Pattern of linear growth velocities of infants from birth to 12 months in Madura, Indonesia

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Summary

The paper analyses growth velocity data of infants aged 0–11 months from Madura, Indonesia, with the aim of identifying the time of onset of linear growth retardation. Velocities are calculated as average velocities for mid-point intervals, so that they can serve as comparative information for further studies. Secondly, the relation between weight and length velocities is tested. Thirdly, growth velocities are related to birthweight, length at birth and ponderal index (PI=weight/height3 × 100 in g/cm3). The anthropometric information of infants is taken from two large longitudinal studies, the East Java Pregnancy Studies Phase I and Phase II (EJPS I and II). These were conducted from August 1981 to December 1985 and January 1987 to December 1989, respectively, in two villages in Madura, Indonesia. The results support the following hypotheses: linear growth in the first year in Madurese infants shows two periods of deceleration. The early phase starts in the first month and is related to intra-uterine growth. It lasts up to about 4–6 months. Children with normal birthweight but with a low PI grow slowest in length after birth. The second period is towards the second half of the first year, when differences in linear velocity decrease with the references and velocity distribution change. Differences in weight velocity increase during this period. Factors outside the intricate fetal mother–child relationship could start to play a role.

keywords linear growth, infants, Indonesia

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Introduction

Growth retardation is a worldwide problem, (UNICEF 1993; WHO/FAO 1992; Waterlow 1994a) and perhaps the most prevalent nutritional problem today (Waterlow 1994b). In a previous study we found that children in Madura, Indonesia, had an early onset linear growth retardation (Kolsteren et al. 1996), which was already present in the second month of life when the weight-for-height Z-scores (WHZ) were well above National Centre for Health Statistics (NCHS) references. Accumulation of the linear deficit was most pronounced in the first 6 months of life, when most of the linear deficit accumulated by the age of 5 years occurs. Accumulation of the linear growth deficit also happened at a time when weight increments were comparable to NCHS children. Previous studies in the same study population showed that food intake and breastfeeding could not explain the growth dynamics in the first year of life (Kusin et al. 1991; Van Steenbergen et al. 1991). These findings prompted our study of growth dynamics using weight and height velocities.
Cross-sectional growth studies, although easier to perform than longitudinal ones, unfortunately are limited in their interpretation. They study attained growth and ‘stunted’ children to identify linear growth retardation. A group of stunted children is very heterogeneous. It may comprise (1) children undergoing the effect of decreased growth velocity at the moment of study, (2) children in a rest phase before catch-up, (3) children in full catch-up phase but still with a deficit, and (4) children who have caught up in growth to their maximal capacity given the circumstances but who have a remaining deficit. Another limitation is the time lag before deceleration in linear growth becomes apparent. For children with a decreased growth velocity it takes time to cross the cut-off line and become identifiable as being stunted (Rona 1981; Soysa & Waterlow 1981). Velocity data derived from longitudinal studies enable identification of slower growing children even if they are not stunted.

We analysed growth velocity data of infants aged 0–11 months from Madura, Indonesia, aiming to identify the time of onset of linear growth retardation. Velocities are calculated as average velocities for mid-point intervals, so that they can serve as comparative information for further studies. Secondly, the relation between weight and length velocities is explored. Thirdly, growth velocities are related to birthweight, height at 1 week and ponderal index (Hill et al. 1984; Miller & Jekel 1989; Kramer et al. 1989; Miller & Hassanein 1971).

Subjects and methods

The study and study population

The anthropometric information about infants is taken from 2 large longitudinal studies, the East Java Pregnancy Studies Phase I and Phase II (EJPS I and II), which were conducted from August 1981 to December 1983 and January 1987 to December 1989, respectively, in two villages in Madura, Indonesia. Both studies were longitudinal and community-based. A group of women were randomly selected to receive a low or high-energy supplement from about 28 weeks of pregnancy onwards. A growth-monitoring programme for their children was set up at the same time (Kusin & Sri Kardjati 1994). Children for whom more than one measurement was available during the first year of life were selected for our analysis.

In the first phase (EJPS I) infants were weighed within 24 hours of birth. Length was measured at 1 week, because it was culturally not acceptable to do so at birth. In EJPS II infants were enrolled at any age when the study started. Weight and length measurements were taken at monthly intervals thereafter.

The study design and information about the villages and their population were published by Kusin and Sri Kardjati (1994) and Kusin et al. (1991). In summary, 2 isolated rural villages on the southern coast of the island of Madura were selected. The entire population is Muslim. Agriculture, sea and inland fishing are the main occupations of males; women participate in farming, selling of fish and repairing fishing nets. The staple foods are rice and maize, complemented by fish and to a lesser extent, pulses.

Infants are breast-fed from birth. Colostrum is given and the baby is fed on demand. Virtually all infants (98%) are breast-fed up to the age of 12 months. Force-feeding by hand is common practice, starting as early as a few days after birth to about 4 months. No harmful effects of force-feeding were observed, and it did not reduce breast-milk intake (Kusin & Sri Kardjati 1994; Kusin et al. 1991).

The growth of Madurese children is characterized by the early appearance of linear growth retardation. At the age of 1 month, the height-for-age Z-score (HAZ) is \(-0.61\) for boys and \(-0.54\) for girls (NCHS references). By the age of 12 months, the average HAZ is \(-2.39\) for boys and \(-1.90\) for girls (Kolsteren et al. 1996).

Length was measured to the nearest millimetre. All anthropometric measurements were taken in duplicate according to standard techniques (Weiner & Lourie 1969). Standardization (Habicht 1973) was exercised during the initial training period and repeated every 3 months. On the same occasion, standard weights were used to check the accuracy of scales. We combined the data of both studies (EJPS I and II) and calculated indices for both by similar methods. Infants with at least 2 length measurements during the first year of life were selected. For our calculations we used the CDC Anthropometrical
Software package (CASP) (Jordan 1987), DataEase (DataEase International Inc.) and SPS PC+4.01 (SPSS Inc., Chicago).

Calculation of growth velocities

Individual growth velocities were calculated between two measurement points $H_o$ and $H_i$, 30–90 days apart. The underlying assumption is that growth, when looked at over short time-periods, can be regarded as linear. There is, however, an underestimation of the first period of the interval and an overestimation of the last period of the interval, given the typical deceleration of growth velocities over time. This deceleration is particularly important in the first months of life. But the calculated growth velocity will be quite close to the actual growth velocity for the age at mid-interval of the measurement period.

The calculations for this particular analysis were being done in the following way. Let $H_o$ be the height at the beginning of the period and $H_i$ the height at the end. The respective ages (in days) are $A_o$ and $A_i$. The calculated average height velocity per month is then 

$$v = (H_i - H_o)/(A_i - A_o) \times 30.4$$

This gives us the length increment in cm per month at the mid-interval age of the child ($A_m$), calculated as

$$A_m = (A_i + A_o)/2.$$  

Mid-interval ages were rounded to the nearest month, and average velocities computed at these rounded ages, for 1–11 months. Velocities were calculated over an interval of 60–90 days, except at 1 month of age: here the period was 30–60 days.

Analysis of growth velocity data

Mean velocities were calculated for weight and height from 1 to 11 months and compared with Fels (Roche et al. 1989) and Wroclaw (Borysławski 1988) velocity data. The latter were also used because they give computed mean velocities (and their standard deviations) for monthly periods in the first year of life, and are not smoothed, as in the Fels study. Differences between means were tested for significance using the Student t-test. The distributions of length and weight velocities were compared to a normal distribution using the Kolmogorov–Smirnov test. The percentile ratios ($p_{97}$–$p_{50}$)/($p_{50}$–$p_{3}$) were calculated as skewness indicators.

Growth velocities were further analysed by referring to Rohrer’s ponderal index (PI=weight/height$^3 \times 100$ in g/cm$^3$) (Miller & Hassanein 1971), calculated at 1 week, birthweight (BWT) and height at 1 week. We consider it as a measure of fetal growth (Hill et al. 1984; Miller & Jekel 1989; Kramer et al. 1989; Miller & Hassanein 1971).

Neonates were classified as having a low ($\leq 2.2$) or normal ($>2.2$) ponderal index following the reference values proposed by Miller and Hassanein (1971), and low (<2500 g) or normal birthweight. Height at 1 week was divided in quartile classes. Height and weight velocities at subsequent months were compared by univariate analysis of variance for the difference categories of PI, BWT and height, alone or in combination. This was done for the 303 infants for whom height at 1 week was available.

Results

Velocities could not be calculated for all infants. Sometimes the measurement points were too near or too far apart, recruitment stopped at the end of 1985 and new recruitment of neonates started in 1987. From the 1336 infants selected, 1088 velocities could be calculated, yielding a total of 5504 velocity calculations (2675 for girls and 2829 for boys) for the first 12 months. For 70% of infants, more than 3 velocity calculations were available and for 50% more than 6.

Mean length velocities of Madurese infants, compared with the Fels data (Roche et al. 1989) and the Wroclaw longitudinal study (Borysławski 1988), are presented in Table 1 and 2 and Figures 1 and 2. The growth pattern of Madurese infants is similar to the Fels data but growth velocities are consistently lower, particularly in the first 3 months after birth. The differences are significant for boys throughout the 12 months and for girls after 1 month. The proportion of infants not growing (0 millimetre per month length velocity) increases progressively with age, i.e. 3% at month 1, 5% at month 6 and more than 10% at age 9–11 months (Tables 3 and 4).

From month to month about 70% of Madurese infants change in relative position of the velocity distribution by more than 0.5 SD (data not shown). Changes are random, going up as much as going down. The velocity distribution is increasingly
skewed with age. The Kolgomorov-Smirnov test indicates that the velocity distribution can be compared with a normal distribution for both boys and girls up to 6 months. After 6 months the probability to correctly accept the null hypothesis is below 0.05 throughout. The indicators of skewness are near 1 up to 4 months, reaching a value of 1.4 for boys and 1.5 for girls at 5 months. Thereafter values fluctuate around 2.

The Wroclaw curve is different from the Fels curve, with initially increased velocities during the first 2–3 months and exceeding the Fels curve up to 7 months. Madurese infants grow more slowly than Wroclaw infants up to 6 months; thereafter, they grow at comparable speed.

Weight velocities are presented in Table 5 and 6. Compared to the Fels data, differences are small but significant except at age 5 months for boys and at
Infants were grouped in quartile classes by length at 1 week (Table 9). The shortest infants have the highest linear increase the first month whereas the tallest ones have the lowest. This negative relation between length category at one week and length velocities is noticeable with a decreasing difference between classes up to 4 months. The PI at one week also differs between quartile classes. Short infants have the highest ponderal index (2.6 vs 2.11 for the tallest group, ANOVA $F=33; P<0.001$). The differences in velocity at 1 month between quartile classes for length at 1 week decrease slightly when PI is entered as covariant in the ANOVA. The results, however, stay highly significant ($F=26; P<0.001$). Velocities at older ages are not modified when PI is entered as covariant in the ANOVA.

**Discussion**

The results indicate that growth velocities of Madurese infants are lower than the reference in the first month and that the difference is most pronounced during the first 6 months of life. Linear growth retardation is thus concentrated during the first 6 months of life in this population. Although length velocities, at the age of 1 month, are significantly lower for boys only, there probably is a difference for girls as well. One should keep in mind that we compare the velocity of the Madurese infants at 1 month with the Fels velocity from 1 to 2 months (which in our reasoning would be mid interval 1.5 months), a period when growth is already slower.

On the other hand, it is remarkable that at this age, length velocities of the Madurese infants are higher than in the Polish population. Attained height data from other populations document the largest increment between birth and the age of 1 month (Tanner et al. 1966a,b; Prader et al. 1989; Roche et al. 1989; Baumgartner et al. 1986; Borysiewski 1985). Velocity data from a Swiss longitudinal study (Prader et al. 1989) document equal growth velocities at 0.5 and 1.5 months. Unfortunately neither the Wroclaw nor the Swiss study explains these low velocity values in the first month. This phenomenon was not observed in infants in Zaire (Van Lerberghe 1990), Sudan, India, Sweden or Denver (USA) (Healy et al. 1988). Here velocity curves show the
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Table 3  Percentile distribution of growth velocities for boys of Madura and indicators of skewness (two last columns)

<table>
<thead>
<tr>
<th>Months</th>
<th>P.3</th>
<th>P.10</th>
<th>P.25</th>
<th>P.50</th>
<th>P.75</th>
<th>P.90</th>
<th>P.97</th>
<th>P97-P50/P50-P3</th>
<th>P75+P25/2</th>
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<tbody>
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<td>1</td>
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<td>1.30</td>
<td>2.48</td>
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<td>3</td>
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<td>1.93</td>
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<td>3.91</td>
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<tr>
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Table 4  Percentile distribution of growth velocities for girls of Madura and indicators of skewness (two last columns)

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<th>Months</th>
<th>P.3</th>
<th>P.10</th>
<th>P.25</th>
<th>P.50</th>
<th>P.75</th>
<th>P.90</th>
<th>P.97</th>
<th>P97-P50/P50-P3</th>
<th>P75+P25/2</th>
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<tbody>
<tr>
<td>1</td>
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<td>1.91</td>
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<td>3.2</td>
<td>0.80</td>
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highest values immediately after birth, decreasing progressively.

That the accumulation of a linear deficit is mostly concentrated in the first 6 months of life in Madurese infants is based on comparison with the Wroclaw data, which express more variability, since they report observed averages. The length velocities of British and Swiss infants (Prader et al. 1989; Tanner et al. 1966), which also represent non-smoothed data, show exactly the same trend as the Wroclaw data; the velocities are higher than the Fels ones for the first half and lower for the second half of the first year of life. The Fels data are derived from a function that averages out growth variation and ‘constructs’ a curve. Although modelling of growth data is used frequently to alleviate the problem of missing data and measurement fluctuations (Karlberg et al. 1994; Karlberg 1989), it does entail a risk of smoothing out some degree of biological growth variability. Poor fitting of the models has indeed been reported. In calculations of the velocities based on the Fels data, 8% of data were discarded because of poor fitting (Roche et al. 1989).

Although Tonglet (1994) recently found that the Infancy Childhood Puberty model (Karlberg 1989) fitted longitudinal linear growth data from an African population well, the model left 30% of the variation unexplained. This would support the assumption that real growth variability is more important than the Fels data indicate. The Polish population might therefore reflect linear growth at this period more realistically. Piwoz et al. (1992)
also pointed out the lack of variability in the Fels data and the resulting danger of misclassifying an important proportion of children as growth retarded. The indicators of skewness and the Kolgomorov-Smirnov test demonstrate that up to 4–5 months the distribution can be regarded as Gaussian. The lower mean velocity values indicate a shift in the whole population, and the factor or group of factors affecting linear growth are therefore common to the whole population in this particular period. The increase in skewness after 5 months is always positive, whereas in the Wroclaw population it fluctuates from left to right and sometimes becomes Gaussian again. This unidirectional increase in skewing in the Madurese sample could be an indication of an additional sub-population.

Whitehead and Paul (1984) reported that when using the Fels weight velocity data as reference, one
identifies growth delay earlier than when data from a European breast-fed population are used for comparison. Other studies support the finding of higher weight velocities in bottle-fed infants (Dewey et al. 1991, 1992; Duncan et al. 1984; Axelsson & Moussa 1985). The weight velocity data of Madurese infants being only slightly lower than, or identical to, the Fels data in the first 6 months, are thus not indicative of sub-optimal weight increase. The differences also become progressively smaller. Weight velocities appear to be good, particularly in the first 6 months. After 6 months, weight increments of the Madurese infants increasingly differ from the Fels data.

The process of linear growth retardation is very apparent in the first 6 months of life, at a time when weight increase can be considered adequate. This contradicts other studies reporting changes in length after changes in weight (Waterlow 1994; Martorell et al. 1994; de l’Costello 1989; Golden 1994; Walker & Golden 1988; Nabarro et al. 1988). This reported relation was, however, studied in the course of a nutrition rehabilitation activity or on more severely undernourished populations.

A number of authors have reported that weight at birth alone fails to measure fetal growth and fetal nutrition (Hill et al. 1984; Miller & Jekel 1989; Kramer et al. 1989; Miller & Hassanein 1971). They proposed that during the first part of fetal development, weight gain merely reflects cell growth. During pregnancy, an early deficiency would affect cell growth and give small infants, whereas a second-trimester deficiency would affect the degree of thinness. Ponderal index and birthweight combined

<table>
<thead>
<tr>
<th>n</th>
<th>PI &gt; 2.2</th>
<th>PI ≤ 2.2</th>
</tr>
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<tr>
<td>BWT ≥ 2,500</td>
<td>4.2 (216)</td>
<td>1.9 (57)</td>
</tr>
<tr>
<td>BWT &lt; 2,500</td>
<td>3.8 (10)</td>
<td>3.5 (20)</td>
</tr>
</tbody>
</table>

Anova: PI effect: F = 56.6, P = 0.00.
BWT effect: F = 3.68, P = 0.046.
F for the two-way interaction = 20.9, P = 0.014.

<table>
<thead>
<tr>
<th>n</th>
<th>PI &gt; 2.2</th>
<th>PI ≤ 2.2</th>
</tr>
</thead>
<tbody>
<tr>
<td>BWT ≥ 2,500</td>
<td>999 (216)</td>
<td>1026 (57)</td>
</tr>
<tr>
<td>BWT &lt; 2,500</td>
<td>763 (10)</td>
<td>1196 (20)</td>
</tr>
</tbody>
</table>

Anova: PI effect: F = 1.514, P = 0.217.
BWT effect: F = 0.012, P = 0.912.
F for the two-way interaction = 4.5, P = 0.033.

<table>
<thead>
<tr>
<th>Age (months)</th>
<th>Q1</th>
<th>Q2</th>
<th>Q3</th>
<th>Q4</th>
<th>F*</th>
<th>P</th>
</tr>
</thead>
<tbody>
<tr>
<td>1</td>
<td>4.9</td>
<td>4.2</td>
<td>3.2</td>
<td>2.6</td>
<td>2.3</td>
<td>&lt;0.0001</td>
</tr>
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<td>2.3</td>
<td>2.1</td>
<td>2.1</td>
<td>2.7</td>
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</tr>
<tr>
<td>4</td>
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<td>2.0</td>
<td>1.9</td>
<td>1.7</td>
<td>3.8</td>
<td>0.011</td>
</tr>
<tr>
<td>5</td>
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<td>1.9</td>
<td>1.9</td>
<td>1.8</td>
<td>0.3</td>
<td>0.86</td>
</tr>
</tbody>
</table>

* Q1, quartile class; Q4, lowest quartile class; Q3, highest quartile class.

* Univariate analysis of variance.

Table 7 Mean length velocities (cm per month) at 1 month by birthweight (BWT) and ponderal index (PI) class (n=number of infants)

Table 8 Mean weight velocities (g per month) at 1 month by birthweight and ponderal index class (n=number of infants)

Table 9 Mean length velocities (cm/month) at ages 1 to 5 months by quartile class for length at one week
allow a distinction between proportionate (normal PI) and disproportionate (low PI) intra-uterine retarded low birthweight infants.

Low and normal birthweight classes alone do not identify differences in linear or weight velocities at 1 month after birth in Madurese infants. This supports the previous observation by Kramer et al. (1989) that the ponderal index was a better indicator of intra-uterine growth retardation. It is the group of Madurese infants with normal birthweight and low PI who have the lowest length velocities at 1 month, and a weight velocity indicative of catch-up growth. According to the above model, this would indicate that late intra-uterine deficiency affects linear growth capacity.

Low PI infants were also the tallest. They reflect last-trimester growth retardation, when after the linear peak weight gain should be highest. They catch up in weight irrespective of birthweight, but drop in length velocity. This could be due to two phenomena: a re-channelling of growth or intra-uterine growth retardation. This last point is of even more interest if one considers that this group of infants have normal birthweights. Low-birthweight newborns are thus not the sole growth-retarded infants.

Some authors have proposed that the uterine environment determines size at birth more than the child’s intrinsic growth capacity. After birth, infants re-channel to their growth potential (Smith et al. 1976; Garn & La Velle 1984; Davies 1988). The re-channelling occurs in the first months after birth for those infants picking up. The ones ‘lagging behind’ did so towards mid-infancy, and not, as in our population, soon after birth. In the Madurese infants, this lagging is noticeable in the first month after birth. Kramer et al. (1989; 1990) also reported that for a given birthweight, intra-uterine growth-retarded infants are longer and thinner and have larger heads than normal infants. We do indeed find in the Madurese population that the wasted and tallest infants grow less well. Thus intra-uterine growth retardation rather than growth re-channelling could be the explanation (Roberts 1981; Milner 1988; Rappaport 1993; Girard 1989; Rosa 1970; Karlberg et al. 1987; Karlberg 1989).

Our reasoning points to the following possible hypothesis: Linear growth in the first year in Madurese infants has 2 periods of deceleration. The early phase, starting in the first month, is related to intra-uterine growth. It lasts up to about 4–6 months. The second period occurs towards the second half of the first year, when differences in linear velocity decrease compared to the references and the velocity distributions change. At this period, differences in weight velocity increase compared to the reference. Factors outside the intricate fetal mother–child relationship could start to play a role.

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