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Critical Growth Phases for Adult Shortness

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Previous growth studies have not explored how different growth phases—the fetal, infancy, childhood, and puberty phases—interact with each other in the development of adult shortness. In this paper, the authors attempt to describe the importance of each growth phase for adult shortness and the effect of growth in one phase on other, subsequent phases. The authors analyzed data from a longitudinal population-based growth study of 2,850 healthy, full term Swedish children born between 1973 and 1975. The height values were transformed into a centimeter score (CMS) by subtracting the raw values from the reference mean values for a particular age and sex. Subnormal growth in any growth phase, as defined by a decrease of 3 CMS or more during a growth phase, was associated with significant increased risk for final heights below 0, –6, and –12 CMS. For children with subnormal growth during one, two, and three phases, the percentages for final height below –12 CMS (a standard deviation score of approximately –2) were 0.5%, 9.4%, and 75%, respectively. Most children (57/62) with a final height below –12 CMS had subnormal growth in two or three phases. Height gains during the four growth phases were interdependent. The infancy phase was negatively associated with fetal growth ($r = -0.33$, $p < 0.01$); the childhood phase was positively associated with infancy growth ($r = 0.21$, $p < 0.01$); and the puberty phase was negatively associated with childhood growth ($r = -0.10$, $p < 0.01$). *Am J Epidemiol* 2000;152:125–31.

body height; growth

During the past decade, evidence has accumulated regarding the long term consequences of growth in early life (1). There are four distinct human growth phases—fetal growth, infancy, childhood, and puberty (2). Adult shortness may result from subnormal growth during any one or more phases. Most investigators agree that growth during the fetal and infancy phases is critical for final height (3–10). Children born small for gestational age have a sevenfold increased risk for adult shortness (5). Growth stunting in early life also increases risk for adult short stature (11–13). The risk for adult shortness associated with subnormal growth during childhood and puberty is known to be important as well. One clear example is provided by the spontaneous growth that occurs in Turner syndrome, involving subnormal growth in childhood and the absence of the pubertal growth spurt (14). Precocious puberty also reduces final height significantly (15–17). Little is known, however, about the risk for adult shortness due to subnormal growth in more than one growth phase and the relative importance of each phase for final height. It is not clear whether the gain in height during one growth phase is positively or negatively correlated with the gain in other, subsequent growth phases.

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Abbreviations: CMS, centimeter score; SDS, standard deviation score.

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The aim of this study was to determine which growth phases are critical for adult shortness. The study also aimed to reveal the effect of growth in one phase on other, subsequent phases. For this purpose, we analyzed data from a large longitudinal growth study to evaluate the importance of each of the four distinctive phases.

MATERIALS AND METHODS

The data for this study came from a large longitudinal growth study of 3,650 healthy, full term children born between 1973 and 1975 in Sweden. Children without any growth-related disorders were defined as healthy; those with various growth-related disorders such as diabetes mellitus, endocrine disorders, intestinal disorders, heart disorders, and kidney disorders were excluded (5). An average of 14.4 height measurements were made for each child from birth to maturity. A growth chart was produced for each child, and any child who had gained less than 0.5 cm during the past year and who had reached the age at peak height velocity at least 2 years before the last examination was considered to have reached his or her final height. The final height value was treated as height at 18 years of age in the analysis, although for some children the last measurement was made later, at 19 or 20 years of age. Midparental height was computed as the average of mother's and father's height.

Height values at 0, 2, 8, and 18 years of age were included in the analysis. These points were taken to represent the four human growth phases, as defined by the infancy-childhood-puberty growth model (2). Fetal growth was gauged by

length at birth, while growth during the infancy, childhood, and puberty phases was represented by height and change in height between birth and 2 years of age, between 2 and 8 years of age, and between 8 and 18 years of age, respectively. Some children were measured at, for instance, 7.8 years of age and others at 8.2 years of age, so a linear interpolation was made to the specific age of interest (i.e., 8.0 years). Children without growth data available at 0, 2, 8, and 18 years of age ($n = 800$) were excluded from the present analysis. This left a final sample size of 2,850.

The statistical analyses were based on the centimeter unit rather than the commonly used standard deviation score (SDS) unit. The reason for this choice has been explained previously (9). The key issue in using centimeters rather than SDS is that the standard deviation of height increases from approximately 2 cm at birth to 6 cm in adulthood. Thus, the SDS unit will not have the same measured centimeter scale value at various pediatric ages. To pool data for children of the two sexes and of different ages, we expressed the height of an individual in centimeters from the reference mean, i.e., height centimeter score (CMS) (9). In principle, this standardized score is the same as the SDS but is obtained without dividing ($\text{height}_{\text{observed}} - \text{height}_{\text{reference}}$) by the $\text{standard deviation}_{\text{reference}}$. The mean reference values for height were derived from the same Swedish population (5).

We defined adult shortness in three ways: a final height below the mean value, a final height 6 cm below the mean, and a final height 12 cm below the mean. The corresponding values for the CMS units were 0, –6, and –12 CMS. The standard deviation in final height was close to 6.0 cm for both sexes; thus, –6 CMS was approximately 1 standard deviation below the reference mean values for final height, corresponding to the 16th percentile. Consequently, –12 CMS represents approximately –2 SDS, or the second to third percentile. The cutoff points for subnormal growth during the four growth phases were defined as a decrease of 3 CMS or more during any phase. Other cutoff points could have been chosen; we simply chose this value by dividing –12 CMS, which corresponds to the commonly used –2 SDS, by 4 (four phases).

Student's t test was used to test the difference in means between two groups. Both univariate analysis and multiple linear logistic regression analysis were applied to compute relative risks and/or odds ratios and their 95 percent confidence intervals. Pearson correlation analysis was used to analyze the relations between different growth phases. Only a two-tailed significance test was used, and a p value below 0.05 was regarded as statistically significant. All statistics were computed using SAS software (18).

RESULTS

Table 1 gives the mean parental heights and final heights (with standard deviations) for the children included in this study and the children excluded from the study. The mean values for father's height, mother's height, midparental height, and final height were similar for the two groups ($p >$ 0.30), except that final height for boys included in the study was 1 cm greater than that for boys excluded from the study. The results of the subsequent analyses were very similar for boys and girls; therefore, the results based on the pooled sample are presented.

Subnormal growth during any of the four growth phases was associated with significant increased risks for final heights 0, 6, and 12 cm below the reference means (table 2). For instance, children with subnormal growth during any one of the four growth phases had a 6- to 14-fold increased risk for a final height below –12 CMS. The percentages of children who had a final height below –12 CMS were similar for subnormal growth during the fetal, infancy, and childhood phases (9.1 percent, 9.2 percent, and 8.9 percent), and the percentage was 2 percent lower for subnormal growth during the puberty phase (6.9 percent). The computed relative risks were smaller than the odds ratios for final height below 0 and –6 CMS, but were approximately the same for a final height below –12 CMS.

Table 3 gives the frequencies of subnormal growth phases based on the three cutoff points for adult shortness, i.e., final heights below 0, –6, and –12 CMS. The proportions of children with subnormal growth in one, two, and three growth phases were 35.6 percent, 11.1 percent, and 1.3 percent, respectively. For children with subnormal growth during one, two, and three growth phases, the percentages of children with a final height below –12 CMS were 0.5 percent, 9.5 percent, and 75 percent, respectively. Most children (57/62) with a final height below –12 CMS had subnormal growth in two or three of the four growth phases.

Midparental height was positively correlated with the child's gain in height CMS during each of the four growth phases. Its correlation with change in height CMS during

TABLE 1. Mean parental heights and final heights of Swedish children included in and excluded from an analysis of growth phases and adult height, 1973–1993

Variable	Included (n = 2,850)			Excluded (n = 800)			p value†
	No.	Mean	SD*	No.	Mean	SD*	
Father's height (cm)	2,281	179.82	6.70	660	179.55	6.84	0.37
Mother's height (cm)	2,220	166.50	5.94	638	166.47	5.94	0.92
Midparental height (cm)	2,187	173.16	5.09	633	172.99	4.92	0.45
Final height of boys (cm)	1,438	180.61	6.66	416	179.69	6.25	0.01
Final height of girls (cm)	1,412	167.50	6.03	384	167.77	5.98	0.44

* SD, standard deviation.
† Student's t test for difference in means between the included and excluded groups; children without data available at 0, 2, 8, or 18 years of age were excluded.

TABLE 2. Odds ratios and relative risks for reaching a final height 0, 6, or 12 cm below the reference mean value (having a centimeter score (CMS) of <0, <–6, or <–12) in relation to subnormal growth* in the fetal, infancy, childhood, and puberty growth phases, Sweden, 1973–1993

Growth phase and height gain (CMS)	Total		Final height <0 CMS				Final height <–6 CMS				Final height <–12 CMS			
	No.	%	No.	%	OR† (95% CI†)	RR† (95% CI)	No.	%	OR (95% CI)	RR (95% CI)	No.	%	OR (95% CI)	RR (95% CI)
Fetal														
≤–3	275	9.6	233	84.7	6.1 (4.4, 8.6)	1.8 (1.7, 1.9)	110	40.0	4.2 (3.2, 5.5)	2.9 (2.4, 3.5)	25	9.1	6.9 (4.3, 10.8)	6.3 (3.9, 10.3)
>–3	2,575	90.4	1,226	47.6			351	13.6			37	1.4		
Infancy														
≤–3	382	13.4	294	77.0	3.7 (2.9, 4.8)	1.6 (1.5, 1.7)	145	38.0	4.2 (3.3, 5.2)	3.0 (2.5, 3.5)	35	9.2	9.1 (5.9, 14.0)	8.4 (5.1, 13.7)
>–3	2,468	86.6	1,165	47.2			316	12.8			27	1.1		
Childhood														
≤–3	514	18.0	455	88.5	10.2 (8.0, 13.1)	2.1 (1.9, 2.2)	228	44.4	7.2 (5.9, 8.9)	4.4 (3.8, 5.2)	46	8.9	14.3 (8.0, 25.4)	13.1 (7.5, 22.9)
>–3	2,336	82.0	1,004	43.0			233	10.0			16	0.7		
Puberty														
≤–3	582	20.4	477	82.0	5.9 (4.8, 7.3)	1.9 (1.8, 2.0)	212	36.4	4.6 (3.8, 5.7)	3.3 (2.8, 3.9)	40	6.9	7.5 (4.4, 12.8)	7.1 (4.2, 11.8)
>–3	2,268	79.6	982	43.3			249	11.0			22	1.0		

* Subnormal growth was defined as a height gain at least 3 cm less than the gain in the height reference mean during a growth phase (≤–3 CMS).
† OR, odds ratio; CI, confidence interval; RR, relative risk.

the childhood phase was the greatest ($r = 0.39$, $p < 0.01$) (table 4). Length CMS at birth was negatively correlated with gain in height CMS during the period 0–2 years of age ($r = –0.33$, $p < 0.01$), but it was positively correlated with change in height CMS during the childhood ($r = 0.21$, $p < 0.01$) and puberty ($r = 0.14$, $p < 0.01$) phases. Change in height CMS during infancy had a positive influence on the gain in height CMS during the childhood phase ($r = 0.28$, $p < 0.01$), but it was negatively correlated with change in height CMS during puberty ($r = –0.11$, $p < 0.01$). The change in height CMS during childhood was negatively correlated with the change in height CMS during puberty ($r = –0.17$, $p < 0.01$). Height CMS at 2 years of age was positively correlated with the gain in height CMS that followed during the period 2–8 years of age ($r = 0.42$, $p < 0.01$), while height CMS at 8 years of age was negatively correlated with the gain in height CMS that occurred during the period 8–18 years of age ($r = –0.10$, $p < 0.01$).

Logistic regression analyses showed that subnormal growth during any growth phase was associated with significant increased risk for final heights below 0, –6, and –12 cm from the reference means (table 5). Note that the odds ratios estimated from the logistic regression analyses were much larger than those estimated from univariate analyses (table 2) for subnormal growth during any one phase. The odds ratios were similar when 0 and –6 CMS were used as the cutoff points, but they increased remarkably when –12 CMS was used as the cutoff point. No significant collinearity between variables was observed in logistic regression analysis using SAS (PROC LOGISTIC) (18).

When midparental height was included in the logistic regression model, the estimated odds ratios were unchanged for subnormal growth during the fetal and infancy phases but became much smaller for subnormal growth during the childhood and puberty phases, especially for a final height below –12 CMS. The puberty growth phase had the largest odds ratio before adjustment for midparental height. However, after adjustment for midparental height, the odds ratio for the infancy growth phase was the greatest.

DISCUSSION

Adult shortness could be attributed to subnormal growth during any growth phase. It has been shown that both intrauterine growth retardation and growth stunting in early life are associated with an increased risk for adult shortness (3–10). This study shows that most adult shortness is a result of subnormal growth in multiple phases, rather than a single phase. Another important message is that the four growth phases are associated. Height gain in one growth phase may influence the magnitude of the gain in height in subsequent phases.

These data came from a large longitudinal population-based study, which allowed us to evaluate all growth phases from birth to maturity. The cutoff point for defining adult shortness used to be –2 SDS, or the third percentile (3–5). In this study, however, we defined both adult shortness and subnormal growth in centimeters and introduced the centimeter

TABLE 3. Distribution of subnormal growth phases in healthy full term Swedish children (*n* = 2,850), 1973–1993*

Pattern no.	Growth phase†				Total		Final height (CMS‡)						Midparental height (CMS)	
							<0		<–6		<–12			
	Fetal	Infancy	Childhood	Puberty	No.	%	No.	%	No.	%	No.	%	Mean	SD‡
Three phases subnormal					36	1.3	36	100.0	35	97.2	27	75.0		
1	S	S	S	N	9	0.3	9	100.0	8	88.9	7	77.8	–6.12	3.54
2	S	N	S	S	16	0.6	16	100.0	16	100.0	11	68.8	–5.07	3.91
3	N	S	S	S	11	0.4	11	100.0	11	100.0	9	81.8	–7.81	3.00
Two phases subnormal					315	11.1	295	93.7	189	60.0	30	9.5		
4	S	S	N	N	5	0.2	5	100.0	4	80.0	0	0.0	0.13	1.06
5	S	N	S	N	60	2.1	57	95.0	31	51.7	1	1.7	–3.97	3.74
6	N	S	S	N	107	3.8	98	91.6	62	57.9	11	10.3	–3.28	4.58
7	N	S	N	S	42	1.5	41	97.6	27	64.3	7	16.7	–3.74	4.56
8	N	N	S	S	37	1.3	37	100.0	32	86.5	6	16.2	–5.04	3.62
9	S	N	N	S	64	2.2	57	89.1	33	51.6	5	7.8	–1.80	4.63
One phase subnormal					1,015	35.6	761	75.0	212	20.9	5	0.5		
10	S	N	N	N	121	4.2	89	73.6	18	14.9	1	0.8	–0.83	4.89
11	N	S	N	N	208	7.3	130	62.5	33	15.9	1	0.5	–0.56	4.60
12	N	N	S	N	274	9.6	227	82.9	68	24.8	1	0.4	–2.24	4.85
13	N	N	N	S	412	14.5	315	76.5	93	22.6	2	0.5	–1.19	4.71
No phase subnormal														
14	N	N	N	N	1,484	52.1	367	24.7	25	1.7	0	0.0	1.75	4.72
Total					2,850	100.0	1,459	51.2	461	16.2	62	2.2		

* None of the children had subnormal growth during all four growth phases.
† S, subnormal growth (i.e., a decrease of ≥3 in centimeter score during any growth phase); N, normal growth.
‡ CMS, centimeter score; SD, standard deviation.

TABLE 4. Correlation coefficients (*r*) between height centimeter score (CMS) and the change in height CMS with growth (during the fetal, infancy, childhood, and puberty phases) and midparental height in healthy full term Swedish children (*n* = 2,850), 1973–1993*

	Height CMS at age (years):				Gain in height CMS from:		
	0	2	8	18	0–2 years	2–8 years	0–18 years
Midparental height	0.23**	0.43**	0.48**	0.59**	0.27**	0.39**	0.29**
Height CMS at:							
Birth		0.43**	0.37**	0.41**	–0.33**	0.21**	0.14**
2 years			0.82**	0.71**	0.71**	0.42**	0.002
8 years				0.80**	0.57**	0.86**	–0.10**
18 years					0.43**	0.64**	0.51**
Gain in height CMS from:							
Birth to 2 years						0.28**	–0.11**
2 years to 8 years							–0.17**

* Because results were similar for boys and girls, pooled results are presented.
** Significant at $p < 0.01$.

score into the analyses (9). Thus, our results are not directly comparable to those of previous studies.

The findings suggest that adult shortness is usually associated with subnormal growth in multiple phases, rather than any one single phase. Most adult shortness is the result of subnormal growth in two or three phases. Of the 62 children who were short in terms of final height (–12 cm below the reference means), 57 (92 percent) had a poor height gain during two or three phases. As many as 75 percent of the children with a poor gain during three phases became short in adulthood; this represents 44 percent (27/62) of all of the short adults. For those with a poor gain during two phases, 9.4 percent became short as adults; they represented 48 percent (30/62) of all short adults. The probability of adult shortness when subnormal growth occurred in only one phase was very low (0.5 percent). For the first time, we can conclude that adult shortness is, to a large extent, a result of multiple growth phase deficits. For instance, there were 121 children with sub-

TABLE 5. Odds ratios for a final height below a centimeter score (CMS) of 0, –6, or –12, as estimated by multiple logistic regression analysis, in Swedish children (*n* = 2,850), 1973–1993*,†

Growth phase	Odds ratio excluding midparental height		Odds ratio including midparental height	
	OR‡	95% CI‡	OR	95% CI
Final height below reference mean				
Fetal	6.8	4.7, 9.9	5.5	3.5, 8.6
Infancy	4.8	3.6, 6.3	4.1	2.9, 5.9
Childhood	13.0	9.6, 17.5	9.4	6.5, 13.6
Puberty	9.5	7.4, 12.1	7.3	5.4, 9.8
Final height ≥6 cm below reference mean				
Fetal	4.9	3.5, 6.9	4.4	3.0, 6.4
Infancy	6.5	4.8, 8.8	5.5	3.8, 8.1
Childhood	12.2	9.1, 16.3	8.4	5.9, 11.8
Puberty	12.4	9.2, 16.7	8.8	6.2, 12.5
Final height ≥12 cm below reference mean				
Fetal	15.3	6.9, 33.8	14.7	5.8, 37.2
Infancy	39.4	16.7, 92.9	38.9	14.5, 104.1
Childhood	28.4	12.8, 63.0	16.1	6.4, 40.4
Puberty	51.7	21.2, 125.7	26.9	11.0, 81.2

* The independent variables were subnormal growth during the fetal, infancy, childhood, and puberty growth phases. Subnormal growth was defined as a decrease of ≥3 CMS during a particular growth phase.
† All results were significant at $p < 0.0001$.
‡ OR, odds ratio; CI, confidence interval.

normal birth length but normal growth during the subsequent three phases; only one (0.8 percent) had a final height 12 cm below the reference mean.

Previous growth studies used to attribute adult shortness to subnormal growth in one phase, most often fetal growth retardation or infancy growth stunting (3–14). The possible influence of one growth phase on subsequent phases has been less explored. We found that the four growth phases are not independent. The fetal and infancy phases are negatively correlated, which is consistent with previous reports that short babies tend to catch up during the first 2 years of life (3–9). However, being short at birth is a disadvantage for height gain during the childhood and puberty phases; a larger birth size confers greater growth potential on a child in the long run. Knowledge of these relations could help pediatricians to anticipate a child's growth pattern. Birth length has a long term influence on postnatal growth. Short infants appear to catch up somewhat during the first 2 years of life, but their gains in height fall behind again in subsequent phases.

The gains in height are positively correlated between the infancy and childhood phases but negatively correlated between the childhood and puberty phases. Height at 2 years of age is important for final height prognosis; children who are born short and those who become short during the first 2 years of life have similar risks for adult short stature (9). Children who are taller at 2 years of age tend to grow better during childhood. Better growth during the first 2 years of life is an advantage for growth during the childhood phase, but this advantage may be partly compromised later by a smaller gain in height during the puberty phase. Less gain in height during the childhood phase may prompt more gain in height during the puberty phase. Catch-up growth during puberty is also possible, and it is more likely to occur if the child has experienced malnutrition in childhood (12, 19). It is known that children with a constitutional growth delay may show a late onset of puberty, one that is delayed by more than 2 years; thus, the overall gain in height from 8 years of age onward is increased because of a longer effective growth span (20).

It is interesting to note that the relations between the different growth phases are different. The relation between the fetal and infancy growth phases is well understood: Short newborns usually show catch-up growth in early postnatal life due to relaxation from the potential physiologic or pathologic constraints in the uterine environment (8, 10). The biologic meaning and nature of the relations between other growth phases remains to be explored. Better childhood growth may prompt earlier onset of the pubertal growth spurt, and thus relatively less height gain during the puberty phase because of a shorter effective growth span. The timing of the pubertal growth spurt has a significant effect on final height (15). So far, there has not been any information available on the relative importance of the various growth phases for final height. The reason for this is the lack of longitudinal studies following children from birth to final height. The present study provides readers with such information, with new insight into the complexity of postnatal growth, by considering the individual growth phases.

Midparental height, as an indicator of the genetic potential in stature for a child, has a positive influence on postnatal growth during all growth phases. The odds ratios for adult shortness were virtually the same for the fetal and infancy phases, but they decreased dramatically for the childhood and puberty phases when midparental height was included in the logistic regression model. It appears that the influence of subnormal growth during childhood and puberty interacts with midparental height. However, the influence of intrauterine growth retardation and infancy growth faltering is not affected by midparental height—an estimate of the genetic potential in stature. We speculate that either the genetic influence on stature becomes apparent from the childhood phase (9) or the consequences of subnormal growth in early life—the fetal and infancy phases—are more profound and more difficult to redress in later life. For instance, most children tend to show catch-up growth after growth faltering caused by infectious disease; however, catch-up growth does not occur if the illness afflicted a child during infancy (21). The fetal and infancy phases should be the most critical period for preventing severe adult shortness.

Some caution should be taken in interpreting our findings. One important issue is the definition of subnormal growth. There is no unique documented definition for subnormal growth in the literature. We defined subnormal growth as a decrease of 3 CMS or more in a certain growth phase. This definition was largely empirical and pragmatic, and –3 CMS does not represent the same degree of subnormal growth in different phases. An interesting topic could be raised for further studies: What are the most effective cutoff points for capturing subnormal growth in various growth phases? In addition, there are diverse pictures of growth in different populations. The Swedish population under study was relatively homogeneous, while populations in other countries, such as the developing countries, have much more diversity. Our findings are not necessarily applicable to other populations without further validation.

We conclude that subnormal growth during any growth phase is associated with an increased risk for adult shortness. Most adult shortness is the result of subnormal growth in two or three growth phases. The four growth phases are associated: Height at the start of each growth phase influences growth in the subsequent phase. Being small at birth prompts catch-up growth during the infancy phase but less gain in height during the childhood and puberty phases. The infancy and childhood growth phases are positively associated, while the childhood and puberty phases are negatively associated. The effects of subnormal growth during the childhood and puberty phases are influenced by midparental height, while the effects of subnormal growth during the fetal and infancy phases are not affected by genetic potential in stature.

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