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Growth faltering in Madura, Indonesia: a comparison with the NCHS reference and data from Kasongo, Zaire

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Summary Weight and height data of Madurese children from two longitudinal studies, the East Java Pregnancy Studies I and II, are analyzed cross-sectionally and compared with the NCHS reference and a rural population in Kasongo, Zaire. A total of 1945 children are included in the analysis, giving 25,202 weight and 17,196 height measurements. Madurese children start to accumulate a weight deficit compared with NCHS data at the age of 4 months. A linear deficit is already noticeable at 1 month, increasing rapidly in the 1st year of life. This period is responsible for most of the linear deficit accumulated by the age of 5 years. Madurese children are relatively fatter than NCHS children up to the age of 12 months. Comparing the Madurese children with those in Kasongo reveals that the accumulation of their linear deficit occurs at a period when weight increments follow those of Kasongo. Since in the studied population food intake and breastfeeding could not explain the growth dynamics in the 1st year of life, it is suggested that the explanation may lie in micro-nutrient deficiency and intra-uterine development.

Introduction

Malnutrition among preschool children is still widely prevalent in developing countries. According to Unicef, 7.8% of the world's children are wasted and 42.2% are stunted.[1] Thus, short stature represents the most important expression of the inability of preschool children to express their growth potential, an indicator of poverty, the unfavourable environment in which they live and the increased health risks to which they are subjected.[2,3]

The onset, magnitude and degree of stunting differ across populations due to variations in the underlying factors. A comparison of growth data between populations of different backgrounds can contribute to the understanding of the underlying mechanisms responsible for the expression of an accumulated deficit in linear growth.

Two large databases were available for such an analysis: the growth data of children from a rural population in Madura, Indonesia and those of a rural African population in Kasongo, Zaire. This article documents the cross-sectional analysis of these data sets. We are aware of the limitations of cross-sectional data in identifying the timing of growth decelerations but they serve the purpose for comparison. The growth velocities of these children will be presented in another article.

Subjects and methods

Weight and height data of Madurese children were gathered during two longitudinal studies—the East Java Pregnancy Studies I and II (EJPS), conducted from August 1981 to December 1985 and from January 1987 to December 1989, respectively.

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The study designs for the separate studies have been published earlier.[4,5] In summary, two isolated, rural villages, located along the southern coast of the island Madura were selected for the study. Agriculture, sea and inland fishing are the main occupations of males; women participate in farming, selling fish and repairing fishing nets. The main staple foods are rice and maize, complemented by fish and, to a lesser extent, pulses. In the first phase, children entered the study at birth; in the second phase they were enrolled at any time before the age of 60 months. In the 1st year of life, weight and length measurements were taken once a month, and thereafter 3-monthly until 5 years of age. Due to the surveillance system for pregnant women and under-5 children (monthly home visits), birthweight was known for the majority of the children. Length measurement at birth was culturally not acceptable, so the first recording was done at 1 month of age. Weight was measured to the nearest 100 g and length/height (recumbent below 2 years of age) to the nearest mm. All anthropometric measurements were taken in duplicate, according to standard techniques,[6] and quality control was done 3-monthly.

For the present analysis, the data of the two studies were combined. A total of 25,202 weight and 17,196 length measurements of 1945 children, 995 girls and 950 boys, were included in the cross-sectional analysis.

Means, medians and standard deviations for weight and height for children of the same age have been calculated. Since children were not measured at exactly the same age and because we did not want to lose too many data, the following approach was used: the average weights, heights and z-scores for children by month were calculated by averaging the measurements, for the period ± 15 days. We assumed that over such a small period the growth of the children could be considered linear.

The periodic survey of the African children was conducted in the town of Kasongo, Zaire as part of a research project on the provision of rational and comprehensive health services.[7] About 25% of the adult male population had a salaried job; others were engaged in agriculture or commercial activities. All women were farmers, cultivating maize, sorghum, beans and groundnuts. The data from Kasongo were chosen for comparison because they are good quality anthropometric data of a large sample of African children, followed longitudinally. The weight and height data were collected at 3-month intervals and have been analyzed according to the same procedures as described above.[7]

EJPS data have been compared with the NCHS reference and the anthropometric data of the Kasongo child population. Z-scores for weight-for-age (WAZ), weight-for-height (WHZ) and height-for-age (HAZ) have been calculated using the NCHS reference values and the mean and standard deviations of the Kasongo sample. Differences in mean Z-scores have been tested for significance using Student's *t*-test.

The CDC Anthropometrical Software package (CASP)[8], DataEase (DataEase International Inc. Sapphire International) and Statgraphics (Statistical Graphics Corporation) were used for the calculations.

Results

Attained weight (kg) and height (cm) of Madurese children by age and sex are presented in Tables I and II.

Comparison with the NCHS reference

Weight-for-age (WAZ), height-for-age (HAZ) and weight-for-height (WHZ) of Madurese and Kasongo children, expressed in Z-scores of the NCHS reference median, are shown in Figs 1–3, respectively.

Madurese boys and girls were lighter than NCHS children from birth onwards but in the 1st 2–3 months their WAZ curve did not deviate from the NCHS reference. The period from 3 to 12 months was one of a fast and increasing weight deficit, followed by a period of less lag in weight increase from the age of 12 months to about 30 months. After this age the WAZ curve stabilized. At all ages the mean

TABLE I. Attained height and weight of girls from Madura

	Height (cms)			Weight (kgs)		
Age (mths)	No.	Median	Mean (SD)	No.	Median	Mean (SD)
0				855	2.93	2.92 (0.39)
1	249	52.7	52.94 (2.57)	273	4.00	3.99 (0.61)
2	462	55.1	55.30 (2.69)	508	4.50	4.54 (0.66)
3	525	57.0	57.16 (2.78)	590	5.15	5.21 (0.76)
4	454	59.0	59.04 (3.90)	511	5.70	5.76 (0.77)
5	463	60.4	60.49 (2.93)	523	6.15	6.18 (0.83)
6	531	62.2	62.06 (3.13)	617	6.50	6.52 (0.85)
7	457	63.5	63.26 (3.11)	523	6.80	6.82 (0.89)
8	437	64.5	64.44 (3.28)	509	7.00	6.97 (0.93)
9	538	65.7	65.74 (3.08)	625	7.15	7.25 (0.94)
10	456	66.7	66.74 (3.29)	536	7.35	7.41 (0.95)
11	431	67.5	67.59 (3.24)	502	7.45	7.52 (0.97)
12	505	68.9	68.85 (3.29)	589	7.70	7.78 (0.97)
15	272	71.1	71.35 (4.41)	334	8.45	8.32 (1.04)
18	229	74.1	73.97 (4.25)	291	8.80	8.72 (1.07)
21	212	76.8	77.12 (4.94)	272	9.50	9.31 (0.97)
24	213	79.1	79.69 (4.97)	281	10.00	9.65 (0.79)
27	227	80.1	80.53 (4.88)	303	10.00	9.70 (0.88)
30	232	81.3	81.25 (5.08)	310	10.00	10.05 (0.92)
33	216	84.0	83.52 (4.33)	279	10.00	10.29 (0.87)
36	211	86.2	85.14 (4.09)	280	10.05	10.63 (0.91)
39	233	86.4	86.39 (5.16)	293	11.00	10.83 (1.01)
42	206	87.6	87.69 (5.28)	268	11.00	11.09 (1.11)
45	172	89.5	89.98 (4.99)	222	11.00	11.56 (1.33)
48	142	91.1	91.91 (5.05)	182	12.00	12.02 (1.37)
51	130	92.6	93.38 (5.64)	166	12.00	12.27 (1.30)
54	143	94.3	94.40 (4.90)	166	12.50	12.63 (1.30)
57	142	95.2	95.98 (4.55)	161	13.00	12.94 (1.31)
60	146	96.4	96.29 (4.98)	172	13.00	13.16 (1.34)

Madurese HAZ was significantly lower than the NCHS values (except for girls at the age of 1 month who were 0.6 cm smaller) and the deficit was more pronounced than for WAZ. The accumulation of the linear deficit differed by age. From the age of 1 month, the HAZ deficit rapidly increased up to 15 months for boys and 18 months for girls. There was a recovery between 21 and 27 months and then another faltering—slower than in infancy—between 27 and 42 months, after which the HAZ curve stabilized. Girls' growth in weight and height tends to be better than that of boys.

The relatively better gain in weight than in height in the 1st 4–5 months of life resulted in fatter Madurese children than NCHS children of comparable height. The downward trend in WHZ started in the second half of infancy (crossing the NCHS median at the age of 9 months) and continued up to 27 months, after which age no further deterioration was observed.

HAZ score fell from − 0.5 to − 2.1 by the age of 12 months, with a slight further fall to − 2.7 Z-score by 60 months, indicating that growth stunting was confined largely to the 1st 12 months of life.

Kasongo boys and girls had a better WAZ than the NCHS median at birth until the age of about 5 months, but from the age of 2

TABLE II. Attained height and weight of boys from Madura

	Height (cms)			Weight (kgs)		
Age (mths)	No.	Median	Mean (SD)	No.	Median	Mean (SD)
0				894	2.98	2.97 (0.39)
1	274	53.6	53.80 (2.80)	307	4.20	4.20 (0.64)
2	472	55.7	55.82 (2.98)	543	4.80	4.82 (0.77)
3	575	58.2	58.05 (3.19)	644	5.50	5.53 (0.86)
4	487	60.1	59.87 (3.29)	559	6.10	6.13 (0.85)
5	505	62.0	61.59 (3.39)	572	6.53	6.60 (0.95)
6	553	63.6	63.12 (3.41)	632	7.00	6.99 (0.93)
7	481	64.8	64.39 (3.47)	553	7.20	7.27 (0.96)
8	462	65.8	65.46 (3.19)	533	7.50	7.44 (0.98)
9	554	67.0	66.80 (3.31)	628	7.60	7.67 (0.98)
10	465	67.8	67.53 (3.38)	528	7.90	7.85 (1.02)
11	465	68.4	68.36 (3.38)	510	8.00	7.95 (1.02)
12	503	69.7	69.62 (3.48)	559	8.10	8.11 (0.99)
15	265	71.1	71.60 (4.11)	313	8.50	8.47 (1.05)
18	224	74.2	74.24 (4.73)	276	9.00	9.02 (0.97)
21	203	76.2	76.10 (5.04)	253	9.50	9.24 (0.99)
24	213	78.7	79.06 (5.38)	272	10.00	9.68 (0.86)
27	217	80.1	81.20 (5.65)	270	10.00	9.95 (0.83)
30	241	82.9	82.75 (5.10)	303	10.00	10.23 (1.00)
33	237	84.6	84.09 (5.02)	298	10.50	10.62 (1.04)
36	195	86.2	85.49 (4.45)	255	11.00	10.89 (1.18)
39	217	86.7	86.64 (4.92)	266	11.00	11.13 (1.34)
42	194	88.2	88.15 (5.27)	244	11.25	11.58 (1.43)
45	189	90.3	90.26 (5.49)	230	11.25	11.72 (1.30)
48	157	91.6	92.15 (5.32)	180	12.00	12.23 (1.47)
51	138	94.1	93.78 (5.10)	167	12.50	12.51 (1.34)
54	145	95.0	94.93 (5.19)	166	13.00	12.81 (1.33)
57	161	96.1	95.58 (4.74)	190	13.00	12.99 (1.36)
60	158	97.0	97.12 (4.91)	192	13.25	13.33 (1.36)

months they started to lose weight until 12 months. Kasongo children stabilized WAZ earlier than Madurese children and their deficit was much smaller.

HAZ was less than NCHS throughout. There was a rapid decline in the 1st 3 months for both sexes but thereafter growth curves differed by sex. Girls remained at -1 Z-score from 4 to 12 months, then accumulated a deficit until the age of 21 months. Boys lagged behind from 3 to 12 months, after which their deficit did not increase.

WHZ was above the NCHS median at birth, increasing up to the age of 3 months to values of 0.8. Thereafter, the WHZ decreases steadily, crossing the NCHS median at 9 months. This downward trend stabilizes around 18 months of age at -0.3, followed by a gradual increase. By the age of 27 months the values fluctuate around the NCHS mean until the age of 5 years.

Comparison of Madurese and Kasongo children

WAZ and HAZ of Madurese children, expressed in Z-scores of the Kasongo values, are shown in Figs 4 and 5.

Madurese infants weighed less than the Kasongo counterparts at birth (Madurese new-

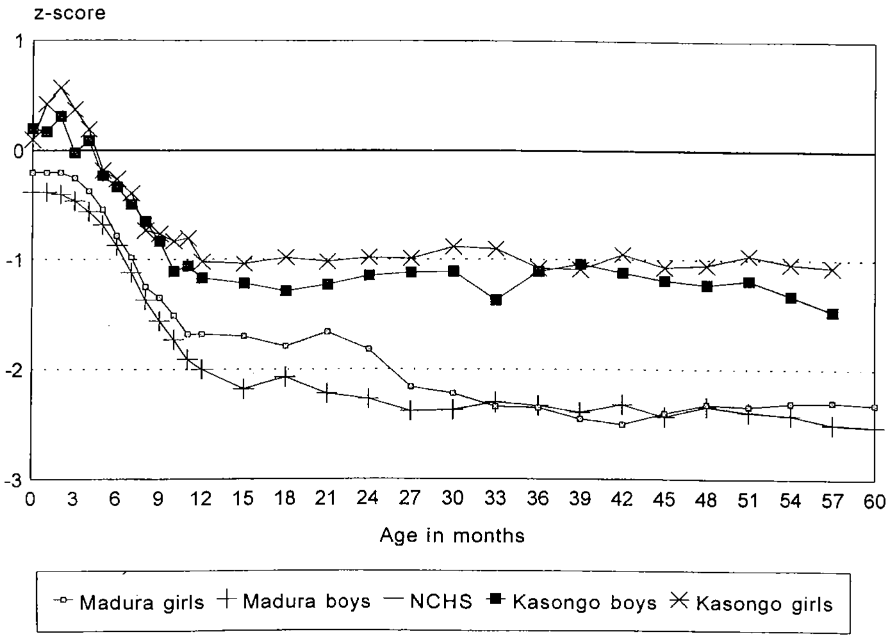

FIG. 1. Attained weight of Madurese children in Z-scores using NCHS reference values.

borns at about − 0.3 Z-score and Kasongo newborns at + 0.2 Z-score) and throughout the age range. They recovered part of the difference in WAZ between 2 and 5 months and remained at − 0.5 Z-score until the age of 11–12 months. Thereafter, they started to lag behind in WAZ until the age of 39 months. At older age, WAZ stabilized and even recovered.

The Madurese children were also shorter than the Kasongo children. Differences in HAZ were significant at all ages for both sexes, except for girls of 1 month of age. In the pattern of the Madurese HAZ curve, compared with the Kasongo reference, three distinct periods can be distinguished: (i) a fast increasing deficit from as early as the 1st month up to 9–12 months, followed by (ii) a shallow fall in HAZ up to 5 years, interrupted by (iii) a period between 21 and 30 months when Madurese children's HAZ seems to increase relative to Kasongo.

Sex differences were less pronounced in comparisons between Madurese and Kasongo children than with NCHS.

Discussion

NCHS references for weight and height are widely used in developing countries as it is largely accepted that environmental factors are largely responsible for the growth deficits observed in these countries.[9–11]

The Madurese and Kasongo growth curves and the NCHS reference pattern show some differences and many similarities. First of all, the WHZ is well above the NCHS values during the 1st year of life but an accelerating weight deficit starts after 3 months, with most of the accumulated deficit occurring in the 1st year of life.

HAZ is lower in Madura and Kasongo children from the earliest measurements onwards and rapidly decreases. Most of the linear deficit is accumulated in the 1st year of life.

Among the phenomena around 3–4 months which could explain the accumulating weight deficit shown in developing countries, the following are important. It is a period when, traditionally, weaning foods are introduced and when the feeding patterns of infants change. At the same time, infants are

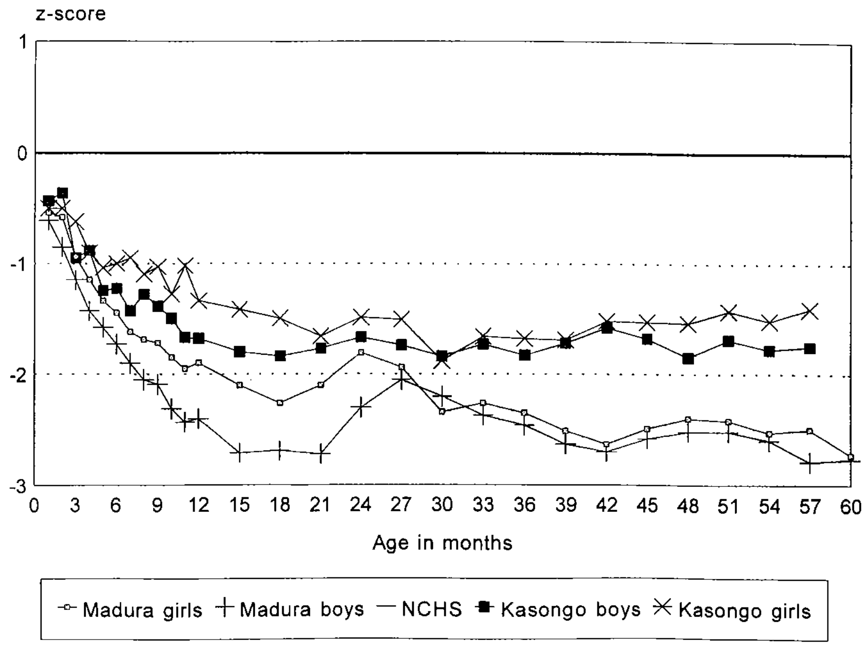

FIG. 2. Attained height of Madurese children in Z-scores using NCHS reference values.

more exposed to infections by the introduction of weaning foods at a time when there is a decline in the protection provided by maternal antibodies.

Findings in this study show that the process of stunting is less related to weight or energy reserves than assumed. Among Madurese infants, the deceleration in HAZ occurs almost from birth, when WHZ was even better than the NCHS reference and weight accumulation follows the NCHS trend. HAZ stabilizes at a WAZ of around − 2 which continues to decline. In the Kasongo population, HAZ continues to decrease after the WAZ has stabilized at − 1.

The weight and height curves of Madurese children, expressed in Z-scores of Kasongo children, allow a more detailed assessment of possible underlying factors leading to the observed growth deficits.

Madurese infants recovered their WAZ in the 1st 3–6 months and maintained it at the level at birth in the following 6 months, viz. − 0.5 Z-score of Kasongo values. Here a different picture of accumulation of weight deficit emerges, for which two explanations are plausible. (i) The 1st months of life are an adaptation to the normal growth potential of the children. Madurese infants had a lower birth weight than their Kasongo counterparts, who had birthweights higher than the NCHS reference. What we might be observing is heavier Kasongo babies finding their new growth channel and Madurese babies 'catching up'. It is known that intra-uterine growth-retarded babies tend to catch up in weight in the 1st months of life.[12–14]

On the other hand, birthweight is more an expression of intra-uterine environment than genetic potential [12–15] so that children after birth have to readjust to their growth channel.[16] (ii) Both infant populations studied were predominantly breastfed and it could be that the NCHS reference is not entirely appropriate to assess adequacy of growth in such populations. A number of authors have demonstrated that the weight increase of breastfed children differs from that of bottle-fed ones, who make up the bulk of the NCHS reference children.[17] Breastfed children in affluent societies were found to be fatter than NCHS children in the 1st months of life and

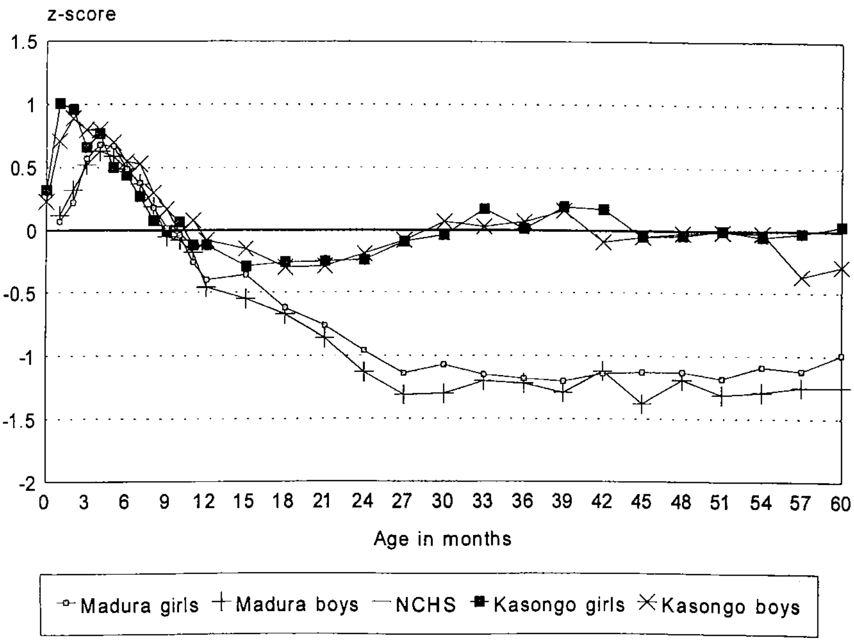

FIG. 3. Weight for height of Madurese children in Z-scores using NCHS reference values.

leaner in the 2nd half of their 1st year of life.[18–25] It is also unlikely that the decline in WAZ of Madurese infants in the 2nd half of the 1st year is attributable to an inadequacy of energy and protein intake. In a study on the consumption of breast milk and weaning foods of the same infants in Madura, no correlation was found between weight and height velocity and energy and protein intake.[4,26] Whitehead also found that the use of a reference of breast-fed children identifies different periods of weight faltering as compared with the NCHS reference.[17]

The onset of a deficit in HAZ among Madurese infants, even using Kasongo values for comparison, indicates that the linear deficit starts in the 1st month of life. Indeed, when we are able to measure a difference in accumulated deficit, the process leading to 'stunting', it is of an earlier date. The process of stunting in the 1st months of life takes place at a period when weight does not falter and follows the Kasongo and the NCHS trend. When the Madurese children lose weight compared with the Kasongo children, the linear deficit is stable. It appears that different processes affect weight and height of children. Since the deceleration in HAZ occurs very early and when WHZ is superior to that of NCHS infants, part of the phenomenon may be attributed to a continued effect of intra-uterine growth rate. Evidence indeed suggests that there is a relationship between intra-uterine development and early postnatal linear growth, the effect being stronger the earlier the fetus was exposed to nutritional stress during gestation.[15,28–33] Energy supplementation to pregnant Madurese mothers resulted in better linear growth in their offspring.[34] Other studies showed that energy and protein intake during pregnancy affect birthweight and postnatal linear growth, but their effects are limited.[34–36]

Several authors have shown that in addition to energy and protein, micro-nutrients can affect linear growth, even in developed countries.[37] Perhaps this could explain the increase in linear deficit which affected more boys than girls in the present analysis. Zinc is a micronutrient which has been shown to affect linear

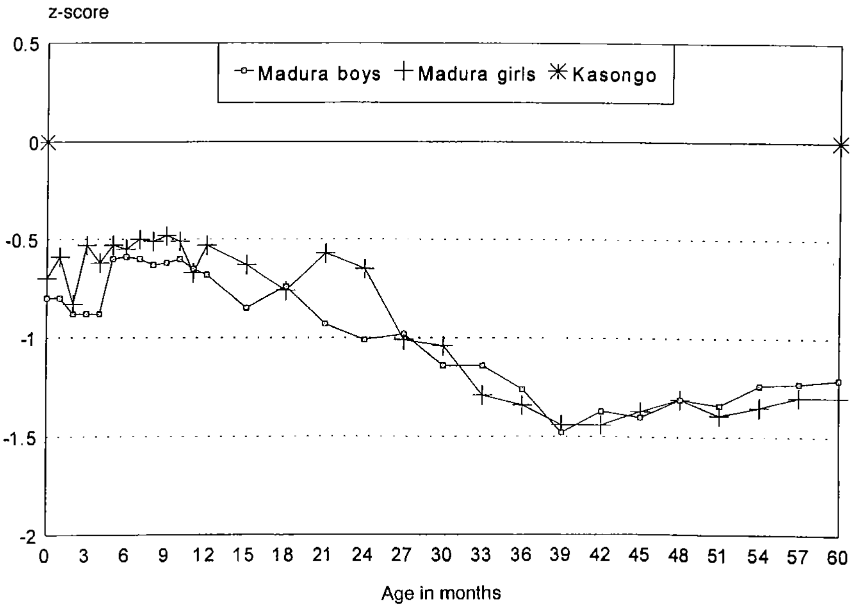

FIG. 4. Attained weight of Madurese children expressed as Z-scores using the Kasongo data as reference.

growth more often in boys than in girls.[38–43] Other nutrients, however, can also influence linear growth, such as vitamin A and iron, and interactions between micro-nutrients also occur. The effect of micro-nutrient intake during pregnancy and in the early postnatal period on the linear growth of infants should be investigated further.[44]

Differences in the accumulation of a WAZ and HAZ deficit between Madurese and Kasongo children may be explained by environmental and/or genetic factors. The latter is not likely to be a major determinant as growth curves of Indonesian children from higher socio-economic classes are comparable with the NCHS reference as is currently also observed in other Asian populations.[45,46] No data are available to determine whether the difference between the two child populations was primarily caused by a dietary inadequacy—due to poverty or socio-cultural maternal behaviour—or whether low food intake was secondary to morbidity. Among Madurese children morbidity and concomitant lack of appetite appear to be more detrimental than the amount of breast milk and food offered to the child.[5]

The better performance of Kasongo toddlers may be due to the health services introduced as part of the operational research project in this field.

After the age of 12 months, the apparent recovery in HAZ around 24 months compared with the NCHS is difficult to interpret. It could be that we are dealing with a mixed population of children measured standing and recumbent, combined with the change in reference. The fluctuations could, however, also be genuine. Many studies only ascribe what they see as changes round this age to the change in reference population, but is this really always so? The HAZ of the Kasongo population between 21 and 27 months (Fig. 2) is the average Z-score of the scores calculated using a standing and recumbent reference. This produces a smoother curve. There is definitely a need to address this question in more detail in further studies. Children should be measured standing *and* recumbent from 21 to 27 months of age.

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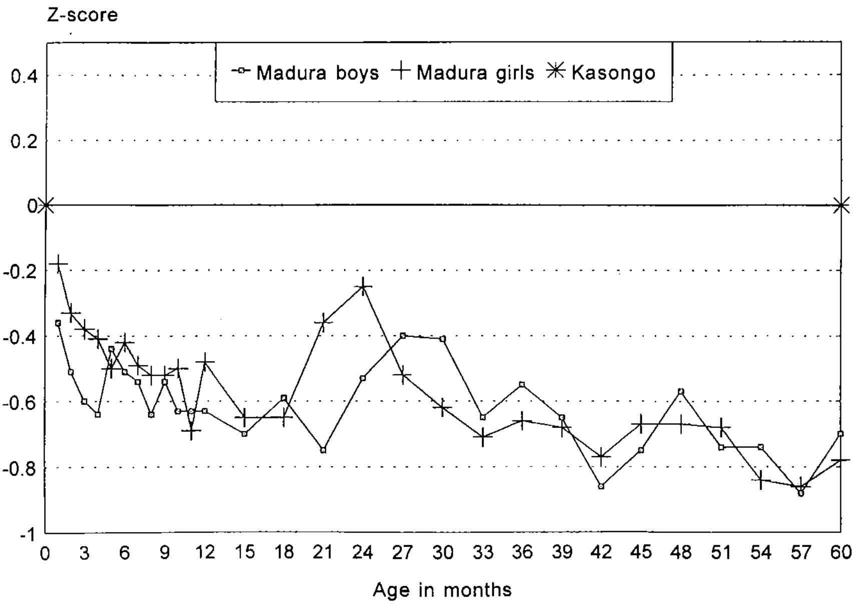

FIG. 5. Attained height of Madurese children expressed as Z-scores using the Kasongo data as reference.

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References

1 Child malnutrition. Progress toward the world summit for children goal. New York: UNICEF, 1993.
2 Tomkins A. The risk of morbidity in a stunted child. In: Waterlow JC, ed. Linear Growth Retardation in Less Developed Countries. Nestlé Nutrition Workshop Series, vol. 14. New York: Raven Press, 1988; 185–99.
3 Martorell R, Mendoza F, Castillo R. Poverty and stature in children. In: Waterlow JC, ed. Linear Growth Retardation in Less Developed Countries. Nestlé Nutrition Workshop Series, vol. 14. New York: Raven Press, 1988; 57–73.
4 Kusin JA, Sri Kardjati, van Steenbergen WM, Renqvist UH. Nutritional transition during infancy in East Java, Indonesia: 2. A longitudinal study of growth in relation to the intake of breast milk and additional foods. Eur J Clin Nutr 1991; 45:77–84.
5 Kusin JA, Sri Kardjati. Maternal and child nutrition in Madura, Indonesia. Amsterdam: Royal Tropical Institute, 1994.
6 Weiner JS, Lourie JA. Human Biology, a Guide to Field Methods. Oxford: Blackwell Scientific Publications, 1969.
7 Van Lerberghe W. Kasongo. Child Mortality and Growth in a Small African Town. London: Smith-Gordon, 1990.
8 Jordan MD. Anthropometrical Software Package. Tutorial Guide and Handbook. Atlanta: Centers for Disease Control, 1987.
9 Martorell R. Genetics, environment, and growth: issues in the assessment of nutritional status. In: Velasquez A, Bourges H, eds. Genetic Factors in Nutrition. London: Academic Press, 1984; 97–115.
10 Martorell R, Yarbrough C, Lechtig A, Delgado H, Klein RE. Genetic-environmental interactions in physical growth. Acta Paediatr Scand 1977; 66:579–84.
11 Habicht JP, Martorell R, Yarbrough C, Malina RM, Klein RE. Height and weight standards for preschool children. How relevant are ethnic differences in growth potential? Lancet 1974; i:611–14.
12 Tanner JM. Catch-up growth in man. Br Med Bull 1981; 37:223–38.
13 Stewart AL, Reynolds EOR, Lipscomb AP. Outcome of very low birthweight: survey of world literature. Lancet 1981; ii:1038–41.
14 Tenovuo A, Kero P, Piekkala P, Korvenranta H, Sillanpää M, Erkkola R. Growth of 519 small for gestational age infants during the first two years of life. Acta Paediatr Scand 1987; 76:636–46.

15 Roberts DF. Genetics of growth. Br Med Bull 1981; 37:139–246.

16 Davies DP. The importance of genetic influences on growth in early childhood with particular reference to children of Asiatic origin. In: Waterlow JC, ed. Linear Growth Retardation in Less Developed Countries. Nestlé Nutrition Workshop Series, vol. 14. New York: Raven Press, 1988; 75–90.

17 Smith DW, Truog W, Rogers JE, Reizer LJ, Skinner AL, McCann JJ. Shifting linear growth during infancy: illustration of genetic factors in growth from fetal life trough infancy. J Pediatr 1976; 89:225–30.

18 Rowland MGM, Paul AA, Whitehead RG. Lactation and infant nutrition. Br Med Bull 1981; 37:77–82.

19 Axelsson I, Moussa M. Growth of breast-fed infants. Am J Dis Child 1985; 139:219.

20 Butte NF, Garza C, O'Brian Smith E, Nichols BL. Human milk intake and growth in exclusively breast-fed infants. J Pediatr 1984; 104: 187–95.

21 Hitchcock NE, Gracey M, Owles EN. Growth of healthy breast-fed infants in the first six months. Lancet 1981; ii:64–5.

22 Salmenpera L, Perheentupa J, Siimes MA. Exclusively breast-fed healthy infants grow slower than reference infants. Pediatr Res 1985; 19:307–12.

23 Evans TJ. Growth and milk intake in normal infants. Arch Dis Child 1978; 53:749–60.

24 Duncan B, Schaefer C, Sibley B, Marques Fonseca N. Reduced growth velocities in exclusively breast-fed infants. Am J Dis Child 1984; 138:309–13.

25 Dewey KG, Heinig MJ, Nommsen LA, Peerson JM, Lönnerdal B. Growth of breast-fed and formula-fed infants from 0–18 months: The DARLING study. Pediatrics 1992; 89:1035–41.

26 Van Steenbergen WM, Kusin JA, Sri Kardjati, Renqvist UH. Nutritional transition during infancy in East Java, Indonesia: 1. A longitudinal study of feeding pattern, breast milk intake and the consumption of additional foods. Eur J Clin Nutr 1991; 45:67–75.

27 Waterlow JC. Observations on the natural history of stunting. In: In: Waterlow JC, ed. Linear Growth Retardation in Less Developed Countries. Nestlé Nutrition Workshop Series, vol. 14. New York: Raven Press, 1988; 1–16.

28 Karlberg J. A biologically-oriented mathematical model (ICP) for human growth. Acta Paediatr Scand 1989; Suppl. 350:70–94.

29 Rosa FW. International aspects of perinatal mortality. Clin Obstet Gynecol 1970; 13:57–78.

30 Girard J. Hormonal regulation of fetal growth. In: Senterre J, ed. Intrauterine Growth Retardation. Nestlé Nutrition Workshop Series, vol. 18. New York: Raven Press, 1989; 23–37.

31 Karlberg J, Engström I, Karlberg P, Fryer JG. Analyysis of linear growth using a mathematical model I. From birth to three years. Acta Paediatr Scand 1987; 76:478–88.

32 Rappaport R. Endocrine control of growth. In: Waterlow JC, ed. Linear Growth Retardation in Less Developed Countries. Nestlé Nutrition Workshop Series, vol. 14. New York: Raven Press, 1988; 109–26.

33 Milner RDG. Determinants of growth in utero. In: Waterlow JC, ed. Linear Growth Retardation in Less Developed Countries. Nestlé Nutrition Workshop Series, vol. 14. New York: Raven Press, 1988; 91–108.

34 Kusin JA, Sri Kardjati, Houtkooper JM, Renqvist UH. Energy supplementation during pregnancy and postnatal growth. Lancet 1992; 340:623–6.

35 Harrison GA, Brush G, Zumrawi FY. Interrelations between growth, weaning and disease experience in Khartoum infants. Eur J Clin Nutr 1992; 46:273–8.

36 Rona RJ. Genetic and environmental factors in the control of growth in childhood. Br Med Bull 1981; 37:265–72.

37 Prentice A, Bates CJ. Adequacy of dietary mineral supply for human bone growth and mineralisation. Eur J Clin Nutr 1994; 48 (Suppl. 1): S161–77.

38 Golden MHN. The role of individual nutrient deficiencies in growth retardation of children as exemplified by zinc and protein. In: Waterlow JC, ed. Linear Growth Retardation in Less Developed Countries. Nestlé Nutrition Workshop Series, vol. 14. New York: Raven Press, 1988; 143–63.

39 Gibson RS, Vanderkooy PDS, MacDonald AC, Goldman A, Ryan BA, Berry M. A growth-limiting, mild zinc-deficiency syndrome in some Southern Ontario boys with low height percentiles. Am J Clin Nutr 1989; 49:1266–73.

40 Bates CJ, Evans PH, Dardenne M, *et al.* A trial of zinc supplementation in young rural Gambian children. Br J Nutr 1993; 69:243–55.

41 Simmer K, Lort-Phillips L, James C, Thompson RPH. A double-blind trial of zinc supplementation in pregnancy. Eur J Clin Nutr 1991; 45:139–44.

42 Ahmed F, Barua S, Mohiduzzaman M, Shaheen N, Bhuyan MAH. Interactions between growth and nutrient status in school-age children of urban Bangladesh. Am J Clin Nutr 1993; 58:334–8.

43 Allen LH. Nutritional influences on linear growth: a review. Eur J Clin Nutr 1994; 48 (Suppl. 1): S75–89.

44 Waterlow JC. Summary of the causes and mechanisms of linear growth retardation. Eur J Clin Nutr 1994; 48:S210.

45 Chulsip K, Somnasang P, Kirdpon W, *et al.* Observations on the development of stunting in children of the Khon Kaen region of Thailand. Eur J Clin Nutr 1992; 46:475–87.

46 Agarwal KN, Agarwal DK, Benakappa DG, *et al.* Growth performance of affluent Indian children (under-fives). Nutrition Foundation of India, Scientific report 11, 1991.