# Catch-up growth in appropriate- or small-for-gestational age preterm infants

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The aim was to evaluate postnatal growth of preterm infants in childhood and to determine factors that have an effect on catch-up growth (CUG). Ninety-six (42F, 54M) preterm born children with a gestational age of 32.6±2.9 weeks and birth weight of 1815±668 g were evaluated at age 4.7±1.1 years. Preterm children with birth weight and/or length below 10<sup>th</sup> percentile were accepted as small-for-gestational age (SGA) and those above as appropriate-for-gestational age (AGA). Height SDS was similar (-0.5±1.0) in preterm AGA and SGA children. Both groups had low body mass index (BMI) SDS (-0.6±1.4 and -1.0±1.5, respectively). Of the preterm SGA children, 65.8% showed a CUG in height and 3.8% catch- down growth. These rates were 24.6% and 33.5% in preterm AGA children. CUG in height was best explained by birth length and mother's height and CUG in weight by birth weight and mother's weight. In conclusion, although most of the preterm SGA children show CUG, they reach a compromised height in childhood. A number of preterm AGA children show a catch-down growth.

Key words: preterm, growth, appropriate-for-gestational age (AGA), small-for-gestational age (SGA), bone age.

Studies of postnatal growth in preterm infants have revealed conflicting data due to the heterogeneous characteristics of the study groups. Catch-up growth (CUG) was reported in a number of studies<sup>1-4</sup>, while some studies showed compromised height in this group of children in early– to mid-childhood<sup>5-14</sup>. Even if normal height was achieved, preterm children were found to be short for their genetic height potential<sup>15</sup>. Other studies on very low birth weight (VLBW) preterm children followed up to early or late adolescence showed incomplete CUG16-18 - only in males16 or only in preterm children with LBWs for gestational age (SGA) compared to those with birth weights appropriate for gestational age (AGA)<sup>1,8,9,11,12,18</sup>. However, being SGA did not appear to have an effect on CUG among the preterm infants in other studies<sup>6,8,19,20</sup>. While CUG may continue up to mid-childhood and adolescence<sup>2-4,8,11,17,19,22</sup> in some, preterm

children usually show most of their CUG by 2-3 years of age<sup>6,14,21</sup>. Several studies have pointed out the relationship between LBW and future morbidity due to cardiovascular disease<sup>23</sup>; however, the role of CUG has also been implied in the development of disease states<sup>24</sup>. Thus, it would be desirable to follow children with LBW for growth impairment and also for potential risk for future morbidities. The aim of this study was to analyze the growth characteristics of a group of pretermborn children during prepubertal ages.

### Material and Methods

Of 208 children who were born with a gestational age (GA) of <37 completed weeks and admitted to the Neonatology Unit, those who were at least 3.0 years of age at the time of investigation were invited to participate in a cross-sectional growth study in the pediatric

endocrinology and growth unit. Twenty-nine children were not reachable at the postal address and the parents of 25 children refused to come. Altogether, 96 (42F, 54M) preterm born children participated in the study. The children did not have neurologic impairment, severe systemic diseases or malformations. The group included 11 pairs of twins and one set of triplets. The groups of children who were not reachable or refused to come were found to be similar to the study population with respect to GA, birth weight, perinatal risk factors and hospital care. Medical history regarding GA, weight and length at birth (n=72) and details of postnatal history including feeding regimens were taken from the hospital files, while information regarding infancy and subsequent years was ascertained from the parents. All newborn babies <1500 g were either not given enteral feeding or only minimal enteral feeding with breast milk during the first three days. Parenteral nutrition was started on the second day of life. After enteral feeds reached 100 ml/kg/day, breast milk fortifier was added. Babies ≥1500 g were fed enterally starting from the first day of life with a volume of 20 ml/kg/day with increments of 20 ml/kg/day. Weights reached at term (40±2 weeks) were recorded from the files. GA was determined by the mother's last menstrual period, the Ballard assessment<sup>25</sup> and antenatal fetal ultrasound examination. Mean GA of the study group was 32.6±2.9 weeks. Birth weight was 1815±668 g, and expressed as standard deviation score (SDS) (26), was  $-0.8\pm1.0$  SDS. Mean birth length was  $42.7\pm5.1$ cm (-0.6±1.3 SDS) and head circumference (HC) -0.7±1.1 SDS. Forty-seven children were VLBW preterms (birth weight <1500 g and/or GA <32 weeks).

Mean chronological age (CA) at the time of the investigation was 4.7±1.1 years (range 2.9-7.1 years). Following a thorough physical examination, anthropometric measurements including height, weight, skinfold thicknesses (ST) (subscapular and triceps), waist circumference, and HC were taken by standard methods<sup>27</sup> using Harpenden equipment for height/length and sitting height and by one auxanologist (MS). Parental weights and heights were also measured. Body mass index (BMI) of the children and parents was calculated as weight (kg)/height (m²). Target height was calculated as mother's height +

father's height/2 - 6.5 for girls and + 6.5 for boys. Values of height, weight, target height<sup>28</sup>, HC<sup>29</sup>, BMI<sup>30</sup> and ST<sup>31</sup> were expressed as SDSs. Sitting height was expressed as % of height. Corrected height for target height (target height SDS minus current height SDS) was denoted as Height SDS<sub>corrected</sub>. Bone age was determined from the left hand and wrist X-rays by Greulich–Pyle method<sup>32</sup>.

The children were divided into two groups with respect to their birth weight and length for their GA. Those with a birth weight and/or length <10th percentile, namely an SD score below -1.3 SD (33,34) were accepted as SGA preterms (PSGA) (n: 31) and those with a birth weight and/or length ≥10th percentile as AGA preterms (PAGA) (n: 63). Two cases with an uncertain GA were excluded from this analysis. CUG in height was defined as the difference ( $\Delta$ ) between birth length SDS and current height SDS (Δ height SDS), and CUG in weight was defined as the difference ( $\Delta$ ) between birth weight and current weight SDS ( $\Delta$  weight SDS). Those who had a  $\Delta$ height SDS or  $\Delta$  weight SDS over 0.67 SD (35), in other words, those who had moved one centile band up, were accepted as having shown a CUG in height or weight. Those who moved one centile band down were accepted as having a catch-down growth. The education level of the parents was evaluated at three levels: elementary school level (total 8 years of education)=1, high school level=2 and university level=3.

Statistical analyses were done using an SPSS-12 program. Comparison between the means was analyzed by the t-test and comparison between the percentages by the  $\chi^2$  test. Univariate linear correlation was done to analyze the effects of several potential factors on  $\Delta$  height and  $\Delta$  weight SDS. Multivariate linear regression analysis (forward LR method) was then performed to evaluate the effect of confounding factors on CUG. Δ height SDS and  $\Delta$  weight SDS were taken as the dependent variables, and GA, birth weight, birth length, birth HC and maternal weight and height were taken as the independent variables. Values are expressed as means and standard deviations. Significance was accepted as  $p \le 0.05$ .

Consent was obtained from the families, and the study was approved by the ethical committee.

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#### Results

Etiology of preterm birth was unknown in 49.0% of the children. Identified causes in the rest were gestational diabetes mellitus (n:1), hypertension (n:4), multiple births (n:12), preeclampsia (n:7), and others (n:7). Physical examination and body proportions were normal.

As seen in Table I, at age 4.7±1.1 years, all preterm-born children had weight, height and BMI values within normal ranges, but mean values were below the mean values of normal healthy children (p=0.000). Current height, on the other hand, was not significantly different from target height SDS, as is evident from the corrected height SDS of the children. When analyzed individually, heights of 9%

of children were below -2 SD and heights of 16.5% of the children were shorter than their target height SDS by more than 1 SD. It was noteworthy that although subscapular ST was within normal ranges, triceps ST SDS was far below the mean. In fact, 69% of children had triceps ST values below -2 SD and only 12% had subscapular ST values below -2 SD. All children were prepubertal. Bone ages were appropriate for their chronological ages.

When analyzed with respect to their GAs, the PSGA children were shorter and lighter at birth compared to PAGAs, but they made a significantly higher CUG in weight and height, reaching a similar weight and height SDS as the PAGA children at around five years of age (Table II). HC SDS, although still slightly smaller in PSGAs, also showed a CUG, and

Table I. Age and Anthropometric Parameters in the Study Group of Preterm-Born Children (mean ±SD)

Chronological age (yrs)	4.7±1.1 Triceps ST SDS	-3.2±2.2	
Weight SDS	$-0.6\pm0.9$	Subscapular ST SDS	$-0.7 \pm 1.3$
Height SDS	$-0.5\pm1.0$	Bone age (yrs)	$4.3 \pm 1.4$
Head circumference SDS	- 0.8±1.2 Target height SDS	$-0.3 \pm 0.8$	
BMI SDS	- $0.7\pm0.9$ Height $SDS_{corrected}$	$-0.2 \pm 1.0$	

SDS: Standard deviation score. BMI: Body mass index. ST: Skinfold thickness. p=0.000, comparison from the median (0 SD) of normal children.

Table II. Anthropometric Parameters in the PAGA and PSGA Subgroups (mean±SD)

At birth	PAGA	PSGA	P
Gestational age (week)	32.6±3.1	32.6±2.5	NS
Weight SDS	$-0.3 \pm 0.6$	$-1.9 \pm 0.9$	0.000
Length SDS	$0.0 \pm 1.1$	$-1.8 \pm 0.8$	0.000
HC SDS	$-0.2 \pm 0.9$	$-1.6 \pm 0.9$	0.000
BMI	$10.0 \pm 1.7$	$8.8 \pm 1.6$	0.02
At present			
Chronological age (yrs)	$4.8 \pm 1.1$	$4.6 \pm 1.3$	NS
Weight SDS	$-0.5 \pm 1.0$	$-0.6 \pm 1.0$	NS
Height SDS	$-0.5 \pm 1.1$	$-0.5 \pm 1.1$	NS HC SDS
	$-0.6 \pm 1.1$	$-1.1 \pm 1.4$	NS
BMI SDS	$-0.6 \pm 1.4$	$-1.0 \pm 1.5$	NS
Waist circumference (cm)	$51.4 \pm 4.0$	$50.7 \pm 4.5$	NS
Triceps ST SDS	$-3.1 \pm 2.0$	$-3.6 \pm 2.6$	NS
Subscapular ST SDS	$-0.5 \pm 1.1$	$-1.2 \pm 1.6$	0.05
Δ height SDS	$-0.3 \pm 1.4$	$1.4 \pm 1.5$	0.000
Δ weight SDS	$-0.2 \pm 1.0$	$1.3 \pm 1.1$	0.000
Target height SDS	$-0.3 \pm 0.7$	$-0.3 \pm 0.9$	NS
Height SDS <sub>corrected</sub>	-0.2±0.9 -0.2±1.0	NS	

PAGA: Preterm appropriate-for-gestational age. PSGA: Preterm small-for-gestational age. SDS: Standard deviation score. HC: Head circumference. BMI: Body mass index. ST: Skinfold thickness. NS: Nonsignificant.

there was no significant difference between the HC of the PAGAs and PSGAs. There was no difference in target height of the two groups or in BMI- and weight-related indices between the groups, except for a significantly lower subscapular ST in PSGAs.  $\Delta$  height and  $\Delta$  weight SDS were significantly higher in the PSGAs than in the PAGAs. When analyzed on an individual basis, 67.6% and 65.8% of PSGA children showed a  $\Delta$  weight and  $\Delta$  height SDS above 0.67 SD.

There was no significant difference between the PAGA and PSGA groups with respect to several neonatal characteristics, except for length of time it took to institute full oral feeding and for duration of hospitalization (Table III). These periods were longer in the p=002) and mother's height (r=0.331, p=0.002), and current height with target height (r=0.451, p=0.001). No correlation was present between current weight and height measurements and GA.  $\Delta$  Height SDS, on the other hand, showed a negative correlation with birth weight SDS (r=-0.468, p=000), birth length SDS (r=-0.789, p=0.789)p=0.000) and HC SDS (r=-0.442, p $\leq$ 0.001). It did not correlate with target height or father's height but showed a relationship to mother's height SDS (r=0.316, p=0.009).  $\Delta$  Weight SDS showed similar correlations, but also showed a negative correlation with BMI at birth (r=-0.248, p=0.037) and mother's weight SDS (r=0.302, p=0.006). Neither  $\Delta$  height SDS nor  $\Delta$  weight SDS showed any correlation with GA. Multivariate analysis demonstrated that the best

Table III. Neonatal Characteristics of the PAGA and PSGA Children (mean±SD or %)

	PAGA	PSGA	P
Mechanical ventilation (%)	22.0	32.0	NS
Days of O <sub>2</sub>	$2.1 \pm 1.2$	$3.2 \pm 4.9$	NS
Days on antibiotics	$11.8 \pm 10.4$	$13.3 \pm 7.6$	NS
Days until full oral feeding is instituted	$6.6 \pm 6.3$	$13.9 \pm 7.6$	0.001
Days of parenteral nutrition	$4.8 \pm 3.3$	$10.8 \pm 7.4$	NS
Glucocorticoid treatment			
Antenatal (%)	8	23	NS
Postnatal (%)	3	11	NS
Birth asphyxia	3	NS	
Duration of breastfeeding (months)	$9.9 \pm 10.0$	$5.2 \pm 4.3$	NS
Age at start of formula feedings (months)	$3.2 \pm 3.2$	$3.1 \pm 4.0$	NS
Intracranial hemorrhage (%)	10	15	NS
NEC (%)	0	6	NS
Days of hospitalization	$21.9 \pm 19.6$	$35.2\pm23.8\ 0.033$	

PSGA: Preterm small-for-gestational age. PAGA: Preterm appropriate-for-gestational age. NS: Nonsignificant. NEC: Necrotizing enterocolitis.

PSGAs than in the PAGAs probably because PSGAs had experienced more adverse events. Other than these parameters, feeding regimens did not differ among the two groups. Weight reached at term (40±2 weeks) was 2840±20 g in the PAGA and 1954±245 g in the PSGA children and did not show correlation with current height or weight.

Socioeconomic evaluation of the families showed that there was no significant difference between the groups with respect to the parents' level of education (data not shown).

Correlation studies revealed that current weight showed a positive correlation with birth weight (r=0.248, p=0.015), mother's weight (r=0.330, p=0.015)

model that predicted  $\Delta$  height SDS (R<sup>2</sup>=0.746) was birth length SDS (unstandardized coefficient) B=-0.978, p≤0.001), mother's height SDS (B=0.458, p=0.001) and mother's weight SDS (B=0.140, p=0.014) (constant= -0.377). The best model that predicted  $\Delta$  weight SDS (R<sup>2</sup>=0.534) was birth weight SDS (B=-0.763, p≤0.001) and mother's weight SDS (B=0.196, p=0.001) (constant= -0.550).

## Discussion

The results of our study showed that preterm born children, regardless of their GA and birth weight, reach a height slightly compromised compared to the normal population but normal with respect to their parents. Volume 50 • Number 3 Growth in Preterm Infants 211

Our results are in compliance with studies that report a height deficit of 0.5 to 1.0 SD in childhood or older ages in preterm infants with a similar range of GAs (6,10,16-18). Nine percent of the children in our series were short (<-2 SD) and 16.5% more than 1 SD below their target height. In a large series of VLBW boys with a height SDS of -0.4±1.1 at 20 years, 7% were below -2 SD<sup>16</sup>. Even in papers that report favorable results in postnatal growth of preterm children, ~10% of children are short or do not reach their genetic height potential<sup>4</sup>.

In our series of preterm children, BMI was more affected than height and these children were thin. In fact, in 14% of the children, BMI SDS was below -2, whereas it was above 2 SDS in only 3%. It has also been reported in other series that these children are thinner than their counterparts<sup>6,10,12,16,18,19</sup>. It is noteworthy that this thinness is mostly due to inadequate peripheral fat, indicated by the low triceps ST values, while subscapular ST, an indicator of truncal adiposity, is within normal ranges. This finding may have important implications for future morbidities of these children because truncal fat has been found to be more correlated with insulin resistance and cardiovascular risk factors<sup>36</sup>. ST measurement is an easy and noninvasive method for evaluation of adiposity in children<sup>37</sup>.

Advanced bone ages have been reported in PSGA children (38,39), which could further compromise final height in these children. In our group, bone ages were appropriate for chronological age.

Preterm SGA children in our series showed a satisfactory CUG and reached a height similar to PAGA children in mid-childhood and also within the ranges reported for the PSGA children in reported studies<sup>1,8,9,12,15,16,18,20</sup>. PAGA children in our study, on the other hand, reached a height within the ranges reported but slightly at the lower end. Strauss et al.<sup>12</sup> reported a height SDS of ~-0.3 SD in PSGA children in childhood as compared to + 0.2 SD in PAGA children. A height SDS of ~-0.5 SD in PSGAs versus a height SDS of -0.1 in PAGAs was found in the study on a large group of VLBW children by Knops et al. 18. It was noteworthy that PAGA children in our study showed a decrease in height SDS in mid-childhood with respect to their birth data,

indicating that some of these children showed a catch-down growth. Indeed, 41.9% of the PAGA children showed a  $\Delta$  height SDS within one centile band (- 0.67 to + 0.67 SD) and 33.5% showed a  $\Delta$  height SDS <0.67 SD. These rates were 30.4% and 3.8%, respectively, in the PSGA children (p=0.001). Moreover, 66% of the children who were short for their parents and 87% of the ones who were below -2 SD were PAGA children. This catch-down growth has been pointed out in some other studies as well<sup>20</sup>. In fact, an association was reported between poor neurological outcome and catch-down growth in these PAGA children. They did worse than PSGA children.

In our study, there were no major differences with respect to perinatal history and other environmental factors between the two groups of preterms that would account for their different growth patterns. On the contrary, days until full oral feeding was instituted and days of hospitalization were longer in the PSGA children who showed a better CUG. It may be argued that if there are no differences in the peri- and postnatal history of PAGA and PSGA infants, it may be possible that these PAGA children were already on a catch-down growth before birth and their postnatal growth curve could be the continuation of insufficient intrauterine growth. Alternatively, it may be speculated that PAGA children who show a stable intrauterine growth react in a different way postnatally and may be more sensitive to environmental factors than PSGA children. who, once freed from the intrauterine extrinsicconstraining effect, react differently and with an exaggerated catch- up response provided that they are given good care. Animal studies also show that CUG occurs in intrauterine-restricted offspring if postnatal support is given<sup>40</sup>.

There was no difference between premature AGA and SGA children with respect to weight and adipose tissue-related indices, with both groups being thin. In both groups, truncal fat was less affected than peripheral fat (Table II).

When confounding factors on CUG were analyzed, it was seen that at a given GA, those who were shorter at birth showed a significant CUG in height and those who were lighter at birth showed a significant CUG in weight. Mother's height and weight were also effective in the magnitude of CUG in height

and weight, respectively. Different factors have been held responsible for the postnatal growth of preterm children: maternal education<sup>16</sup>, maternal height<sup>11,16</sup>, mother's weight<sup>1</sup>, perinatal factors<sup>11,16</sup>, parental height<sup>1,18</sup>, birth weight<sup>11,17,22</sup> and birth length<sup>17,21</sup>. In our study, although current weight showed a positive correlation with birth weight and current height with target height, CUG was mainly correlated to being SGA at birth and maternal factors.

It may be concluded that the intrauterine constraint in PSGA children is partially overcome post-term. As a result, most of these children show a very satisfactory CUG, and end up with a mildly compromised height and weight. On the other hand, a number of PAGA children show catch-down growth, which projects their curve downwards compared to their status at birth. Thus, PAGA children may have risk of growth impairment and should be followed as meticulously as the PSGA children.

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