Background: Growth in children with Down’s syndrome (DS) differs markedly from that of normal children. The use of DS specific growth charts is important for diagnosis of associated diseases, such as coeliac disease and hypothyroidism, which may further impair growth.

Aims: To present Swedish DS specific growth charts.

Methods: The growth charts are based on a combination of longitudinal and cross sectional data from 4832 examinations of 354 individuals with DS (203 males, 151 females), born in 1970–97.

Results: Mean birth length was 48 cm in both sexes. Final height, 161.5 cm for males and 147.5 cm for females, was reached at relatively young ages, 16 and 15 years, respectively. Mean birth weight was 3.0 kg for boys and 2.9 kg for girls. A body mass index (BMI) >25 kg/m² of 18 years of age was observed in 31% of the males and 36% of the females. Head growth was impaired, resulting in a SDS of head circumference of −0.5 (Swedish standard) at birth decreasing to −2.0 at 4 years of age.

Conclusion: Despite growth retardation the difference in height between the sexes is the same as that found in healthy individuals. Even though puberty appears somewhat early, the charts show that DS individuals have a decreased pubertal growth rate. Our growth charts show that European boys with DS are taller than corresponding American boys, whereas European girls with DS, although being lighter, have similar height to corresponding American girls.

Materials and methods

The study is based on data from 4832 examinations of 354 children and young adolescents with DS, 57% males and 43% females. The children were born between 1970 and 1997. Data from 203 children (120 males, 83 females) with DS were collected from records on all individuals with DS of four different age groups.

**Table 1** Distribution of the number of children and the number of observations for the two groups of Swedish children with Down’s syndrome

<table>
<thead>
<tr>
<th>Group 1</th>
<th>Group 2</th>
<th>Total</th>
</tr>
</thead>
<tbody>
<tr>
<td>Males</td>
<td></td>
<td></td>
</tr>
<tr>
<td>No. of children</td>
<td>120</td>
<td>83</td>
</tr>
<tr>
<td>No. of observations</td>
<td>1363</td>
<td>540</td>
</tr>
<tr>
<td>Females</td>
<td></td>
<td></td>
</tr>
<tr>
<td>No. of children</td>
<td>85</td>
<td>68</td>
</tr>
<tr>
<td>No. of observations</td>
<td>956</td>
<td>571</td>
</tr>
</tbody>
</table>

Abbreviations: BMI, body mass index; DS, Down’s syndrome
from birth until 18 years of age, except those for head circumference, which cover the first four years of life.

The data for each sex were divided into 44 different age groups, one month intervals during the first two years of life, three months intervals during the third year of life, and one year intervals thereafter (table 2). Each child contributed only one single set of data for each age group. If data from more than one examination within an interval were available, the figures from the first examination were used.

The growth charts were compared with those presently used for children with DS, based on American children in studies by Cronk and colleagues (height and weight) and Palmer and colleagues (head circumference). A comparison was also made with the Swedish standard growth charts for healthy children according to Karlberg and colleagues, which correspond well to those of National Center for Health Statistics (NCHS).
Figure 2  Growth charts for height (mean (SDS)) of girls with Down’s syndrome from birth to 4 years of age (A) and 3 to 18 years of age (B).

Figure 3  Growth charts for weight (mean (SDS)) of boys with Down’s syndrome from birth to 4 years of age (A) and 3 to 18 years of age (B).
**Figure 4** Growth charts for weight (mean (SDS)) of girls with Down’s syndrome from birth to 4 years of age (A) and 3 to 18 years of age (B).

**Figure 5** Mean BMI of boys (A) and girls (B) with Down’s syndrome from birth to 18 years of age.
Data for weight and BMI were transformed into logarithms before the statistical analysis in order to obtain normal distributions. All growth charts are based on means and standard deviations using the weighted regression fitness system distributed by Jandel. The software used was Microsoft Excel 97 SR-1 (Microsoft Corporation, Redmond, WA, USA) and SigmaPlot, Scientific Graph System, version 3 for Windows (Jandel Scientific Software, San Rafael, CA, USA).

RESULTS
Figures 1 and 2 present growth charts for height for boys and girls. Mean birth lengths of both boys and girls with DS were 48 (2.3) cm (figs 1A and 2A), corresponding to $-1.5$ SD and $-1$ SD, respectively, on growth charts for healthy Swedish children. The mean final height of males with DS (fig 1B) was 161.5 (6.2) cm ($-2.5$ SD, Swedish standard) and that of females with DS (fig 2B) 147.5 (5.7) cm ($-2.5$ SD), resulting in a difference of 14 cm between the genders. The mean final heights, when plotted on the growth charts of American children with DS, were on the 95th and slightly above the 50th centile, respectively. Individuals with DS reached their final height at relatively young ages, 16 years for males and 15 years for females (fig 1B and 2B).

Figures 3 and 4 show the charts for weight. The boys had a mean birth weight of 3.0 (0.6) kg (fig 3A) corresponding to $-1.2$ SD. The mean weight at 18 years of age was 61 (8.3) kg (fig 3B) corresponding to $-0.4$ SD according to the Swedish standard and the 50th centile of American DS growth charts. Corresponding figures for females with DS were 2.9 (0.3) kg ($-1.5$ SD) and 54 (7.5) kg ($-0.5$ SD and 25th centile), respectively (fig 4A and B). A body mass index (BMI) above 25 kg/m$^2$ was observed in 31% of the boys and 36% of the females at 18 years of age (fig 5A and B).

DISCUSSION
Syndrome specific growth charts have been developed for several different disorders, for example, Down’s syndrome, Turner syndrome, Noonan syndrome, and Prader–Willi syndrome. These charts are important tools in the medical care of these children. Short stature is a cardinal sign of Down’s syndrome. Complicating disorders, such as coeliac disease, hypothyroidism, and growth hormone deficiency may aggravate the growth retardation. For detection of additional growth deviation the use of growth charts specific for children with DS are necessary. In this investigation we present growth charts from birth to 18 years of age for children with DS. The growth pattern is characterised by an impaired growth velocity from birth until adolescence, especially during the age interval of 6 months to 3 years and during puberty. In comparison with healthy boys, the males with DS had mean birth length and final height at 18 years of age corresponding to $-1.5$ SD and $-2.5$ SD, respectively. When the present data were compared to the American DS growth charts the final height corresponds to the 95th centile. The rather marked difference in final height between Swedish and American males with DS cannot be explained at present, but may be caused by factors such as ethnic diversity and differences in size of the study groups.
The girls with DS in the present study had a mean birth length of −1 SD and a mean final height, at the age of 18 years, of −2.5 SD according to the Swedish standard. The final height of the girls with DS was slightly greater than that of the American girls. Birth lengths for our children with DS could not be compared with those of the Americans, as the latter growth charts start at 1 month of age.

The individuals with DS reached their final height at relatively young ages, 16 years for males and 15 years for females. This is in agreement with earlier studies in which an early onset of puberty has been reported. Our results also show that individuals with DS have a reduced pubertal growth spurt, contributing to the low final height. In contrast to the American study our individuals with DS had the same difference in mean final height between the genders as healthy individuals.

Certain groups, in which mental retardation is predominant, such as the Prader–Willi and Bardet–Biedl syndromes, are predisposed to overweight. Despite having a greater mean final height than their American counterparts, the mean weight at 18 years among the Swedish males with DS was close to the 50th centile of the corresponding American males. The mean weight for Swedish girls with DS was at the 25th centile of the American growth charts at the age of 18 years. Even though one third of the individuals with DS were overweight (BMI > 25 kg/m²), as defined by the National Institute of Health (NIH), at the age of 18 years the weight and height data of the American individuals with DS indicate that overweight is a greater problem in the latter group.

Considering the mental retardation associated with DS the growth of the head is of great interest. Our results show that the mean head circumference of the children with DS was smaller than that of healthy Swedish children, but slightly greater than that of American children with DS. In agreement with previous studies there was a gender difference in head circumference, the male head tending to be larger than the female.

Although the optimal choice for the creation of growth charts would be a longitudinal, prospective study based on repeated examinations of a large and representative group, the drawbacks with respect to time constraints and logistics make it a less attractive model. Another way of collecting data is by multiple and detached examinations at separate ages, but given 354 children and 4823 examinations such an analysis would not result in reliable growth charts. In the present study we used both repeated data for each child, as in a longitudinal study, and several examinations of different children in the same age group, as in a cross sectional study. This is a common solution when growth in specific groups with relatively few subjects is analysed.

No children were excluded from the present study as a result of additional disorders. Thus, treated hypothyroidism and coeliac disease should not affect growth to any significant extent. Congenital heart defects may affect growth, but are great consumers of health care and are seen by many different physicians. Growth charts specific for children with DS are therefore important tools in the medical routine follow up as well as in the monitoring of growth promoting treatments.

ACKNOWLEDGEMENTS

This study was supported by grants from the Sävstaholm Society, the Swedish Medical Research Council (Grant No. K00-72X-09748-10A), the Gillberg Foundation, and the Carl Tesdorpf Foundation.

Authors’ affiliations

Å Myrelid, J Gustafsson, Department of Women’s and Children’s Health, Uppsala University, Uppsala, Sweden
B Ollars, G Annerén, Department of Genetics and Pathology, Uppsala University

REFERENCES

Epidemiology of birthweight

Babies with lower birthweights have higher risks of dying in infancy. Populations with lower mean birthweights usually have higher infant mortality rates. So is low birthweight, of itself, an adequate explanation of increased infant mortality? It has been argued that it is not (Allen J Wilcox. International Journal of Epidemiology. 2001; 30:1233–41).

If you plot neonatal mortality (y-axis, logarithmic) against birthweight (x-axis) you get a reversed J-curve with neonatal mortality falling from a very high level at very low birthweights to a minimum at about 3.5 kg (US data) and then increasing again at higher birthweights. (Optimal birthweight tends to be somewhat higher than mean birthweight.) Changing circumstances tend to change the level but not the shape of the curve. Thus, in the USA neonatal mortality fell for all birthweights between 1950 and 1988 so the 1998 curve lies below but parallel to the 1950 curve. (There is, incidentally, no change in the curve at 2.5 kg so the distinction between low birthweight and normal birthweight is arbitrary). Factors, such as maternal smoking or high altitude residence, which reduce birthweight in populations simply shift the reversed-J to the left. This produces the “low birthweight paradox” because low birthweight babies in the reduced-birthweight group then have lower mortality rates than babies of the same birthweight in the standard group. Maternal smoking then appears to be “beneficial” for lower birthweight babies. Wilcox solves the paradox by plotting neonatal mortality against birthweight z-scores for each group. It is then found that the neonatal mortality of babies of smoking mothers exceeds that of babies of non-smoking mothers at all points of the curve. Therefore, maternal smoking reduces birthweight at all levels but the effect on neonatal mortality is independent of birthweight. Wilcox argues that attention should be focussed on preterm births rather than on small preterm births. (The “residual” distribution is the lower tail lying outside the normal, bell-shaped, curve and is almost entirely due to small preterm births.)

Two commentators (Ibid: 1241–3 and 1243–4) accept that the low birthweight/normal birthweight dichotomy is outdated but challenge Wilcox’s conclusions, one because he believes that Wilcox takes too little heed of the social context and the other because she still believes that birthweight can be informative about population health.