Postnatal Growth Patterns of Full-Term Low Birth Weight Infants in Northeast Brazil Are Related to Socioeconomic Status¹,²

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ABSTRACT · Low birth weight has many adverse consequences, some of which might be ameliorated if there is good postnatal compensatory, or catch-up, growth. We monitored growth, morbidity and feeding patterns in a cohort of 133 full-term, low birth weight infants from poor families in Pernambuco, Brazil, and investigated the relative contributions of a number of socioeconomic, maternal and infant variables to postnatal growth. Growth was measured at 4, 8, 17, 26 and 52 wk of age. Differential growth patterns were most marked during the first 8 wk of life, and the gains in z-score during this interval were strongly associated with attained z-scores at 12 months (r = 0.62 for weight and 0.64 for length). In a multivariate model, socioeconomic variables explained 21.4% of the variation in maximum gain in weight-for-age z-score achieved during the 12-month period, maternal weight explained a further 4.4%, infant birth length 4.7% and neonatal illness 5.4%. For maximum gain in length-for-age z-score, socioeconomic variables accounted for 24.4% of the variance, maternal height 4.9%, maternal smoking 3.3% and neonatal illness 3.1%. We surmise that the early differential growth patterns are set in utero and are indirectly affected prenatally by socioeconomic status.

KEY WORDS: · low birth weight · growth · human infants · socioeconomic status · z-score

In affluent populations, most infants with low birth weight (LBW)¹ are the consequence of preterm delivery and are adequately grown for their gestational age. In contrast, in developing countries the majority of low-weight births are full-term infants who have experienced growth retardation in utero, with the weight deficit reflecting stunted linear growth and/ or reduced deposition of soft tissue, notably fat. Since 70% of linear growth normally occurs by 28 wk of gestation, and fetal fat deposition occurs at the end of gestation (Falkner et al. 1994), the differing anthropometric characteristics of LBW newborns permit inferences to be made about the timing and chronicity of fetal insults. Thus stunted newborns with a normal ponderal index [body weight, g × 100/(length, cm)³] are inferred to have suffered an insult early in gestation, whereas nonstunted, wasted newborns with a low ponderal index are considered to have experienced a late insult.

Villar and colleagues in Guatemala have done much to focus attention on the differential postnatal outcomes of stunted vs. wasted LBW newborns, reporting poorer growth and development of those born stunted (Villar et al. 1984). Similar findings have been reported elsewhere (Adair 1989). However, not all stunted LBW newborns appear destined to remain small. For example, in Sweden excellent catch-up of stunted newborns has been reported (Albertsson-Wikland et al. 1993). This could be interpreted in two ways: 1) good postnatal socioeconomic conditions overcome fetal insults or 2) good prenatal socioeconomic conditions protect the fetus from the types of insult that have permanent adverse effects.

Being small at birth and remaining small during childhood is of concern because stunting is associated with reduced cognitive outcomes, school achievement and adult work capacity (Haas et al. 1996, Powell et al. 1995). Low birth weight combined with poor compensatory growth by one year of age has also been reported to be causally linked to cardiovascular disease in later life (Barker 1994). This is part of a larger concept in which it is hypothesized that the timing of the fetal insult has profound effects on metabolism, and that persistence of these metabolic changes, together with changes in the structure of organs, may predispose growth-retarded infants to hypertension, diabetes, coronary heart disease and hemorrhagic stroke (Barker 1994). Opinion is divided, however (Elford et al. 1991 and 1992, Paneth and Susser 1995), and some hold the view that the observed associations between early growth and adult disease may be the result of confounding. For example, indices used to control for socioeconomic differences may have been too crude (Ben-Shlomo and Davey Smith 1991).

In view of the importance attached to postnatal catch-up

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¹ This research was supported by The Wellcome Trust, UK (Grant No. 036605/Z/92). Pedro Lira received financial support from Fundação Coordenação de Aperfeiçoamento de Pessoal de Nível Superior (CAPES), Brazil.

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4 Abbreviations used: ABW, appropriate birth weight; LAZ, length for age z-score; LBW, low birth weight; NCHS, National Center for Health Statistics; WAZ, weight for age z-score.
growth in LBW infants, a fuller understanding of the enabling/inhibiting factors would be both advantageous and of public health interest. In 1993 we intensively monitored a cohort of 133 LBW infants living in poor socioeconomic conditions in northeast Brazil. Despite restricting participation to poor families from a relatively small catchment area, infant growth proved to be highly heterogeneous. In this paper we report the relative contributions of a number of variables to this heterogeneity.

**METHODS**

**Study population.** The study was conducted in five small towns in the state of Pernambuco, northeast Brazil. The largest of these towns, Palmares, has a population of ~60,000. The other four towns are within a 35-km radius. Economic activity of the region is dominated by the production and processing of sugar cane, and little employment is available outside this sector. Female illiteracy is ~30%, and infant mortality is almost 85/1000 live births. The population is ethnically mixed.

Study infants were recruited over a one-year period beginning January 1993 from the maternity wards of two state hospitals (in Palmares and Ribeirão), one private hospital (Palmares), and three smaller government health centers (in Catende, Agua Preta and João-quim Nabuco). There are no other maternity centers in the area, and more than 90% of deliveries occur in these facilities. The incidence of LBW (~9%) and the infants and their mothers have been described previously (Lira et al. 1996).

**Study design.** Eligible LBW newborns were term singletons who met the birth weight criterion (1500-2499 g), had no congenital anomaly, and were from families earning <4 minimum salaries (1 minimum salary is ~US$70/mo) who intended to be resident in the study area in the subsequent six months. Newborns of appropriate birth weight (ABW) were also recruited. Eligible ABW newborns met identical criteria except that their birth weights were between 3000 and 3499 g. After an initial screening for family income and intended residence, all potentially eligible babies were reweighed, measured, and examined by one of two study pediatricians. All newborns weighing <2500 g were immediately assessed for gestational age based on five maturational indices by the method of Capurro et al. (1978). Those with gestational ages of 37 or more completed weeks were invited to participate in the study activities. ABW infants were individually matched to the LBW infants by sex and season of birth, but not by place of birth. Each LBW infant was matched to the next two eligible ABW infants.

All infants in this study, both LBW and ABW, were randomly assigned to receive either 1 mg zinc daily for eight weeks from birth, or a placebo preparation similar in taste and appearance. This intervention had no impact on any of the outcome measures considered, and identical conclusions are reached controlling for treatment allocation.

**Anthropometry.** At enrollment, weights of unclothed infants were recorded to the nearest 10 g, using regularly calibrated baby scales (digital baby scale model 15/2B, Filizola, São Paulo, Brazil and beam baby scale, model 725, Soehnle, Murrhardt, Germany). Weighments were generally carried out within 12 h of birth (72% of all infants), and only 8 infants were weighed after 24 h. Crown-heel lengths were measured with a Harpenden Infantometer (Holtain, Crymych, UK). Standard techniques were again used. A difference of ≤0.5 cm was considered adequate. Infant weights and lengths were converted into weight-for-age z-scores (WAZ) and length-for-age z-scores (LAZ) using median values from the National Center for Health Statistics (NCHS) (1978) as the reference. Exact age in days at the time of measurement was used to calculate these age-standardized measures. It has previously been shown that the growth of elite infants in this region of Brazil is comparable to the NCHS reference population. Information on the infants’ family backgrounds, housing conditions, and maternal smoking, alcohol consumption and work during pregnancy was collected by questionnaire applied in the maternity center at the time of enrollment.

**Morbidity.** All infants were visited at home every day except Sundays until 8 wk of age, and twice weekly until 26 wk. The morbidity questionnaire included daily information on the presence or absence of five maternally perceived symptoms: diarrhea, cough, vomiting, fever and cancroso (the local term for rapid breathing).

**Feeding mode.** Interviewers also recorded the number of breast feedings and the number of other feedings (but excluding water, tea and juice) daily from birth to 8 wk, and twice weekly until 26 wk.

**Ethics.** Ethical permission for the study was obtained from the Ethics Committees of the London School of Hygiene and Tropical Medicine and the Federal University of Pernambuco. Mothers were fully informed about all study procedures by one of the two study pediatricians, and their consent was obtained prior to the infants’ enrollment in the study. Only one mother refused permission for her infant to participate.

**Data analysis.** In examining postnatal growth patterns of LBW infants, our aim was to quantify the extent of their catch-up growth, and then to examine possible mediating factors. Most infants improved in WAZ and LAZ initially, but some then regressed. For some, the initial improvement was very marked, and it was this early differential response that was our particular interest. Since determinants of the initial improvement are likely to differ from those mediating the later decline, we chose to focus on factors that might influence this initial improvement. For the analysis we selected as the outcome measures, the maximum gain in z-score from birth over the course of the first year, for weight and length. This gave a measure of the magnitude of the postnatal catch-up growth, while taking into account the fact that infant age at peak gain would vary.

**Statistical methods.** Hierarchical, or multilevel models (Goldstein 1987) were used to derive smoothed growth curves from the 2–6 data points available, and the maximum attained z-score was identified from the curve. The multilevel approach allows the statistical model (i.e., the formula) of data in which a variable (such as weight or length) is repeatedly measured on the same individuals, giving rise to important within-subject correlations. It is able to accommodate measurements made at unequal intervals, and is efficient even when some data are randomly missing, because data are pooled across subjects in the estimation procedure. In this analysis, a simple two-level growth curve model was used, similar to previous applications in this area (Goldstein 1986, Yang and Leung 1994). The occasions on which the measurements were made constitute the first level of the hierarchy, while the infant, taking all occasions together, constitutes the second level. Within subjects, the relationship between WAZ and age was modeled as:

\[ WAZ_i = \alpha_i + \beta_i \times \text{AGE}_i + \gamma_i \times \text{AGE}_i^2 + \delta_i \times \text{AGE}_i \]

where WAZ denotes the weight-for-age z-score for individual i at time t, and AGE denotes the age of individual i at time t. The effect of weight-for-age z-score at birth (\(\alpha_i\)), as well as the linear rate of change in weight-for-age z-score over time (\(\beta_i\)), and the rate of change as a quadratic function of age \((\gamma_i)\) were allowed to vary randomly from individual to individual. The coefficient associated with the cubic function of age \((\delta_i)\) was not allowed to vary randomly between subjects, as this was not found to produce any improvement in the model fit.

For estimating maximum gain in length-for-age z-score, individual growth curves were fitted without a cubic term in age, as this did not improve the fit of the model. The models were fitted using the software package MLwiN (Institute of Education, London, UK, Prosser et al. 1999). Goodness of fit was assessed by critical examination of the model residuals, both at level one and at level two. Infants whose z-score remained constant or deteriorated from birth were considered.
to have achieved a maximal z-score gain of 0. The few infants who were still improving in length-for-age z-score at 12 mo of age were considered to have reached maximal gain at exactly 12 mo.

The estimated maximum z-score gains were related to individual-level covariates using multiple linear regression. Possible socioeconomic confounders were introduced into the model first, with an entry criterion of $P < 0.2$. Other variables of interest were then entered into the model in a stage-by-stage process, so that the underlying hierarchies in the causal pathway would be respected (Victoria et al. 1997). In practice, this meant that maternal factors were controlled only for socioeconomic status, whereas anthropometric characteristics of the infant at birth were controlled for both socioeconomic status and maternal variables, while neonatal morbidity and feeding patterns were controlled for socioeconomic status, maternal variables and birth anthropometry. Because peak gains occurred very early, postneonatal morbidity and postneonatal feeding mode were not included in the models to minimize the risk of identifying associations resulting from reverse causality.

**RESULTS**

During the year, 133 LBW infants were recruited, with a median birth weight of 2380 g ($-1.95$ z-scores) and a median gestational age of 39 wk. Only five infants weighed <2000 g. Thirty (23%) were stunted alone (length $< -2$ z-scores, ponderal index $< 2.5$), 74 (56%) were wasted alone (length $< -2$ z-scores, ponderal index $< 2.5$) and 23 (17%) were both stunted and wasted. Six infants were neither wasted nor stunted. Of the 260 ABW infants recruited, the median birth weight was 3195 g.

**Weight gain.** Between birth and 12 mo of age (or their last anthropometric follow-up), 120/133 (90.2%) LBW infants had improved in weight-for-age z-scores. The estimated median age at peak WAZ was 77 d (interquartile range 65–90 d). For some, this catch-up was substantial and progressive, while for others it was modest and/or transient. This variation in the pattern of weight gain among the LBW infants was evident immediately after birth (Table 1); by 8 wk the standard deviation of their attained WAZ was twice that at birth, and by 26 wk it was three times as great. Attained WAZ at 12 mo ($n = 100$) was strongly associated with the gain in z-score from birth to 8 wk ($r = 0.62$, $r^2 = 0.386$). Among ABW infants, 213/260 (81.9%) also had improved in WAZ at some point between birth and 12 mo of age (or their last anthropometric follow-up), but the variation in weight gain was slightly less marked than in the LBW infants (Table 1). The mean WAZ from birth to 12 mo of age for ABW and LBW infants is shown in Figure 1, with the latter disaggregated as stunted, wasted or both. As would be expected, infants born both stunted and wasted weighed less at birth than infants born either stunted or wasted alone. By 4 wk, however, these doubly disadvantaged infants were similar in WAZ to their stunted counterparts. The 74 wasted-only infants tended to have higher WAZ than the other two LBW groups at 4 wk of age and onwards.

Associations were sought between maximal WAZ gain in the LBW infants and a number of different variables relating to the infants’ prenatal and neonatal environment. As a first
step, the infants’ socioeconomic background was examined. Seventeen variables relating to housing construction and facilities, income, father’s presence and literacy, and crowding were entered in a stepwise multiple linear regression, and those significant at the 20% level were retained. Three variables remained in the model: presence/literacy of the father (coded 1 = absent, 2 = present but illiterate, and 3 = present and literate), type of toilet facility, and ownership of a television. Together these three variables explained 21% of the variation in maximal WAZ gain, and were used to control other associations for the effects of socioeconomic confounding.

Table 2 shows the associations between maximal WAZ gain and three sets of possible early enabling/inhibiting factors: mother’s size, selected insults during pregnancy, and birth anthropometry. Effects are shown both before and after controlling for socioeconomic status, which is likely to confound all these associations. Associations between infants’ anthropometry at birth and subsequent WAZ gain are additionally controlled for mother’s weight. Mother’s weight and height were both associated with maximal WAZ gain after adjusting for socioeconomic status, with mother’s weight showing the stronger association. A 10 kg difference in mother’s weight was positively associated with a 0.2 z-score difference in maximal WAZ gain. Neither smoking nor work during pregnancy was associated with maximal WAZ gain, but both infant length and ponderal index at birth were, with a 1 z-score difference in birth length positively associated with a 0.3 z-score difference in maximal WAZ gain.

Table 3 presents the associations between maximal WAZ gain and 1) the average number of breast feedings/nonbreast feedings per day during the first four weeks of life, and 2) the proportion of all neonatal days that the infant was reported by his/her mother/caregiver to be suffering from diarrhea, cough, vomiting or fever. Neither the number of breast feedings nor the number of nonbreast feedings was associated with WAZ gain, although marked confounding was seen in the case of breast feeding (indicated by the marked change in the coefficient on controlling for the measured socioeconomic variables), and the potential for residual confounding due to other unmeasured socioeconomic variables should perhaps be considered. Proportion of time sick was strongly associated with maximal WAZ gain, with a 20% absolute difference in the proportion of neonatal days spent sick negatively associated with a 0.2 z-score difference in maximal WAZ gain.

In a final multivariate model, socioeconomic variables explained 21.4% of the variation in maximal WAZ gain, mother’s weight explained a further 4.4%, infant birth length another 4.7%, and illness in the neonatal period a further 5.4%. Thus 35.9% of the total variance of this outcome was explained by the six variables considered (adjusted $R^2 = 0.328$).

Length gain. During the course of the year, 104/133 (78%) LBW infants improved in length-for-age z-scores. Estimated median age at peak LAZ was 212 d (interquartile range 148–249 d). Variability in LAZ increased steadily with age, so that the standard deviation at 12 mo was twice that at birth (Table 4). LAZ gain over the first 8 wk was strongly associated with attained LAZ at 12 mo of age ($r = 0.64$, $r^2 = 0.405$). The mean LAZ gain for stunted infants during the first year was

### Table 2

<table>
<thead>
<tr>
<th>Variable</th>
<th>Distribution</th>
<th>Uncontrolled effect</th>
<th>Controlled effect</th>
</tr>
</thead>
<tbody>
<tr>
<td></td>
<td>Mean (sd)</td>
<td>$\beta$ (SEM)</td>
<td>$P$</td>
</tr>
<tr>
<td>Mother’s weight, kg</td>
<td>51.6 (7.6)</td>
<td>+0.0279 (0.00778)</td>
<td>&lt;0.001</td>
</tr>
<tr>
<td>Mother’s height, cm</td>
<td>152.8 (6.3)</td>
<td>+0.0291 (0.00947)</td>
<td>0.003</td>
</tr>
<tr>
<td>Mother’s body mass index, kg/m$^2$</td>
<td>22.1 (3.0)</td>
<td>+0.0358 (0.0208)</td>
<td>0.088</td>
</tr>
<tr>
<td>Smoking during pregnancy, % prevalence</td>
<td>26</td>
<td>-0.0252 (0.138)</td>
<td>-0.169 (0.126)</td>
</tr>
<tr>
<td>Working during pregnancy, % prevalence</td>
<td>24</td>
<td>+0.139 (0.144)</td>
<td>0.338</td>
</tr>
<tr>
<td>Infant’s weight, z-score</td>
<td>-2.02 (0.39)</td>
<td>+0.0662 (0.158)</td>
<td>0.676</td>
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<tr>
<td>Infant’s length, z-score</td>
<td>-1.90 (0.61)</td>
<td>+0.266 (0.0995)</td>
<td>0.008</td>
</tr>
<tr>
<td>Infant’s head circumference, cm</td>
<td>32.6 (0.84)</td>
<td>-0.0115 (0.0742)</td>
<td>0.877</td>
</tr>
<tr>
<td>Infant’s ponderal index</td>
<td>2.41 (0.19)</td>
<td>-0.699 (0.324)</td>
<td>0.033</td>
</tr>
</tbody>
</table>

1 ($n = 133$). Effects are shown before and after controlling for confounding variables.

2 Effects of mothers’ characteristics and gestational insults controlled for father’s presence/literacy, type of toilet facility and television ownership.

3 Effects of infants’ characteristics at birth controlled for father’s presence/literacy, type of toilet facility, television ownership and mother’s weight.

### Table 3

<table>
<thead>
<tr>
<th>Variable</th>
<th>Distribution</th>
<th>Uncontrolled effect</th>
<th>Controlled effect</th>
</tr>
</thead>
<tbody>
<tr>
<td>Average number of breast feedings per day</td>
<td>7.5 (4.5–10.4)</td>
<td>+0.0103 (0.0130)</td>
<td>0.428</td>
</tr>
<tr>
<td>Average number of nonbreast feedings per day</td>
<td>1.4 (0.1–3.8)</td>
<td>+0.00610 (0.0242)</td>
<td>0.802</td>
</tr>
<tr>
<td>% of time with diarrhea, cough, vomiting and/or fever</td>
<td>3.7 (0.0–17.9)</td>
<td>-0.0119 (0.00307)</td>
<td>&lt;0.001</td>
</tr>
</tbody>
</table>

1 ($n = 133$). Effects are shown before and after controlling for confounding variables.

2 Controlled for father’s presence/literacy, type of toilet facility, television ownership, mother’s weight and infant’s birth length.

3 IQR = Interquartile range (lower and upper quartiles).
In reviewing the results the following may be noted: weight was measured. Appropriate birthweight (ABW) and low birthweight (LBW) were measured. Variation in length gain among ABW infants was less marked than in LBW infants. 

Associations were sought with a variety of potential enabling/inhibiting factors, following the procedures for weight gain that were shown in Tables 2 and 3. The results of the final model for length gain are shown in Table 5. Socioeconomic factors accounted for 24.4% of the total variance in maximal LAZ gain. After controlling for socioeconomic variables, mother’s height (not weight) accounted for a further 4.9% of the variance. A 0.2 z-score difference in mother’s height was positively associated with a 0.2 z-score difference in maximal LAZ gain. Smoking during pregnancy was associated with a 0.3 z-score shortfall in maximal LAZ gain, and a 20% absolute difference in the proportion of days spent sick during the first 4 wk of life was negatively associated with a 0.1 z-score difference in maximal LAZ gain. Infant anthropometry at birth was not associated with this outcome (P > 0.1 in all cases, after adjusting for socioeconomic variables and mother’s height and smoking habit). A total of 35.7% of the total variability in maximal LAZ gain was explained by the variables considered in the model (adjusted $R^2 = 0.321$).

**DISCUSSION**

For many years, the NCHS reference population has been widely used in studies of infant and child growth and has been endorsed by WHO as the yardstick for international comparisons. Its appropriateness, however, is now seriously questioned (WHO Working Group on Infant Growth 1995). Unfortunately no satisfactory alternative reference population is currently available, hence researchers must take particular care in interpreting their findings. In order to have confidence in our interpretations, all analyses reported in this paper have been repeated using a recent, but small, pooled data set based on breast-fed infants (WHO 1994). The same conclusions regarding the determinants of differential growth patterns are obtained when this reference population is used. Although maximum WAZ and LAZ gains were estimated, there were few missing data points, and the smoothed polynomial curves captured well the observed values (data not shown). We therefore consider that our methodological approach and interpretation of the data are valid.

In reviewing the results the following may be noted: 1) WAZ gain differentials were most apparent in the first 8 wk, with some LBW infants having a dramatic initial increase, or take-off, 2) LAZ gain differentials emerged more gradually, 3) 36% of the variability in maximum WAZ and LAZ gains was explained by just six variables (three socioeconomic and three other). The rapidity of onset of differential growth patterns so soon after birth, and the asynchrony of the timing of the peak WAZ and LAZ gains, suggest that the differential response is due, at least in part, to rapid soft tissue gain. This is borne out by comparing the stunted-only group with the group that was both wasted and stunted, as shown in Figure 1. Infants in the latter group experienced accelerated weight gain initially, but then were indistinguishable from the stunted-only infants.

Despite the fact that only poor families were eligible for the study, socioeconomic status explained 21 and 24% of the variance in maximal WAZ and LAZ gains, respectively. How socioeconomic status, if acting only postnatally, could so dramatically differentiate the patterns of take-off is not clear.

**TABLE 5**

**Associations between socioeconomic indicators, maternal risk factors and neonatal morbidity with maximal gain in length-for-age z-score for infants of low birth weight: multi-stage, multivariable model**

<table>
<thead>
<tr>
<th>Variable</th>
<th>$\beta$ (SEM)</th>
<th>$P$</th>
<th>Change in $R^2$</th>
</tr>
</thead>
<tbody>
<tr>
<td>Type of toilet facility</td>
<td>-0.277 (0.0782)</td>
<td>$&lt;0.001$</td>
<td></td>
</tr>
<tr>
<td>Television ownership</td>
<td>+0.329 (0.103)</td>
<td>0.002</td>
<td></td>
</tr>
<tr>
<td>Father’s presence/literacy</td>
<td>+0.118 (0.0555)</td>
<td>0.035</td>
<td></td>
</tr>
<tr>
<td>Number of bedrooms</td>
<td>-0.103 (0.0582)</td>
<td>0.079</td>
<td>0.244</td>
</tr>
<tr>
<td>Mother’s height</td>
<td>+0.0217 (0.00729)</td>
<td>0.004</td>
<td>0.049</td>
</tr>
<tr>
<td>Smoking during pregnancy</td>
<td>-0.258 (0.104)</td>
<td>0.014</td>
<td>0.033</td>
</tr>
<tr>
<td>% of time with diarrhea, cough, vomiting and/or fever</td>
<td>-0.00069 (0.00231)</td>
<td>0.015</td>
<td>0.031</td>
</tr>
</tbody>
</table>

1 ($n = 133$).
2 Each variable controlled for the other three socioeconomic variables (type of toilet facility, television ownership, father’s presence/literacy and number of bedrooms).
3 Controlled for type of toilet facility, television ownership, father’s presence/literacy and number of bedrooms.
4 Controlled for type of toilet facility, television ownership, father’s presence/literacy, number of bedrooms and mother’s height.
5 Controlled for type of toilet facility, television ownership, father’s presence/literacy, number of bedrooms, mother’s height and smoking during pregnancy.
Whatever the ultimate controlling mechanism, differences in postnatal growth among individual infants can only involve differences in 1) dietary intake, 2) prenatal nutrient stores, 3) efficiencies of absorption and utilization, 4) basal metabolism, 5) physical activity and/or 6) morbidity. Socioeconomic status could conceivably affect dietary intake, either by influencing a) the number of breast or supplementary feedings given, b) the volume of breast milk or supplement consumed per feeding and/or c) the quality of the breast milk or supplement. Regarding a), neither the number of breast feedings nor supplementary feedings in the first month were associated with WAZ or LAZ gains, and it was during this period that differential growth patterns were most pronounced. We have no data pertaining to b) or c), but if these were mediating factors one might expect them to affect postnatal growth rather later, when nutrient demands are greater. Socioeconomic status could also influence the fetal store of micronutrients, but again, if this were the mediating factor one might expect a later effect, after existing stores have been depleted. Psychosocial care mediators are unlikely to operate so soon after birth, except through the measured feeding and morbidity variables.

Small maternal stature and low maternal weight, as well as maternal smoking and neonatal illness, were all more common in the poorest families in this population. It is therefore clear that part of the large effect of socioeconomic status was mediated by these variables, which we have shown were associated with infant growth. However, when these associations were examined by path analysis (data not shown), we found that neither the associations between these proximal determinants and the socioeconomic measures, nor the associations between these determinants and the growth outcome, were sufficiently strong to explain more than a small proportion of the overall socioeconomic effect.

Taken together, we surmise that socioeconomic status is acting prenatally, and that the early differential growth patterns are mediated through maternal factors that determine fetal linear and ponderal growth. If the outcome is a wasted LBW newborn, it is reasonable to expect that such a baby would catch up in weight-for-length in a comparable way to postnatally wasted infants. Stunted infants might be downregulated with altered gene expression, reduced cell numbers, altered hormone release and receptor sensitivity, and altered organ structure (Barker 1994, Dahlri et al. 1995, Owens et al. 1994, Widdowson et al. 1972). Interestingly, in Guatemala stunted fetal growth was associated with several socioeconomic indicators, but this was not the case for wasted newborns (Neel and Alvarez 1991). Down-regulation could explain why LBW Indian orphans born in the poorest communities but adopted soon after birth by affluent Swedish families attained an adult height comparable to Indians who had experienced lifelong disadvantage (Gopalan 1994).

In New York City, Naeye et al. (1971) compared organ weights and body size from consecutive autopsies on stillborn and newborn infants according to urban poverty indices of the US Social Security Administration. Those from families below the poverty line were shorter, lighter, and had smaller liver, spleen, thymus and adrenal glands compared with infants from families above the poverty line. These changes together with morphological differences were attributed to maternal undernutrition. Altered organ structure has been found to have marked effects on postnatal function in infants growth-retarded in utero. For example, altered intestinal and pancreatic structure adversely affects nutrient absorption (Boehm et al. 1991 and 1992, Kolacek et al. 1990) and reduced numbers of \( \beta \) cells may limit insulin production (Barker 1994). Recent data suggest that maternal undernutrition may increase the fetal production of untoward cytokines including interferon (IFN-\( \gamma \)) and tumor necrosis factor (TNF-\( \alpha \)) (Hanson et al. 1996, Heyborne et al. 1992). Insulin-like growth factor I (IGF-I) plays a central role in regulating fetal growth, and fetal IGF-I can be modulated by maternal dietary intake (Gluckman 1993). Postnatally, IGF-I concentrations remain low in infants who have been growth-retarded in utero (Theriot-Prevost et al. 1988).

In conclusion, the public health implications are that the differential rates of growth in these LBW infants are associated with socioeconomic status, and that marked differentials exist even within a population that is already labeled “poor.” Five other determinants of maximal WAZ and LAZ gains were maternal weight and infant length (WAZ alone), maternal height and maternal smoking (LAZ alone) and neonatal illness (both WAZ and LAZ). These variables explained an additional 15% of the variance in WAZ gain and 11% of the LAZ gain. This analysis serves to reinforce the need to break the vicious cycle of intergenerational disadvantage through prenatal intervention programs in the short-term and poverty alleviation in the long-term.

LITERATURE CITED


The doi is https://doi.org/10.1007/s11830-019-02147-6


