easily detected influence on growth.
4. We must not fool ourselves by our relatively casual usage of the term 'malnutrition' when we really mean 'smallness'

Anthropometry remains an extremely important tool in assessing and monitoring conditions in developing countries. However, we must be careful in how we interpret and apply these measures.

REFERENCES

1. ACC/SCN (1987) First Report on the World Nutrition Situation.
2. ACC/SCN (1989) Update on the Nutrition Situation: Recent Trends in Nutrition in 33 Countries.
3. ACC/SCN (1992) Second Report in the World Nutrition Situation. Vol I. Global and Regional Results
4. ACC/SCN (1993) Second Report in the World Nutrition Situation. Vol II. Country Trends, Methods and Statistics.
5. ACC/SCN (1994) Update on the Nutrition Situation, 1994.
6. Beaton, G.H.(1989) Small but healthy? Are we asking the right questions? Human Organisation 48, No. 1: 884-888.
7. Beaton, G.H.(1993) Which age groups should be targeted for supplementary feeding? in Nutritional Issues in Food Aid ACC/SCN Symposium Report Nutrition Policy Discussion Paper No. 12; pp 37-54.
8. Beaton, G., Kelly, A., Kevany, R., Martorell, R., Mason, J. (1990) Appropriate Uses of Anthropometric Indices in Children: A Report Based on an ACC/SCN workshop. ACC/SCN State-of-the-Art Series Nutrition Policy Discussion Paper No. 7.
9. Beaton, G.H., Ghassemi, H. (1982) Supplementary feeding programs for young children in developing countries. Am J Clin Nutr 35, supplement, 864-916.
10. WHO (1965) Nutrition in Pregnancy and Lactation. Report of an Expert Committee. WHO Tech Rept Ser No, 302.
11. Pelletier, D.L. (1994) The relationship between child anthropometry and mortality in developing countries: Implications for policy, programs and future research. J Nutr 124, Supplement: 2047S-2081S.
12. Seckler, D. (1979) Small but healthy? Some basic problems in the concept of malnutrition. Mimeographed paper.
13. Seckler, D. (1982) Small but healthy: A basic hypothesis in the theory, measurement and policy of malnutrition. in Newer Concepts in Nutrition and Their Implications for Policy. P.V. Sukhatme (ed) Pune, India: Maharashtra Association for the Cultivation of Science Research Institute. pp 127-137.
14. Calloway, D.H., Murphy, S.P., Beaton, G.H. (1988) Food Intake and Human Function: A Cross-Project Perspective of the Collaborative Research Support Program in Egypt, Kenya and Mexico. Univ of California, Berkeley.
15. Musgrove, P. (1991) Feeding Latin America's Children: An Analytical Study of Food Programs. World Bank, Latin America and the Caribbean Technical Department, Regional Studies Program Report 11, Washington, D.C.
16. Musgrove, P. (1993) Feeding Latin America's Children in Nutritional Issues in Food Aid ACC/SCN Symposium Report Nutrition Policy Discussion Paper No. 12; pp 58-75.
17. Waterlow, J.C., Sch,rch, B. (eds) (1994) Causes and Mechanisms of Linear Growth Retardation. Proceedings of an I.D.E.C.G. Workshop held in London, Jan 15-18, 1993. European J Clin Nutr 48, Supplement 1.
18. Zerfas, A.J., Teller, C.H. (1990). Cross-Country Comparisons and Intra-Country Differentials in Nutritional Status and Infant Feeding: Presentation from DHS in 22 Countries and Recommendations. Unpublished Report. LTS Corporation. Chevy Chase, MD.
19. Lutter, C.K., Mora, J.O., Habicht, J-P., Rasmussen, K.M., Robson, D.S., Sellers, S.G., Super, C.M., Herrera, M.G. (1989) Nutritional Supplementation: Effects on Child Stunting Because of Diarrhoea. Am J Clin Nutr 50:1-8.
20. Lutter, C.K., Mora, J.O., Habicht, J-P., Rasmussen, K.M., Robson, D.S., Herrera, M.G. (1990) Age-specific Responsiveness of Weight and Length to Nutritional Supplementation. Am J Clin Nutr 51: 359-364.
21. Martorell, R., Khan, K., Schroeder, D.G. (1994) Reversibility of stunting: epidemiologic findings in children from developing countries. European J Clin Nutr 48.Supplement 1: S45-S57.
22. Golden, M.H.N. (1994) Is complete catch-up possible for stunted malnourished children? European J Clin Nutr 48, Supplement 1:S58-S71.
23. Beaton, G.H. (1992) Nutrition Research in Human Biology: Changing Perspectives and Interpretations. Am J Human Biology
24. Martorell R., Shekar, M. (1994) Growth-faltering rates in California, Guatemala and Tamil Nadu: Implications for growth monitoring. Food and Nutr Bull 15:185-191. United Nations University.