Growth retardation starts in the first three months of life among rural Guatemalan children

J Rivera[1] and MT Ruel[2]

[1]*National Institute of Public Health, Av. Universidad No. 655, Col. Sta. Maria Ahuacatitlan CP 62508, Cuernavaca, Morelos, Mexico and* [2]*International Food Policy Research Institute (IFPRI), 1200 Seventeenth Street, N.W., Washington, D.C. 20036-3006, USA*

Objective: We tested the hypothesis that growth faltering in rural Guatemala starts earlier than between 3–6 months of life, as generally assumed.

Methods: The sample included children from the INCAP longitudinal trial (1969–1977), who had adequate birth weight (> -1 s.d.) ($n = 79$). Two groups were formed according to weight-for-age (WAZ) at 3 y: Group A: WAZ < -2 s.d. (growth-retarded), and Group B: WAZ ≥ -2 s.d. Weight increments were computed and sex- and gender-specific deficits in weight increments from 0–36 months were calculated by comparing values of the WHO/CDC reference data. For the period between 0–12 months, weight increments were also compared to velocity standards: (1) the Fels data and (2) the WHO growth curves for breast fed infants.

Results: At 3 y of age, growth-retarded children were 3.6 kg smaller than the WHO/CDC median. Depending on the reference data used, between 19 and 34% of the deficit at 3 y of age was due to failure to thrive during the first 3 months of life, an additional 12–19% occurred between 3 and 6 months and 12–25% between 6 and 9 months. By 12 months of age, infants had accumulated 45–80% of their total deficit in weight at 3 y of age. Compared to group B, children from group A had greater morbidity during their first 9 months of life, and their mothers had poorer nutritional status at 3 months postpartum. There were indications that children from group A came from more deprived families.

Conclusions: Growth faltering starts soon after birth in rural Guatemala and thus, effective interventions should be targeted to mothers and their infant as early as possible during the first year.

Sponsorship: Support for data analysis was provided by NIH (grant # HD22440) and by the Pew Charitable Trusts (grant # 92-027216-000).

Descriptors: growth; growth faltering; Guatemala

Introduction

Growth retardation in developing countries is assumed to start between 4–6 months after birth, when low-quality and often contaminated foods are introduced in children's diets (Martorell *et al*, 1994; Beaton, 1993; Waterlow, 1988). High rates of infections and substitution of breast milk by foods of low nutrient density are thought to be the main determinants of growth faltering in the first year of life (Martorell and Habicht, 1986). It is generally thought that in countries like Guatemala where exclusive or predominant breast feeding is almost universal, the early post-natal growth of infants with adequate birth weight is comparable to that of infants from developed countries.

Cross-sectional analyses of data from rural Guatemala suggest that growth retardation is already highly prevalent among 3 month old infants (Ruel *et al*, 1995), and that up to 34% of children have low weight-for-age at 36 months of age. The purpose of the present research was to study longitudinally the timing of growth retardation among Guatemalan children who were born with adequate weight, but who became growth-retarded during their first three years of life.

Received 7 July 1996; revised 7 October 1996; accepted 27 October 1996

Methods

Data and sample

The data were collected by The Institute of Nutrition of Central America and Panama (INCAP) during a longitudinal supplementation trial conducted in rural Guatemala between 1969 and 1977. Detailed description of the study is presented elsewhere (Habicht and Martorell, 1992); a brief summary follows.

Four villages were randomly assigned to receive either a high-calorie, high-protein drink (*Atole*, which contained 381 kJ (91 kcal) and 6.1 g of protein per 100 mL), or a low-calorie, non-protein drink (*Fresco*, which contained 138 kJ (33 kcal) per 100 mL and no protein). The supplements were made available at a central location to all pregnant and lactating women and to all children 7 y of age or younger.

Children who received the *Atole* supplement grew significantly better than children from the *Fresco* villages (Rivera *et al*, 1995). Therefore, only children who received *Fresco* (control group) were included in the analyses because the objective of the present study was to describe usual patterns of growth retardation in this population. Consumption of *Fresco* contributed only marginally to the daily energy intake of children, providing on average 63 kJ (15 Kcal) per day. Thus, the sample consisted of children from the *Fresco* villages whose birth weight was

greater than −1 s.d. from the median of the WHO/CDC reference data (WHO, 1979), and who had complete weight data from birth to 36 months of age (at 11 age points: birth 3, 6, 9, 12, 15, 18, 21, 24, 30 and 36 months; $n=79$). Infants with low birth weight (lbw) were excluded from the analyses because lbw infants usually have a different pattern of early post-natal growth than infants with adequate birth weight (Villar *et al*, 1982, 1984; Tenovuo *et al*, 1987).

Variables

Trained field workers recorded the infants' weight and length (to the nearest 10 g and 1 mm, respectively), using standardized procedures (Lohman *et al*, 1988; Habicht, 1976).

Other variables used in the analyses included: breast feeding duration (in months), as reported by the mother during bi-weekly home visits (this refers to any breast feeding); home diet: mean daily energy intake from home diet (kJ/d), estimated by multiple 24 h recalls conducted with mothers every three months between 15–36 months of age; morbidity: percent days ill with diarrhea, respiratory infections, or fever between birth and 3 y of age, collected by recall every 2 weeks during home visits; maternal anthropometry (weight, height, and body mass index (BMI)), parity, number of years of schooling; and family socioeconomic status derived from Principal Components analysis using data on housing quality and family possessions (Rivera *et al*, 1995).

Analytical methodology

The sample was divided into two groups, based on children's nutritional status at 36 months of age. Children with weight-for-age below −2 s.d. of the median of the WHO/CDC reference population formed group A (growth-retarded), and the rest of the sample formed group B (less growth-retarded). Mean attained weight at every age point was compared between the groups, and with the WHO/CDC reference data. Weight increments were computed for the following age intervals (0–3, 3–6, 6–9, 9–12, 12–15, 15–18, 18–21, 21–24, 24–30 and 30–36 months. Gender- and age-specific expected increments were obtained using the difference between median weight values for the reference population. Deficits in weight increments relative to expected were then obtained for each child and each age interval. The WHO/CDC reference data for attained weight were used as opposed to velocity standards (Roche *et al*, 1989; -WHO Working Group on Infant Growth, 1994), because the latter only provide data for the first 12 months of age. For this period, however, we compared weight increments to the velocity standards developed by Roche and collaborators (1989) using the Fels data, and with the recently developed growth curves for breast fed infants (WHO Working Group on Infant Growth (1994)). Because there were no data available for the 0–1 months interval with either velocity standards, the average of the 1–3 month values were extrapolated over the 0–3 month period.

Differences between groups A and B in weight increments and in child and family characteristics were tested by *t*-test.

Results

Table 1 presents the main characteristics of the sample, by nutritional status at 3 y of age. As expected, the groups were statistically significantly different in all three indicators of nutritional status at 3 y of age. Growth-retarded children also tended to have lower mean birth weight, lower energy intake for home diet, greater morbidity (fever and diarrhea), shorter mothers, larger family size and lower socioeconomic status than children from group B, but only differences in fever and family size were statistically significant.

Figure 1 shows that the growth of children from group B followed the same pattern as the WHO/CDC reference between birth and 3 months of age, started to deviate between 3 and 6 months and showed a frank deterioration after 6 months of age. Children who were growth-retarded at 3 y of age (Group A), however, showed a marked departure from the reference line already by 3 months of age. At 6 months of age, they were approximately 1 kg smaller than children from group B, and the size of this difference remained relatively constant thereafter.

Table 1 Characteristics of the sample, by child nutritional status at 3 y of age

Variable	*Group A ($n=29$) growth-retarded ($WAZ^a < -2$ s.d.)*	*Group B ($n=50$) less growth-retarded ($WAZ^a \geq -2$ s.d.)*
	Mean (s.d.)	Mean (s.d.)
Birth weight (kg)	3.20 (0.26)	3.33 (0.36)
WAZ[a] at 3 y (*z*-scores)	−2.49 (0.43)	−1.30 (0.56)[b]
LAZ[a] at 3 y (*z*-scores)	−3.56 (1.01)	−2.01 (0.84)[b]
WLZ[a] at 3 y (*z*-scores)	−0.55 (0.55)	−0.09 (0.59)[b]
Breast feeding duration (mo)	19.3 (5.4)	19.1 (5.7)
Diet (mean kJ/d; 15–36 mo)	3335 (1176)	3674 (1075)
Diarrhea (% d; 0–36 mo)	8.1 (6.6)	7.2 (5.7)
RI[a] (% d; 0–36 mo)	35.1 (19.3)	28.8 (21.1)
Fever (% d; 0–36 mo)	3.5 (1.7)	2.4 (1.6)[b]
Maternal height (cm)	148.3 (5.3)	149.8 (4.8)
Maternal parity	1.4 (1.0)	1.3 (0.9)
Maternal schooling (y)	1.6 (1.7)	1.6 (1.6)
Family size	9.0 (2.6)	7.7 (2.7)[b]
Family socioeconomic status[c]	−0.1 (0.9)	0.1 (1.0)

[a] Abbreviations: LAZ = length-for-age *z*-scores; WAZ = weight-for-age *z*-scores; WLZ = weight-for-length *z*-scores; RI = respiratory infections.
[b] *t*-test ($P < 0.05$).
[c] A socioeconomic status score (standardized variable: mean = 0, s.d. = 1) was derived by Principal Components Analysis, using data on housing quality and family possessions.

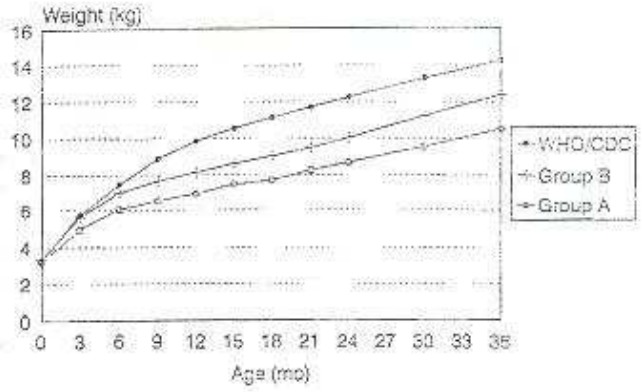

Figure 1 Patterns of growth of rural Guatemalan children, with (group A) and without (group B) growth retardation at 3 y of age (weight-for-age < -2 s.d.), compared to the median of the WHO/CDC reference data, 1979.

Table 2 presents mean weight increments, as well as the deficits in weight increments compared to the WHO/CDC median, by age interval and group. Weight increments were consistently smaller in group A compared to group B, except at ages 12–15 and 18–21 months. Differences were statistically significantly different at 4 y intervals, including the 0–3 and the 3–6 months interval. Deficits in weight increments among group A were markedly larger in the first 9 months of life, compared to later ages. At 3 y of age, children from group A had accumulated a total weight deficit of 3.6 kg compared to the WHO/CDC population. Table 2 shows that up to 19% of this deficit was due to growth faltering between 0 and 3 months of age, and an additional 19% was due to faltering between 3 and 6 months of age. By 9 months of age, children had accumulated up to 62% of the total deficit in weight observed at 3 y of age. Similar patterns of early faltering occurred among children from group B, although the deficits were generally of smaller magnitude, and some catch-up occurred after 24 months of age (as indicated by the negative deficits (Table 2)).

Figure 2 compares the results obtained for group A using the WHO/CDC standards with those obtained when velocity standards were used. It shows the cumulative percentage of total deficit acquired at different age intervals, according to the three sets of reference data used. The weight deficits among our sample of growth-retarded children were consistently greater when the Roche standards were used compared to the other two reference data. According to the Roche standards, up to 80% of the total deficit at 3 y of age was acquired by 12 months of age, compared to 53.5% when the WHO growth curves for breast fed infants (WHO-BF) were used. Irrespective of the reference standards used, between 19 and 34% of the weight deficit at 3 y of age was explained by growth retardation during the first 3 months of life, and an additional 12–19% was due to poor growth between 3 and 6 months of life. At 12 months, between 45 and 80% of the total deficit seen at 3 y of age was acquired.

Table 3 presents additional information to investigate why growth faltering occurred so early in life in this population. Morbidity from respiratory infections, diarrhea and fever was generally higher among children from group A during the first 9 months of life, although only the difference in fever between 6 and 9 months of age was statistically significant. Maternal weight and BMI at 3 months postpartum were also significantly lower among children from group A, suggesting that poor maternal nutritional status may have played a role in children's poor growth.

Discussion

Our study shows that growth faltering starts soon after birth, at least in some countries of the developing world. In Guatemala, depending on the reference standard used, between 19 and 34% of the weight deficit observed in growth-retarded 3 y old children was due to failure to thrive during the first 3 months of life. By 6 months of age, children had accumulated between 33 and 49% of the total weight deficit observed at 3 y of age (3.6 kg). This result was unexpected, considering that all children were at least predominantly breast fed and that complementary foods are not usually introduced before the age of 4–6 months in this population. All children in our sample were breast fed for at least 8 months.

One of the reasons why previous studies have failed to detect early growth faltering may be because they did not

Table 2 Weight increments and deficits[a] in weight increments compared to the WHO/CDC reference data, by age interval and nutritional status at 3 y of age

	Group A (n = 29) Growth Retarded (WAZ < −2 s.d.)				*Group B (n = 50) Less Growth-Retarded (WAZ ≥ 2 s.d.)*			
Age interval (months)	*Weight increment (kg)*	*Deficit in weight increment (kg)*	*% total weight deficit (%)*	*Cumulative percentage (%)*	*Weight increment (kg)*	*Deficit in weight increment (kg)*	*% of total weight deficit (%)*	*Cumulative percentage (%)*
0–3	1.73 (0.88)	0.67 (0.91)	18.6	18.6	2.27 (0.84)[b]	0.23 (0.84)	11.4	11.4
3–6	1.11 (0.55)	0.69 (0.55)	19.2	37.8	1.43 (0.54)[b]	0.37 (0.54)	18.4	29.8
6–9	0.51 (0.46)	0.89 (0.46)	24.7	62.5	0.65 (0.40)	0.75 (0.40)	37.3	67.1
9–12	0.41 (0.45)	0.53 (0.43)	14.7	77.2	0.46 (0.39)	0.50 (0.40)	24.9	92.0
12–15	0.52 (0.47)	0.18 (0.47)	5.0	82.2	0.47 (0.44)	0.23 (0.44)	11.4	103.4
15–18	0.24 (0.39)	0.36 (0.39)	10.0	92.2	0.44 (0.38)[b]	0.16 (0.38)	8.0	111.4
18–21	0.55 (0.42)	0.01 (0.43)	0.3	92.5	0.44 (0.47)	0.10 (0.48)	5.0	116.4
21–24	0.44 (0.42)	0.10 (0.41)	2.7	95.2	0.53 (0.53)	0.03 (0.51)	1.5	117.9
24–30	0.83 (0.48)	0.21 (0.48)	5.8	101	1.22 (0.42)[b]	−0.15 (0.43)	−8.0	109.9
30–36	0.99 (0.72)	−0.04 (0.73)	−1.1	99.9	1.16 (0.55)	−0.20 (0.56)	−10.0	99.9
Total		3.60				2.01		

[a] Deficits in weight increments were calculated comparing weight increments with age- and gender-specific expected increments. Expected increments were computed using differences between median weight values of the WHO/CDC reference data.
[b] Differences between weight increments were statistically significant by *t*-test ($P < 0.05$).

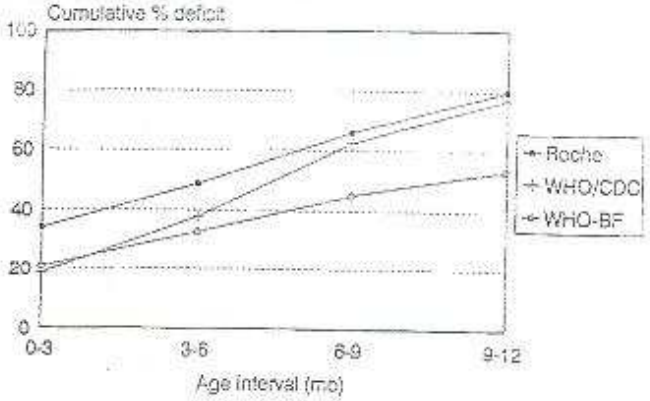

Figure 2 Cumulative percentage of total deficit in weight at 3 y of age acquired at different age intervals (group A), according to three reference data. Roche reference data are based on the Fels longitudinal growth data (Roche *et al*, 1989); the WHO/CDC reference data are attained values (WHO, 1979); the WHO-BF data are the WHO growth curves for breast-fed infants (WHO Working Group on Infant Growth, 1994).

isolate truly growth-retarded children and examine their growth pattern separately. Our study clearly showed that children who were growth-retarded at 3 y of age (WAZ < -2 s.d.) had different growth patterns than less growth-retarded children (WAZ ≥ -2 s.d.), particularly during the early postnatal period.

Our findings challenge current knowledge and understanding of the etiology and timing of growth failure in developing countries. While breast feeding is known to protect against infectious diseases (Brown *et al*, 1989; Victora *et al*, 1987; Martines *et al*, 1994), it does not appear to prevent growth retardation in poor rural Guatemalan children during the early postnatal period. It is possible that non-optimal breast feeding practices such as the use of prelacteal feeds, and teas and sugared water once lactation is established interfere with lactational performance and reduce the infants' breast milk intake. Unfortunately no information is available on the use of complementary liquids and foods before the age of 15 months for this sample.

An alternative explanation could be that breast milk is deficient in some micronutrients which limit growth during the period of predominant breast feeding. The fact that mothers of growth-faltering children had a significantly lower weight and BMI indicates a poorer nutritional status, which could affect breast milk composition or volume. The literature on this topic, however, indicates that milk volume and energy content are little affected by marginal maternal nutritional status, even at BMI values much lower than the average 21.2 kg/m^2 found in our study (Brown *et al*, 1986; Prentice *et al*, 1986). The lower infant birth weight and maternal BMI at 3 month postpartum among children from group A does suggest that maternal diet during pregnancy and lactation may have been less than optimal. This, in turn, could affect their breast milk concentration of certain vitamins, particularly the water-soluble vitamins which are more responsive to maternal dietary intake than minerals and macro nutrients (Subcommittee on Nutrition During Lactation *et al*, 1991).

Growth-retarded children also came from poorer families and spent a larger percentage of days ill during their first few months of life. Although the sample size was generally too small to detect statistically significant differences between groups, the mean values for family socio-economic status, family size, child dietary intake and morbidity suggest that growth-retarded children lived in more deprived conditions and were more prone to infectious diseases.

Our sample was purposely restricted to infants with adequate birth weight (> -1 s.d.) in order to exclude intra-uterine growth-retarded infants who usually have different patterns of growth during their first few months of postnatal life. We also excluded infants who had been exposed to the Atole treatment, because these infants grew better as a result of the intervention. Analyses done on the non-restricted sample, however, showed similar results to those presented here, with even greater deficits acquired during the first 3–6 months of life. Thus, we do not feel that the restrictions made on the sample biased our main finding that growth retardation starts soon after birth.

Studies have shown that anthropometry at 3 y of age is strongly correlated with attained size at adulthood, body composition, work performance, intellectual development and reproductive performance (Martorell, 1995). The pre-

Table 3 Maternal and child characteristics during the first 9 months postpartum, by child nutritional status at 3 y of age

Variable	*Group A (n = 29) growth-retarded (WAZ[a] < −2 s.d.)*	*Group B (n = 50) less growth-retarded (WAZ[a] ≥ −2 s.d.)*
	Mean (s.d.)	Mean (s.d.)
Child morbidity (% days ill)		
Respiratory infections		
0–3 mo	36.6 (35.8)	25.8 (27.0)
3–6 mo	38.3 (31.4)	32.6 (28.9)
6–9 mo	40.6 (30.0)	40.0 (28.7)
Diarrhea		
0–3 mo	9.0 (20.9)	7.7 (20.1)
3–6 mo	5.3 (7.0)	10.3 (14.9)
6–9 mo	12.3 (13.9)	11.9 (16.0)
Fever		
0–3 mo	4.4 (8.9)	1.9 (3.9)
3–6 mo	5.8 (7.8)	2.8 (5.4)
6–9 mo	6.8 (8.9)	2.8 (3.6)[b]
Maternal anthropometry at 3 months postpartum		
Weight (kg)	46.3 (6.2)	50.5 (6.8)[b]
BMI[a] (kg/m^2)	21.2 (2.7)	22.5 (2.9)[b]

[a] Abbreviations: WAZ = weight-for-age *z*-scores; BMI = body mass index.
[b] *t*-test ($P < 0.05$).

sent study showed, in turn, that growth during the early postnatal period is a crucial determinant of attained size at 3 y of age. Research is urgently needed to understand why growth retardation occurs so early in life, even among populations where predominant breast feeding for the first 4–6 months is almost universal. Similar analyses should be replicated in other countries where breast feeding patterns and levels of malnutrition differ in order to improve our understanding of the timing and the etiology of growth retardation, and to design cost-effective interventions to prevent early faltering.

References

Beaton G (1993): Which age group 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. Geneva: ACC/SCN.

Brown KH, Akhtar NA, Robertson AD & Ahmed MB (1986): Lactational capacity of marginally nourished mothers: relationships between maternal nutritional status and quantity and proximate composition of milk. *Pediatr.* 78, 909–919.

Brown KH, Black RE, Lopez de Romana G & Kanashiro HC (1989): Infant feeding practices and their relationship with diarrheal and other diseases in Huascar (Lima), Peru. *Pediatr.* 83, 31–40.

Habicht JP (1976): Estandarización de métodos epidemiológicos cuantitativos sobre el terreno (Standardization of quantitative epidemiological methods in the field). *PAHO Bull.* 76, 375–384.

Habicht JP & Martorell R (1992): Objectives, research design and implementation of the INCAP longitudinal study. *Food Nutr. Bull.* 14, 176–190.

Lohman TG, Roche AF & Martorell R (eds) (1988): *Anthropometric Standardization Reference Manual*. Human Kinetics Publisher: Champaign, IL.

Martines JC, Habicht JP, Ashworth A & Kirkwood BR (1994): Weaning in Southern Brazil: is there a 'weaning's dilemma'? *J. Nutr.* 124, 1189–1198.

Martorell R (1995): Results and implications of the INCAP follow-up study. *J. Nutr.* 125 (Suppl): 1127S–1138S.

Martorell R, Kettel Khan L & Schroeder DG (1994): Reversibility of stunting: epidemiological findings in children from developing countries. In: JC Waterlow, B Schürch (eds). *Causes and Mechanisms of Linear Growth Retardation. Eur. J. Clin. Nutr.* 48 (Suppl): S45–S57.

Martorell R & Habicht JP (1986). Growth in early childhood in developing countries. In: F Falkner, JM Tanner (eds). *Human Growth: a Comprehensive Treatise*, vol. 3 Methodology: Ecological, Genetic, and Nutritional Effects on Growth. Plenum Press: New York, London, pp 241–262.

Prentice A, Paul A, Prentice A, Black A, Cole T & Whitehead R (1986): Cross-cultural differences in lactational performance. In: M Hamosh, AS Goldman (eds). *Human Lactation 2: Maternal and Environmental Factors*. Plenum Press: New York, pp 13–44.

Rivera J, Martorell R, Ruel MT, Habicht JP & Haas J (1995): Nutritional supplementation during the preschool years and body size and composition in Guatemalan adolescents. *J. Nutr.* 125 (Suppl), 1068S–1077S.

Roche AF, Guo S & Moore WM (1989): Weight and recumbent length from 3 to 12 months of age: reference data for 1-month increments. *Am. J. Clin. Nutr.* 49, 599–607.

Ruel MT, Rivera J & Habicht JP (1995): Length screens better than weight in stunted populations. *J. Nutr.* 125, 1222–1228.

Subcommittee on Nutrition during Lactation, Committee on Nutritional Status During Pregnancy and Lactation, Food and Nutrition Board, Institute of Medicine & National Academy of Sciences (1991): *Nutrition During Lactation*. National Academy Press: Washington.

Tenovuo A, Kero P, Pickkala P, Korvenranta H, Sillanpää & Erkkola R (1987): Growth of 519 small for gestational age infants during the first two years of life. *Acta. Paediatr. Scand.* 76, 636–646.

Victora CG, Smith PG, Vaughan JP, Lombardi C, Fuchs SMC, Gigante LP, Smith PG, Nobre LC, Teixeira AMB, Moreira LB & Barros FC (1987): Evidence for protection by breast feeding against deaths from infectious diseases in Brazil. *Lancet.* 2, 319–322.

Villar J, Belizán JM, Spalding J & Klein RE (1982): Postnatal growth of intrauterine growth retarded infants. *Early Hum. Develop.* 6, 265–271.

Villar J, Smeriglio V, Martorell R, Brown CH & Klein RE (1984): Heterogeneous growth and mental development of intrauterine growth-retarded infants during the first 3 years of life. *Pediatrics.* 74, 783–791.

Waterlow JC (1988): Observations on the natural history of stunting. In: JC Waterlow (ed). *Linear Growth Retardation in Less Developed Countries*. Raven Press, Ltd: Vevey: Nestec Ltd., New York, pp 1–16.

World Health Organization (WHO) (1979): *Measurement of Nutritional Impact. A guideline for the measurement of nutritional impact of supplementary feeding programmes aimed at vulnerable groups*. FAP/ 79.1. WHO: Geneva.

WHO Working Group on Infant Growth (1994): *An Evaluation of Infant Growth*. WHO: Geneva.