Stature and birth rank

A study of schoolchildren in St Helena

C D MOYES

Department of Child Health, Cardiff

SUMMARY More than 1200 St Helenian schoolchildren were studied in 1973 and 1978 to determine how social factors affected growth. The association of short stature with large family size was confirmed and shown to be concentrated in the earlier members of the sibship. This was independent of birth interval. Association between birth interval and stature was most pronounced for the spacing following the index child, less for the preceding interval, and least for the family average interval. These results are similar to those of British children, for whom St Helenians form a reasonable model. It is argued that these results are not readily explained by genetic factors nor are they explained by differences of birthweight, but they are compatible with deprivation of parental care and love, for which older children have to compete with other siblings. Deprivation sufficient to retard growth is not confined to extreme circumstances or to broad social groups, but occurs selectively within the heart of the normal family.

Although the stature of an individual is determined by both genetic and environmental factors, the study of large numbers of children allows the separation of major influences on postnatal growth and recognition of areas of social vulnerability.1

A study of St Helenian children in 1973 showed a pronounced relationship between family size and short stature which was not an effect of overcrowding.2 A further survey was therefore designed to elucidate this correlation.

Background

St Helena is a small island in the mid-Atlantic Ocean, with a healthy climate. The population numbers about 5500 and is entirely of mixed race (predominantly Caucasian and West African with a small Chinese element). Nutrition is of Western type with overloading of carbohydrates and no frank malnutrition. There are no tropical diseases and medical care is free and modelled on the British pattern. Adult morbidity results largely from atherosclerosis, diabetes, obesity, and cancer. Infant mortality is 9/1000 and death between 1 and 15 years 3/1000.

The culture is entirely British and is little affected by racial variance or class, which is almost entirely manual. Families are larger than in Britain and there is considerable support in child rearing from the maternal aunts and grandmother, who may even foster children. Older siblings are also important in the rearing of large families. Modern contraception is very recent and does not significantly affect this study. Illegitimacy is common and accepted.

St Helena provides a fair model for the lower social class patterns of Western countries except for the greater family support of the mother.

Method

Each child aged between 5 and 15 years was measured in stocking feet by one person, and received a general medical examination and racial score (separately for black and oriental features) by a second observer. All children below the 3rd centile (British standards of Tanner et al.3) were investigated with a blood count, urea, urine analysis (and wrist and chest x-rays if appropriate), and medical records since birth were examined. Social information was supplied by parents and teachers.

All but 2 of the 1295 children were seen, and initial exclusions were 32 British expatriates, 14 with uncertain data, 9 residents of a children's home, and 5 with serious handicaps. 18% of the children below the 3rd centile were considered to have possible organic causes of short stature and were excluded from further analysis.

The unmodified growth charts were not entirely suitable, as 7·4% of children were below the 3rd
centile and there was an increasing deviation from the British means with age, which was similar for both sexes (Fig. 1). The data were therefore modified to correct for this age bias.

Results

The increased prevalence of short stature in large families was confirmed (Fig. 2) and is significant for both the 10th centile ($\chi^2=10.9, n=3, P<0.05$) and the 3rd centile ($\chi^2=14.6, n=3, P<0.01$).

Race. Racial scoring showed a normal distribution curve from the white children to the black. Oriental ancestry was rarely more than 1 in 8 and did not correlate with short stature. Analysis did not support any effect of racial background on the other findings.

Social class. The occupation of the fathers of the children was almost entirely manual and was unrelated to short stature except that fewer of the 34 children from ‘white collar’ classes were short, but this was not statistically significant. The effect on other results was negligible.

Illegitimacy. The absence of a putative father from the home provided a rough measure of illegitimacy, and was only slightly correlated with short stature.

Fostering. 87 (7.2%) children did not live with their natural mothers, but did not show any difference in stature from those who did. This reflects the stable and affectionate care of the mother-substitute, generally the aunt or grandmother.

Birth rank. Examination of birth rank by each family size showed that the high proportion of short children in the larger families was concentrated among the earlier born (Fig. 3), the later members being of similar size to those from small families, who showed no consistent trends with birth order. In families with 6 or more children this trend was
statistically significant ($\chi^2=14.2$, $n=5$, $P<0.02$), was similar for both sexes, and existed independently of the immediately subsequent birth interval.

**Birth interval.** In small families there was no clear relationship between growth and family spacing, but families with 6 or more children did show a greater proportion of short stature in those whose birth was followed by another sibling within a short period (Fig. 4). This was not quite statistically significant, however ($\chi^2=3.58$, $n=2$, $P<0.1>0.05$). There was a smaller relationship with the preceding birth interval and less again for the overall family average.

**Longitudinal data.** Children aged between 10 and 15 years had been measured 5 years earlier. Children from larger families were smaller on both occasions but there was no difference in growth velocity from those of small families during school years, nor did the birth of further siblings affect the velocity during this period.

**Discussion**

Apart from the absence of significant social class differences and the effects of closer family support, these results are strikingly similar to those obtained in Britain.\(^1\)\(^4\)-\(^5\)

Grant\(^6\) also found birth rank to be a major correlate with short stature, finding that the later born child of any consecutive pair within a family tended to be taller than the immediately preceding one, and suggested that the advent of each additional child to a family adds as a check to the growth of all preceding sibs. The National Child Development Study\(^7\) found that, at any given parity, those children with no younger siblings were 1.1 cm taller than those with two or more, and concluded that the presence of younger siblings retarded the growth of their elders.

Grant\(^8\) also found a short birth interval after the birth of a child was associated with short stature, but (as in our study) this was not statistically significant. There are a number of possible explanations for these relationships of short stature and family structure.

**Genetic.** Many studies have shown that short parents tend to have short children. However, this correlation is just as great in societies where there is undoubted malnutrition, as shown by Martorell et al.\(^7\) who argued that ‘socioeconomic and nutritional status is correlated across generations’. Lacey and Parkin\(^8\) considered the continuity of social disadvantage to be a major cause of this association in Newcastle upon Tyne. The association with birth rank cannot be explained by a genetic hypothesis, particularly if individual families are studied by sibling pairs.\(^9\)

**Birthweight.** First-born children have lighter mean
birthweights than subsequent siblings, but after the second child further increases are slight. The number of first-born St Helenian children from large families included in this study is too small to affect the results. The National Child Development Study found that analysis of other factors affecting the height of British children at 7 years, including parity and number of younger siblings, was very similar whether or not gestation and birthweight were allowed.

**Nutrition.** There was no evidence of frank deficiency states among St Helenian children. A questionnaire of older children showed that there were very few families who ate fish or meat less than once a week. It would not be surprising however, if the quantity of food offered was less and the quality poorer in larger families. However, it is difficult to see why later born children should receive better diets when the capital income per head of the family is least—except in those cases where the eldest are earning (a small minority in St Helena, where the number of school leavers who are unemployed is very high). Jacoby et al. investigated the diets of British children and found no significant differences according to birth rank or even family income. Grant also concluded that the relationship of growth to birth rank was unlikely to be a direct result of food shortage. Davis et al. noted a close link between short stature and feeding difficulties, and achieved an increase in height velocity in some children after advice had been given to parents on the manner of offering food. This suggests that in these children short stature was indeed a result of undereating, and not due to a shortage of offered food.

**Recurrent illness.** It is possible that large families are more exposed to infectious diseases, although Tanner did not consider recurrent minor infections to be themselves a cause of longstanding growth retardation, and the younger children of large families should be more exposed to these than their elder siblings.

**Parental care.** It has been known for many years that children from emotionally unhappy deprived homes grow less well. Widdowson showed that children in an orphanage unexpectedly did not benefit from a dietary supplement and this was apparently due to their unhappiness under a harsh matron. Lacey and Parkin considered that some deprivation was present in the lives of at least 30% of Newcastle children less than the 3rd centile in height, although they could only demonstrate a clear deficiency of growth hormone in severe cases. The suggestion that deprivation may cause poor growth in 'normal' families is implied by Grant who stated 'There is a slight suggestion that the last born is in the most favourable position for growth, and the degree to which the growth of older sibs is depressed may, to some extent, turn on the length of time for which they remain in this favoured position'. This hypothesis also fits well with the pronounced relationship between short stature and the number of younger siblings found by the National Child Development Study.

The relationship of growth to the birth interval can also be understood in these terms. The mean family birth interval in large families shows some association with stature, but the spacing immediately preceding the index child is more convincing, and that immediately following even more so (Fig. 3). A younger sibling is a more successful competitor for parental love and attention than his older brother or sister, who may therefore suffer relative deprivation. Later children may have benefited in their earlier years from the absence of competition from younger siblings as well as from substitute mothering by the older ones. There was no evidence in this study that further inhibition of growth occurred during school years, suggesting that it is in the young child that relative deprivation of parental care and love may be reflected in suboptimal growth. Thus, even if short stature is a result of undereating, the primary problem lies in the family: and it is there we should focus our concern.

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**References**

The following articles will appear in future issues of this journal:


Autosomal hypophosphataemic bone disease responds to 1,25-(OH)\textsubscript{2}D\textsubscript{3}. C R Scriver, T Reade, F Halal, T Costa, and D E C Cole

Sickle cell disease in Saudi Arabs. R P Perrine, P John, M Pembrey, and S Perrine

Prader-Willi syndrome after age 15 years. B M Laurance, A Brito, and J Wilkinson

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C D Moyes

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