PHYSICAL DEVELOPMENT AT 7 YEARS OF AGE IN RELATION TO VELOCITY OF WEIGHT GAIN IN INFANCY WITH SPECIAL REFERENCE TO INCIDENCE OF OVERWEIGHT*

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As a result of a carefully controlled study on obese children, Börjeson (1962) concluded that obesity in childhood is not a problem of physical health, and that although it may be a major psychological problem for some children, in most cases there is no evidence of emotional conflict caused by obesity itself. Nevertheless, most physicians consider obesity in children to be a major health problem calling for preventive and therapeutic action. Besides a widespread moral attitude towards overconsumption of food as the suspected cause of overweight, this concern gains support from the claim that obesity in adults implies a considerably increased risk of secondary disease and premature death (Kannel et al., 1967) and the fact that very often adult obesity starts in childhood (Mullins, 1958). In the view of physicians responsible for adult care, treatment of obesity should therefore start in childhood. Paediatricians, however, have all experienced extreme difficulties in endeavours to achieve long-term weight reduction in obese children (Lloyd, Wolff, and Whelen, 1961; Spranger and Dörken, 1967; Lodi, 1970). Therefore the possibility of early preventive action, preferably in infancy, has to be considered seriously. This preventive attitude has been supported by the findings of Knittle and Hirsch (1968) that the number of fat cells in adipose tissue is significantly increased in rats overfed during the first 10 weeks of life. It is not yet known whether human infants behave in the same way as rats in this respect. It has been found in the human species as well as in the rat, however, that the total number of fat cells in adult life cannot be reduced (Björntorp and Sjöström, 1971). In a study on obese children and adults published recently, Brook, Lloyd, and Wolff (1972) showed that the early onset of obesity is associated with an increased number of fat cells.

In a longitudinal study in Sheffield, England, Eid (1970) found a significantly increased incidence of overweight and obesity in those children who had gained weight rapidly during the first six months of life, as compared with normally growing infants. However, these results may not be representative for a 'normal' population, since it was shown later by Taitz (1971) that no less than 60% of artificially fed infants from the same region gained weight above the 90th centile of generally accepted British standards.

In Sweden the physical life conditions have for a long time been fairly similar for the great majority of infants; the frequency of breast-feeding is generally low, and artificial nutrition has been highly standardized during the last decades. The market for baby food is completely dominated by two manufacturers, whose products are similar and in accordance with the recommendations of leading paediatricians. The information about infant nutrition provided by child welfare centres and private paediatricians is uniform throughout the country, and the attendance rate at the well baby clinics approaches 100%. In case of deviations from normal weight development, substantial efforts are made to correct errors in nutrition which may have occurred. The present investigation was started with the aim of finding out how many of the obese children in a representative population of school children started as 'rapidly weight gaining' infants, and how many of such infants in a representative infant population will eventually be obese, if infant nutrition is fairly well controlled by a uniform programme.

MATERIAL

The material consisted of all first grade pupils, 7 years of age, in the elementary schools in the city of Uppsala in the autumn of 1970 for whom

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there was documented evidence of good health supervision during their first year of life. Such evidence was accepted as positive if the record from the well baby centre(s) was available and contained weight data for at least every fourth month during the first year and at least one weight between the age of 10 and 18 months. In total there were 1,279 7-year-olds starting school in the autumn of 1970 (621 boys and 658 girls). For 156 boys and 151 girls the health records were either not available or incomplete, mainly because the children were born in other parts of the country or had spent a great part of their first year in day nurseries. The final material thus comprised 465 boys and 507 girls (total 972) representing a general population of 7-year-olds in a Swedish urban community with at that time approximately 100,000 inhabitants. Because of the dominating role of the University and other schools in the life of the town, the general educational level of the population is higher than the mean for Sweden as a whole. It should be noted that no children were excluded because of previous or recent disease, except those with such a severe handicap that it was impossible for them to attend a class in elementary school.

In Uppsala practically 100% of all deliveries take place in the Department of Obstetrics and Gynaecology of the University Hospital. A notice of each newborn infant (giving, among other information, the birth weight and birth length) is sent to the nearest well baby centre within one week. The nurse at the centre pays a home visit when the baby is approximately 2 weeks old; thereafter the infant is taken to the centre for routine examinations at 6 weeks, 3 months, 4½ months, 6 months, 8 months, 10 months, and 12-15 months. On each of these occasions, and sometimes also at shorter intervals, the exact date, the weight, and the general state of the infant are noted on a standardized record. For most children the controls continue up to the age of 6 years. At 7 years, the health record is sent to the school health service. During the first two months in the first grade all children undergo a medical examination.

**Methods**

All well baby centres, as well as the newborn nursery of the hospital, use the same scales of the balance type (Stathmos baby scale). The birth length is measured with a scale engraved along the nursing table. For the 7-year-olds the same type of measuring equipment is used in all schools. The height is measured in the standing position against a firm wall and with a fixed scale. For weighing, scales of the balance type (Stathmos 304) are used throughout, the children wearing no clothes except underpants. With the data thus recorded, the following computer operations were performed on a CD 3600:

1. **Substitution of missing birth length data.** The birth weight was available for all children, but in 262 cases the birth length had not been recorded. For these cases, a substitute birth length was calculated from the formula:

   \[
   \text{Birth length (cm)} = 3 \cdot 627 \times \text{Birth weight (kg)} + 37 \cdot 549
   \]

   which was derived from the corresponding data of the 710 cases with recorded birth length.

2. **Calculation of height increase from birth to 7 years.** In order to obtain a comparable measure for height increase for all children, the height recorded at approximately 7 years (mean 86.5 months, standard deviation 3.3 months) was corrected to a probable height attained at exactly 84 months, assuming a uniform growth rate of 0.016 cm/day (Karlberg and Iggbom, 1959).

3. **Height for height.** As a standard the mean curve representing weight for height in the range 100-140 cm from the diagram of Karlberg and Iggbom (1959) was used and expressed in the following formula:

   \[
   \text{Standard weight for height} = 49 \cdot 025 - 0 \cdot 8552 \times H + 0 \cdot 00524 \times H^2 \text{ where } H = \text{measured height (see Fig. 1).}
   \]

   As can be seen in Fig. 1,
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the standards of Karlberg and Iggbom correspond perfectly well with the means in our population.

4. Pattern of weight gain during infancy. From the recorded weights and the corresponding dates the weights attained at each month from 1 to 12 were calculated by linear interpolation. In some cases the values for months 11 and 12 were obtained by extrapolation from earlier measurements. From the calculated weights attained the weight gains for each monthly interval as well as the weight gain from birth to 1, 2, ..., 12 months were computed by simple subtraction. As a measure of the deviation of the growth curve from linear growth, the variable Q was defined as the ratio between the maximum monthly growth velocity and the mean growth rate.

STATISTICAL METHODS

Standard methods were used for the computation of means, standard deviations, centiles, correlation coefficients, and t-values. All $\chi^2$ tests were performed with Yates's correction. For the procedure of multiple regression the standard program B34T (Thornber, 1966) was used. Computation of relative risk and population attributable risk percent are described later.

RESULTS

DISTRIBUTION OF PHYSICAL GROWTH DATA IN INVESTIGATED POPULATION

As will be shown later, rather striking differences between boys and girls were found in the relations between certain variables. All variables will therefore be presented separately for boys and girls.

BIRTH WEIGHT AND BIRTH LENGTH (Table I)
The means and standard deviations are in accordance with generally accepted Swedish standards, and the difference between the sexes is the usual one.

<table>
<thead>
<tr>
<th></th>
<th>Birth Weight</th>
<th>Birth Length</th>
</tr>
</thead>
<tbody>
<tr>
<td></td>
<td>N</td>
<td>Mean</td>
</tr>
<tr>
<td>Boys</td>
<td>465</td>
<td>5.01</td>
</tr>
<tr>
<td>Girls</td>
<td>507</td>
<td>3.370</td>
</tr>
</tbody>
</table>

$\frac{t}{t} = 3.3931 P < 0.001$  $\frac{t}{t} = 3.875 P < 0.001$

GROWTH DURING THE FIRST YEAR OF LIFE The growth curves for boys and girls correspond closely to those published by Karlberg and co-workers (1972). Since most of the subsequent analysis is based on growth rates rather than weight attained, the data are presented in Fig. 2 as velocity of weight gain as a function of age (third, 50th, and 97th centiles). As can be seen, boys cover a considerably wider range than girls, a greater number of boys

![Fig. 2. Velocity of weight gain 3rd, 50th, and 97th centiles.](http://jech.bmj.com/)

![Fig. 3. Distribution of height increase 0-7 years.](http://jech.bmj.com/)
gaining weight rapidly. This is true especially during the first six months. This finding is in accordance with similar observations by others (Eid, 1970; Fomon et al., 1971). It is also interesting to note that at the lower edge of the distribution—around the third centile—there is no sex difference, as many boys as girls being slow weight gainers.

**HEIGHT INCREASE FROM BIRTH TO 7 YEARS** The distribution of this variable is shown in Figure 3. The small difference between the sexes is not statistically significant.

**WEIGHT FOR HEIGHT AT 7 YEARS** For each child the standard weight corresponding to his/her individual height was calculated and weight for height was expressed as the percentage deviation of the measured weight from this standard weight. The mean values for boys and girls are very close to each other, but there is a striking difference in the distribution, girls predominating at both ends of the distribution curve (Fig. 4). Thus, 153 girls but only 105 boys were either underweight (below —10%) or overweight (above +10%). This difference is statistically significant ($P < 0.01$).

**PREVALENCE OF OVERWEIGHT AND OBESITY** All those children are classified as overweight whose measured weight is more than 10% but not more than 20% above their standard weight for height. All children with a weight exceeding their standard by more than 20% are classified as obese. From Table II it can be seen that the prevalence of both overweight and obesity is higher in girls but not significantly so.

**ANALYSIS OF RELATION BETWEEN GROWTH VELOCITY IN INFANCY AND (a) HEIGHT INCREASE FROM BIRTH TO 7 YEARS AND (b) WEIGHT FOR HEIGHT AT 7 YEARS**

In Table III the variables used in the subsequent correlation and regression analyses are shown.

### TABLE II
**PREVALENCE OF UNDERWEIGHT, OVERWEIGHT, AND OBESITY IN 7-YEAR-OLDS**

<table>
<thead>
<tr>
<th></th>
<th>Underweight</th>
<th>Overweight</th>
<th>Obese</th>
<th>Overweight + Obese</th>
<th>All Extreme</th>
</tr>
</thead>
<tbody>
<tr>
<td></td>
<td>W &lt;90% SW</td>
<td>W &gt;110% to 120% SW</td>
<td>W &gt; 120% SW</td>
<td>W &gt; 110% SW</td>
<td>Underweight + Overweight + Obese</td>
</tr>
<tr>
<td><strong>N %</strong></td>
<td>N %</td>
<td>N %</td>
<td>N %</td>
<td>N %</td>
<td>N %</td>
</tr>
<tr>
<td><strong>Boys</strong></td>
<td>45 9.7</td>
<td>48 10.3</td>
<td>12 2.6</td>
<td>60 12.9</td>
<td>105 22.6</td>
</tr>
<tr>
<td><strong>Girls</strong></td>
<td>67 13.2</td>
<td>65 12.8</td>
<td>21 4.1</td>
<td>86 17.0</td>
<td>153 30.2</td>
</tr>
<tr>
<td><strong>$\chi^2$</strong></td>
<td>2.64 ns</td>
<td>1.24 ns</td>
<td>1.36 ns</td>
<td>3.13 ns</td>
<td>6.80 P &lt; 0.01</td>
</tr>
</tbody>
</table>

$W =$ measured weight; $SW =$ standard weight for height; $ns =$ not significant.
Correlation Analysis

Correlation coefficients between the two dependent variables and all the independent variables were in each instance found to demonstrate weak relationships. There does not seem to be any crucial period in the first year of life which is more important than another for the prediction of further physical development (Table IV). The results of a multiple regression analysis with weight for height and height increase from 0 to 7 years as dependent variables are summarized in Tables V and VI.

**Prediction of Height Increase** (Table V)
As expected from the correlation analysis, the variable weight gain in 0-12 months proved to be the most important in all instances. There seems to be no difference between boys and girls, and the explained fraction of this variable amounts to only 23% in both sexes. Thus, it may be stated that in the investigated population physical development during the first 7 years was affected to only a minor degree by factors detectable by weight measurements in infancy.

**Prediction of Weight for Height at 7 Years** (Table VI)
With this variable, also, weight gain from birth to 12 months proved to have the greatest explanatory power. For both sexes, birth weight occupies the second place. In boys, but not in girls, $r^2$ is further increased by a number of other variables describing the pattern of growth during infancy, which, for boys, appears to be able to explain almost twice as much of the total variation in weight for height at 7 years as for girls. This marked difference is probably a reflection of the finding presented earlier that more boys tend to grow rapidly during the first months, but that more
girls tend to be overweight at 7 years, and it points to other factors than infant nutrition underlying the observed growth rates and correlations. The most important result of this analysis, however, is the fact that as little of the variation as 9% for girls and 18% for boys can be explained by physical data from the first year. Other factors seem to be of much greater importance.

**Evaluation of Risks of Overweight and Obesity**

The preceding regression analysis was based on the total material, which of course is dominated by normally developed children. The finding that factors affecting early growth seem to be of only minor importance for further development in a total population of children does not *a priori* exclude the possibility that other relations may exist for the more extreme cases. In the multiple regression analysis, linear relationship between variables was assumed throughout, an assumption which may be incorrect. An attempt was therefore made in the following analysis to identify a group or groups of infants with a substantially increased risk of later overweight or obesity. If a therapeutic or preventive programme is to be applied to such a risk group, the defined population should fulfil the following two requirements:

(a) *high relative risk*, i.e., for the individuals included in the risk group the risk of becoming overweight should be substantially higher than that of individuals not considered to be at risk;

(b) *high 'population attributable risk percent'* (Cole and MacMahon, 1971), which is a measure of the proportion of the total amount of disease in a population which is attributable to the investigated risk factor and thus theoretically preventable by elimination of this factor, provided the risk factor is causally related to the disease. According to Cole and MacMahon, the two concepts are defined as follows:

\[
(a) \quad R = \frac{R_e}{R_o}
\]

\[
(b) \quad A_P\% = \frac{P_e (R - 1)}{1 + P_e (R - 1)}
\]

\[
A_P\% = \text{population attributable risk percent}
\]

\[
P_e = \text{proportion of the total population which is exposed to the risk factor}
\]

In the multiple regression analysis, birth weight and weight gain from birth to 12 months were shown to be the most important predictors for relative weight at 7 years. For a preventive program directed at infants, birth weight is of no interest; therefore, the risk groups were defined solely on the basis of weight gain during the first year.

**Preventive Model 1** In the first attempt, weight gain during the entire first year was taken as a single criterion. After an analysis of the risk of developing overweight in later years as a function of weight gain in infancy, a total weight gain of 7·5 kg or more was chosen arbitrarily as a suitable limit for the definition of the risk group. This limit corresponds roughly to the 90th centile for girls and the 70th centile for boys. The expected relative risk and population attributable risk percent emanating from such a definition of the risk group are shown in Table VII.

**Preventive Model 2** A preventive program using total weight gain during the entire first year as a criterion for identification of individuals at risk would probably come too late for active intervention. A more suitable program from a practical point of view would presumably be a policy of intervention at any time an infant shows a tendency to excessive weight gain. In order to test the possible yield of such a program, all infants were identified whose weight gain during any three-month period (0-3, 1-4, to 9-12) exceeded the 97th centile for that period. Separate centiles for boys and girls were computed. The results are included in Table VII. For girls, the values for both groups are lower than in model 1, whereas for boys there appears a puzzling discrepancy between the values for obesity, on the one hand, and those for all degrees of overweight, on the other hand, the theoretical possibilities for prevention of simple overweight being practically zero on the basis of this model, whereas 50% of the obese cases could be prevented if a causal relationship exists.

**Preventive Model 3** In the search for still more specific criteria for a reasonable risk group, weight gain for months 1 to 4 and for months 9 to 12 was
**VELOCITY OF WEIGHT GAIN IN INFANCY**

**TABLE VII**

**COMPARISON OF THREE DIFFERENT PREVENTIVE MODELS**

Risk groups defined according to three different criteria (see text). Expected yield of preventive program evaluated by assessment of relative risk and population attributable risk percent \( (\text{AP}%) \)

1. Test of significance of difference between relative risk and 1·0
2. Test of significance of difference between boys and girls

(Standardized Difference between Proportions (Cochran, 1954; Sheche, 1966))

<table>
<thead>
<tr>
<th>Model</th>
<th>Degree of Overweight</th>
<th>Sex</th>
<th>Relative Risk</th>
<th>( \text{AP} )%</th>
<th>( \chi^2 ) (1)</th>
<th>( \chi^2 ) (2)</th>
</tr>
</thead>
<tbody>
<tr>
<td>1</td>
<td>&gt;10%</td>
<td>Boys</td>
<td>2·8</td>
<td>10</td>
<td>18·35 ***</td>
<td>27·50 ***</td>
</tr>
<tr>
<td></td>
<td></td>
<td>Girls</td>
<td>1·9</td>
<td>36</td>
<td>6·65 **</td>
<td>3·25 ns</td>
</tr>
<tr>
<td></td>
<td>&gt;20%</td>
<td>Boys</td>
<td>2·3</td>
<td>8</td>
<td>1·44 ns</td>
<td></td>
</tr>
<tr>
<td></td>
<td></td>
<td>Girls</td>
<td>1·7</td>
<td>29</td>
<td>0·44 ns</td>
<td></td>
</tr>
<tr>
<td>2</td>
<td>&gt;10%</td>
<td>Boys</td>
<td>1·3</td>
<td>0</td>
<td>0·53 ns</td>
<td>0·41 ns</td>
</tr>
<tr>
<td></td>
<td></td>
<td>Girls</td>
<td>0·0</td>
<td>5</td>
<td>0·02 ns</td>
<td></td>
</tr>
<tr>
<td></td>
<td>&gt;20%</td>
<td>Boys</td>
<td>7·4</td>
<td>51</td>
<td>13·43 **</td>
<td>8·55 **</td>
</tr>
<tr>
<td></td>
<td></td>
<td>Girls</td>
<td>1·3</td>
<td>5</td>
<td>0·05 ns</td>
<td></td>
</tr>
<tr>
<td>3</td>
<td>&gt;10%</td>
<td>Boys</td>
<td>3·5</td>
<td>27</td>
<td>26·66 ***</td>
<td>25·54 ***</td>
</tr>
<tr>
<td></td>
<td></td>
<td>Girls</td>
<td>1·6</td>
<td>8</td>
<td>3·37 ns</td>
<td></td>
</tr>
<tr>
<td></td>
<td>&gt;20%</td>
<td>Boys</td>
<td>16·9</td>
<td>70</td>
<td>29·89 ***</td>
<td>15·95 **</td>
</tr>
<tr>
<td></td>
<td></td>
<td>Girls</td>
<td>1·3</td>
<td>5</td>
<td>0·05 ns</td>
<td></td>
</tr>
</tbody>
</table>

*Levels of significance:*

- **P** < 0·05
- ***P** < 0·01
- ****P** < 0·001
- **ns** not significant

**TABLE VIII**

**2 × 2 CONTINGENCY TABLES FOR COMPUTATION OF RELATIVE RISK AND POPULATION ATTRIBUTABLE RISK PERCENT**

(Table VII)

<table>
<thead>
<tr>
<th>Model</th>
<th>Sex</th>
<th>Weight Gain in Infancy</th>
<th>Overweight &gt; 10%</th>
<th>Normal</th>
<th>Overweight &gt; 20%</th>
<th>Normal</th>
</tr>
</thead>
<tbody>
<tr>
<td>1</td>
<td>Boys</td>
<td>Rapid</td>
<td>33 107</td>
<td>6 134</td>
<td>6 319</td>
<td></td>
</tr>
<tr>
<td></td>
<td></td>
<td>Normal</td>
<td>27 298</td>
<td>6 134</td>
<td></td>
<td></td>
</tr>
<tr>
<td></td>
<td>Girls</td>
<td>Rapid</td>
<td>18 43</td>
<td>4 57</td>
<td></td>
<td></td>
</tr>
<tr>
<td></td>
<td></td>
<td>Normal</td>
<td>68 378</td>
<td>17 429</td>
<td></td>
<td></td>
</tr>
<tr>
<td>2</td>
<td>Boys</td>
<td>Rapid</td>
<td>12 62</td>
<td>7 67</td>
<td>5 386</td>
<td></td>
</tr>
<tr>
<td></td>
<td></td>
<td>Normal</td>
<td>48 343</td>
<td>5 386</td>
<td></td>
<td></td>
</tr>
<tr>
<td></td>
<td>Girls</td>
<td>Rapid</td>
<td>13 63</td>
<td>4 72</td>
<td></td>
<td></td>
</tr>
<tr>
<td></td>
<td></td>
<td>Normal</td>
<td>73 358</td>
<td>17 414</td>
<td></td>
<td></td>
</tr>
<tr>
<td>3</td>
<td>Boys</td>
<td>Rapid</td>
<td>23 47</td>
<td>9 61</td>
<td>3 392</td>
<td></td>
</tr>
<tr>
<td></td>
<td></td>
<td>Normal</td>
<td>37 358</td>
<td>3 392</td>
<td></td>
<td></td>
</tr>
<tr>
<td></td>
<td>Girls</td>
<td>Rapid</td>
<td>19 57</td>
<td>4 72</td>
<td></td>
<td></td>
</tr>
<tr>
<td></td>
<td></td>
<td>Normal</td>
<td>67 364</td>
<td>17 414</td>
<td></td>
<td></td>
</tr>
</tbody>
</table>
Obese boys (mean, N=12) —
Obese girls (" N=21) —
All boys (" N=465) —
All girls (" N=507) —

**FIG. 5.** Velocity of weight gain during infancy in children who were obese at 7 years, mean values.

Relative risk and population attributable risk percent as a function of sample size are shown in Figure 6. The degree of exposure to the risk factor, the velocity of weight gain, is inversely proportional to the sample size, the smallest sample containing the most rapidly growing infants. If a true dose-response relationship exists between risk factor and outcome, the relative risk should steadily decrease as the sample size increases.

As can be seen in Fig. 6, there is no such relationship; the opposite trend for obese boys is in all probability due to the small number of obese boys included in this study. In girls, model 3 would not serve as a reasonable basis for a preventive program, since both the relative risk and population attributable risk percent were very low in all instances. In boys, however, the value of the latter criterion increased steadily up to a sample size of 15%, and therefore this size was chosen for comparison of model 3 with models 1 and 2 in Table VII. The limits of weight gain during the two critical periods corresponding to different sample sizes are shown in Table IX.

Comparing the three models presented in Table VII, it can be concluded that for girls both criteria...
assume low values in all instances, and the relative risk is significantly increased above the random value only for model 1. Even in this case, however, only 10% of all overweight cases would be preventable. For boys, model 3 would promise the most favourable results, at least as far as obesity alone is concerned. If a causal relationship exists, inhibition of rapid weight gain in boys during the first four and the last four months of the first year would prevent 70% of the cases of obesity at 7 years of age.

**DISCUSSION**

The principal aim of the present study was to find an answer to the question whether obesity in school children is preventable by better control of infant nutrition. The results do not give a definite answer but they allow a discussion from a number of partially new aspects.

1. The children included in this investigation represent a population which in infancy was fed according to reasonable principles and which was supervised fairly closely by the child welfare centres. In this respect they seem to differ rather favourably from the population in the Sheffield region described by Taitz (1971), and probably also from the population from which Eid (1970) obtained his results. It should be noted that the growth pattern in infancy in our population corresponds fairly closely to that described for British children (Tanner, Whitehouse, and Takaishi, 1966), whereas the infants in the Sheffield region apparently have much higher growth rates, at least during the first months (Taitz, 1971). If the hypothesis of infant overnutrition as an important primer of childhood obesity is correct, one would expect a lower prevalence of obesity and overweight in our population compared with that studied by Eid. Unfortunately, the prevalence of overweight among 7-year-olds in Eid’s material is not known, but from the incidence in the different groups studied and with the assumption that between 10% and 50% of all children belong to the rapid weight gaining group, the prevalence of overweight (10-20%) among 6 to 8-year-olds in the Sheffield region can be estimated to be somewhere between 5% and 8% (our material: 11·6%) and that of obesity (> 20%) between 3% and 6% (our material: 3·4%). The only possible conclusion from this comparison is that our program of infant nutrition has done nothing to reduce the overall prevalence of overweight at 7 years of age; possibly the degree of overweight may be somewhat less severe in our material, but even so the effect would be very little.

2. During the last four decades there has been a profound change in infant nutrition in Sweden. Whereas in the 1930s and 1940s the majority of infants were breast-fed during the first months (Broman, Dahlberg, and Lichtenstein, 1942), artificial feeding was predominant for the infants born in 1963. The Swedish standard curve by Karlberg and Iggbom (1959) used for comparison with our data in Fig. 1 is based on measurements (Broman et al., 1942) made more than 30 years ago. As is shown in Fig. 1, the change in nutrition has had no influence whatsoever on the mean weight for height in 7-year-olds, though the mean height has increased by approximately 2 cm.

3. The multiple regression analysis has shown that only 10-20% of the variation in weight for height can be explained by factors whose effect was detectable in infancy. We may therefore conclude that other factors seem to be much more important.

4. The relation between the growth pattern in infancy and physical properties at 7 years of age is stronger in boys than girls. The striking sex difference is unexplained; but it is interesting to note that in the study of Fomon et al. (1971) both calorie intake and weight gain were higher in males than in females.

5. The velocity of weight gain in infancy is a better predictor of total height increase up to the age of 7 years than of the weight/height relation at 7 years. Furthermore, there are no differences between the sexes in this respect. This finding indicates, at least, that the relations between the pattern of growth in infancy and the physical properties at 7 years of age are rather complex, and that it may be hazardous to draw conclusions about cause and effect simply on the basis of statistically significant correlations.

6. The generally held assumption that fat babies are overfed babies gained some support from the recent study of Taitz (1971). On the other hand, Rose

### TABLE IX

**Preventive Model 3: Limits of Weight Gain (kg/4mth) During First and Last 4-Month Periods of First Year, for Risk Groups of Different Size**

<table>
<thead>
<tr>
<th>Percent of Children included</th>
<th>Limit of Weight Gain</th>
<th></th>
<th></th>
</tr>
</thead>
<tbody>
<tr>
<td></td>
<td>Boys</td>
<td>Girls</td>
<td></td>
</tr>
<tr>
<td></td>
<td>0-4 mth 9-12 mth</td>
<td>0-4 mth 9-12 mth</td>
<td></td>
</tr>
<tr>
<td>2</td>
<td>4.95 2.86</td>
<td>4.39 2.67</td>
<td></td>
</tr>
<tr>
<td>5</td>
<td>4.50 2.56</td>
<td>4.08 2.48</td>
<td></td>
</tr>
<tr>
<td>10</td>
<td>4.23 2.40</td>
<td>3.91 2.21</td>
<td></td>
</tr>
<tr>
<td>15</td>
<td>4.15 2.27</td>
<td>3.75 2.08</td>
<td></td>
</tr>
<tr>
<td>20</td>
<td>4.05 2.20</td>
<td>3.67 1.98</td>
<td></td>
</tr>
</tbody>
</table>
TORE MELLBIN AND JEAN-CLAUDE VUille

and Mayer (1968) found that the level of physical activity is more strongly correlated to the amount of subcutaneous fat as estimated by skinfold measurements than calorie intake, and that 'the extremely thin infants moved more and ate more than the normal babies, while the extremely fat ones moved less and ate less' (Mayer, 1969). Lack of correlation between calorie intake and velocity of growth had also been reported by Wallgren (1945) and Rueda-Williamson and Rose (1962). The significance of these findings has recently been questioned by Fomon et al. (1971), who found a definitely positive correlation between calorie intake and weight gain.

7. A program designed to prevent obesity in school children by inhibiting infants from growing too fast would be of no use in girls, since at 7 years of age the weight of the majority of girls who gained weight rapidly during infancy will be normal for their height, and since the majority of obese 7-year-old girls gained weight normally during infancy.

In boys, such a preventive program could be more effective, provided that there really is a causal relationship between rapid weight gain in infancy and the occurrence of overweight in later years. In view of the marked sex difference and the absence of a demonstrable dose-response effect (model 3, p. 232), the assumption of a causal relationship appears rather dubious. The relative importance of genetic factors and of prenatal and postnatal influences for the determination of the rate of growth in infancy and the degree of overweight at 7 years of age cannot be evaluated quantitatively. Our results suggest, however, that these influences do not operate in the same way in boys and girls. The study does not support the generally held view that infant nutrition is the dominant factor.

8. The relationship between growth velocity during infancy and the weight/height configuration at 7 years of age is not simply linear. The study of different preventive models suggests that in boys continuous fast growth during the whole of the first year is related primarily to low degree of overweight at 7 years, whereas definitely obese 7-year-old boys rather present a history of short rapid growth spurts at different periods in infancy. This observation suggests the hypothesis that obesity does not represent just one extreme of a continuous normal distribution, but that obese individuals form a population of their own.

9. In recent years there has been a tendency to divide obesity into two categories, hyperplastic and hypertrophic. The former category is characterized by an increased number of fat cells, early onset, usually a more severe degree of overweight and resistance to therapy, but no metabolic anomalies. The second category is characterized by an increase in volume of the fat cells, later onset, and more profound metabolic changes (Berchtold et al., 1971). According to this classification, childhood obesity would generally seem to be of the hyperplastic type. The prognosis as to weight reduction would therefore be unfavourable, but in view of the absence of metabolic anomalies it might well be that the risk of maturity onset of diabetes and cardiovascular disease is in fact lower than is generally believed, and that the need for treatment and prevention of childhood obesity therefore is less urgent. In any case, this question needs to be penetrated more thoroughly if recommendations for the management of rapidly growing babies are to be based on knowledge of the real risks involved.

Summary

The relationship between growth velocity in infancy and further growth up to the age of 7 years (total height increase from birth, as well as weight for height at 7 years) was investigated in 972 children born in 1963 and starting school in the city of Uppsala in the autumn of 1970. By multiple regression analysis it was shown that in both sexes 23% of the variation in height increase could be explained by the growth pattern during infancy. In the case of weight for height, however, weight data from the first year proved to be a less efficient predictor, especially in girls, where only 9% of the total variation could be explained by these variables.

A study of three different theoretical preventive models showed that in girls it is not possible to define—on the basis of weight data from the first year—a group of infants with a significantly increased risk of developing obesity before the age of 7 years. In boys, however, such a risk group could be delineated with a very high population attributable risk percent (70%). A preventive program could be operative only if the relationship between risk factor and outcome is causal. In view of the marked sex difference and other observations, the present study does not support the hypothesis that infant overnutrition is an important cause of obesity in a population of Swedish urban children.

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Physical development at 7 years of age in relation to velocity of weight gain in infancy with special reference to incidence of overweight.

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