Effect of Birth Size and Catch-Up Growth on Adult Blood Pressure and Carotid Intima-Media Thickness

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Key Words

Cardiovascular risk factors • Short stature • Catch-up growth • Small for gestational age

Abstract

Aims: To investigate the effect of birth size and weight gain during childhood on blood pressure and carotid intima-media thickness (cIMT) in young adulthood. **Methods:** The relationship of birth size with systolic blood pressure (SBP), diastolic blood pressure (DBP), and cIMT was investigated in 243 adults, aged 18–24 years. SBP, DBP, and cIMT were also analyzed in 4 subgroups: subjects either born small for gestational age with short stature (SGA-S) or with catch-up growth (SGA-CU), or born appropriate for gestational age with idiopathic short stature or with normal stature (controls). **Results:** Adult weight SDS and fat mass were positively related to SBP and DBP, adjusted for birthweight SDS which was not related to SBP and DBP. Birth size was also not related to cIMT. Subgroup analyses showed no differences in blood pressure between subgroups, but cIMT was significantly greater in SGA-CU subjects than in controls after correction for age, gender and artery diameter. This difference became borderline significant after additional correction for smoking and

SBP. **Conclusion:** Not birth size but childhood weight gain, especially fat mass, determines young adult blood pressure. Postnatal catch-up growth appears to have a greater influence on cardiovascular disease markers than birth size.

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Introduction

Vascular health deteriorates during life as cardiovascular risk factors induce structural vascular changes that can lead to atherosclerosis. This process already starts in childhood [1, 2]. Some studies showed an association between low birthweight and an increased risk for cardiovascular diseases (CVD) [3], but also postnatal catch-up in weight has been acknowledged as a determinant of cardiovascular risk [4–9]. As 90% of subjects born small for gestational age (SGA) show catch-up in weight and length during childhood [10, 11], these subjects might be at risk for CVD later in life.

The relationship between birthweight and adult blood pressure is one of the most investigated associations based on the 'fetal origin' hypothesis, but clarification is still needed as several studies and reviews showed conflicting

results [12–18]. One review reported that the association between birthweight and adult blood pressure may be overestimated due to bias in the reporting of results [19]. On the other hand, this has been opposed as well [20, 21].

We hypothesized that weight gain during childhood, not birth size, is related to adult blood pressure and carotid intima-media thickness (cIMT). Blood pressure and cIMT are accurate predictors of cardiovascular events later in life [22–25], and cIMT may be regarded as a valid marker for generalized atherosclerosis [26]. We therefore investigated the influence of birth length, birthweight, adult size and body composition on systolic (SBP), diastolic blood pressure (DBP) and cIMT in 243 young adults born term. Additionally, we investigated if there were differences in SBP, DBP and cIMT between four clinically recognizable subgroups of young adults; born SGA with either short adult height (SGA-S) or normal adult height (SGA-CU), or born appropriate for gestational age with either idiopathic short stature (ISS) or normal adult height (controls).

Subjects and Methods

Subjects

The PROgramming factors for GRowth And Metabolism (PROGRAM) study investigates a cohort of 323 healthy subjects aged 18–24 years. The participants were selected on being born SGA (birth length <-2 SD) [27] or showing short stature (adult height <-2 SD) [28], using registration data from academic hospitals in the Netherlands. In addition, healthy subjects were recruited to participate as controls, using advertisement. Only Caucasian subjects born singleton and at 36 weeks or more of gestation were invited to participate. All participants fulfilled the same inclusion criteria: an uncomplicated neonatal period without signs of severe asphyxia (defined as an Apgar score below 3 after 5 min), without sepsis or long-term complications of respiratory ventilation, such as bronchopulmonary dysplasia. Subjects were excluded if they had been suffering from any serious condition or had been receiving any treatment known to interfere with growth (e.g. growth hormone deficiency, severe chronic illness, emotional deprivation, growth hormone treatment, treatment with glucocorticosteroids, radiotherapy) or if they had endocrine or metabolic disorders, chromosomal defects, syndromes or serious dysmorphic symptoms suggestive of a syndrome. Birth data were taken from records of hospitals, community health services and general practitioners. The Medical Ethics Committee of Erasmus University Medical Centre, Rotterdam, the Netherlands, approved this study. Written informed consent was obtained from all participants.

Of 323 participants who entered the study, 80 had incomplete blood pressure and cIMT data, resulting in a total number of 243 eligible subjects for analyses. There were no differences in anthropometric and dual-energy X-ray absorptiometry (DXA) measurements between the included and excluded participants.

Additionally, based on SD scores of birth length and adult height, the subjects were assigned to one of four subgroups. In order to increase the statistical power for subgroup comparison, the cut off values were set at -2 SDS and -1 SDS. Of the 243 subjects, 170 fulfilled the inclusion criteria for the subgroup analyses: born SGA (<-2 SDS) with a short adult height (<-2 SDS; SGA-S, $n = 34$), born SGA (<-2 SDS) with catch-up growth resulting in a normal adult height (>-1 SDS; SGA-CU, $n = 44$), born appropriate for gestational age (>-1 SDS) with growth retardation resulting in a short adult height (<-2 SDS; ISS, $n = 28$), born appropriate for gestational age (>-1 SDS) with a normal adult height (>-1 SDS; controls, $n = 64$).

Measurements

All participants visited Erasmus University Medical Centre in Rotterdam. They had been fasting for 12 h and had abstained from smoking and alcohol for 16 h. Height was measured to the nearest 0.1 cm by a Harpenden stadiometer and weight to the nearest 0.1 kg by a scale (Servo Balance KA-20-150S, Servo Berkel Prior, Katwijk, Netherlands). All anthropometric measurements were performed twice, and the mean value was used for analyses. All subjects filled out a questionnaire on alcohol use (dichotomous: yes or no), smoking (yes or no) and their socioeconomic status (SES), which was based on educational level [29]. Lean body mass and fat mass were measured by one DXA machine (Lunar Prodigy, GE Healthcare, Chalfont St Giles, UK). Quality assurance was performed daily. The intra-assay variation for lean tissue and fat tissue has been reported to be 1.57–4.49 and 0.41–0.88%, respectively [30]. Blood pressure was measured in the sitting position after 5 min rest with an automatic device (Accutorr Plus, Datascope Corp., Montvale N.J., USA). Blood pressure was measured every 5 min for 1 h and the average value of 13 measurements was taken to reflect 24-hour blood pressure. IMT was measured in supine position by recording of ultrasonographic images of both left and right carotid artery using the same 7.5-MHz linear array transducer (ATL Ultramark IV, Advanced Tech. Laboratories, Bethel, Wash., USA). On the R wave of the electrocardiogram, three longitudinal images of the near and far wall of the common carotid artery were frozen and stored on videotape. These frozen images were digitalized and displayed using a frame grabber (VP 1400-KIT-512-E-AT, Imaging Technology). The common carotid IMT was determined as the mean of the mean near and far wall measurements of both the left- and right-side common carotid artery.

Statistical Analysis

To correct for gestational age, gender and age, SD scores for birth length, birthweight, adult height and adult weight were calculated [27–28]. Multiple linear regression (MR) analysis was performed to determine the association between birth size and SBP, DBP and cIMT. Firstly, we entered age, gender, adult height SDS, birth length SDS and birthweight SDS to investigate the influence of birth size on the outcomes (model A). The interaction term birth length SDS $\times$ adult height SDS was added to all models because the study group had been selected based on birth length and adult height, in order to ensure that the effect of these variables was modeled correctly. Secondly, for SBP and DBP heart rate was entered, and for cIMT artery diameter was entered (model B). Thirdly, adult weight SDS was added for birthweight SDS, to relate adult size to the outcomes (model C). Fourthly, birthweight SDS

Table 1. Clinical characteristics of the total study group and the four subgroups

Variables	Total group (n = 243)	SGA-S (n = 34)	SGA-CU (n = 44)	ISS (n = 28)	Controls (n = 64)
Male, %	37.9	29.4	38.6	39.3	40.6
Age, years	20.9 (1.7)	20.6 (1.7)	21.4 (1.4)	20.7 (1.8)	20.7 (1.8)
Birth length, cm	47.7 (3.1)	45.0 (2.2)	44.5 (2.0)	49.4 (1.2) ¹	50.9 (1.8) ¹
Birth length SDS	-1.46 (1.47)	-2.99 (0.88)	-2.85 (0.85)	-0.56 (0.48) ¹	0.14 (0.73) ¹
Birthweight, kg	2.81 (0.69)	2.45 (0.47)	2.17 (0.38)	3.23 (0.60) ²	3.36 (0.58) ²
Birthweight SDS	-1.12 (1.42)	-2.02 (0.92)	-2.36 (0.75)	-0.13 (1.23) ²	0.08 (1.17) ²
Adult height, m	1.69 (0.11)	1.57 (0.07)	1.75 (0.08) ³	1.59 (0.06)	1.78 (0.08) ³
Adult height SDS	-1.03 (1.41)	-2.61 (0.59)	-0.11 (0.75) ¹	-2.40 (0.36)	0.38 (0.91) ¹
Adult weight, kg	63.7 (12.4)	56.9 (12.2)	71.2 (13.2) ³	54.6 (10.5)	69.4 (11.1) ³
Adult weight SDS	-0.63 (1.43)	-1.50 (1.64)	0.21 (1.11) ³	-1.96 (1.66)	0.10 (0.93) ³
BMI	22.4 (3.5)	23.1 (5.0)	23.2 (3.6)	21.5 (4.0)	21.8 (2.8)
Fat mass, kg*	15.2 (8.0)	16.3 (9.4)	18.3 (8.2)	13.7 (8.4)	15.0 (8.0)
Fat mass, SDS*		0.15 (1.17)	0.40 (1.02)	-0.17 (1.05)	0.00 (1.00)
Trunk fat/total fat ratio*	0.48 (0.06)	0.47 (0.06)	0.49 (0.07)	0.46 (0.06)	0.47 (0.05)
Lean body mass, kg*	45.4 (10.1)	37.9 (7.8)	49.3 (9.9) ³	37.9 (6.3)	51.2 (9.5) ³
Lean body mass, SDS*		-1.41 (0.82)	-0.21 (1.04) ³	-1.41 (0.67)	0.00 (1.00) ³
Alcohol users, %	75.7	76.5	80.0	82.1	78.1
Smokers, %	25.5	20.6	35.0	39.0	20.3
cIMT, mm*	0.52 (0.05)	0.50 (0.05)	0.53 (0.06) ⁴	0.50 (0.05)	0.52 (0.05)
Artery diameter, mm*	6.66 (0.48)	6.38 (0.41)	6.76 (0.42) ⁴	6.43 (0.45)	6.79 (0.48) ³
cIMT/artery diameter ratio	0.078 (0.007)	0.079 (0.007)	0.079 (0.008)	0.077 (0.007)	0.076 (0.007)
SBP, mm Hg*	110.0 (9.0)	107.8 (10.2)	112.4 (10.2)	107.5 (10.2)	110.1 (7.2)
DBP, mm Hg*	66.1 (6.0)	66.2 (8.0)	66.6 (6.1)	65.2 (6.9)	66.1 (5.0)
Heart rate, beats/min*	65 (9.0)	71 (9.6) ⁵	65 (9.1)	67 (7.7)	64 (8.4)

Values are given as means (SD). Asterisk signifies not corrected for age, gender and adult size.

¹ p < 0.01 compared with the other subgroups.

² p < 0.001 compared with SGA-S and SGA-CU.

³ p < 0.01 compared with SGA-S and ISS.

⁴ p < 0.05 compared with SGA-S and ISS.

⁵ p < 0.05 compared with SGA-CU and controls.

was added again to the model. When birth size and adult size are shown in one model, one can investigate if the change in weight (centile crossing) between birth and adulthood is related to the outcomes (model D). For SBP and DBP, corrections were made for SES, smoking, alcohol use, and an additional model was made with fat mass and lean body mass to specify adult weight SDS. For cIMT, corrections were made for SES, smoking, alcohol use, and SBP. Using a sample size of 243, we calculated that we could detect at least 2% extra variance in SBP, DBP and cIMT attributable to birthweight (with 80% power and a significance level of 0.05). Differences between the subgroups with regard to the group characteristics were determined using ANOVA. Bonferroni correction was used for pairwise group comparisons. An ANCOVA model was used to determine differences between the groups corrected for age, gender and for variables with a p value < 0.1 in the MR analyses. Controls served as the reference group. The statistical package SPSS version 15.0 (SPSS, Inc., Chicago, Ill., USA) was used for analysis. Results were regarded as statistically significant if p was < 0.05.

Results

Clinical characteristics of the total study population are shown in table 1. The study population consisted of 243 subjects with a mean age (SD) of 20.9 (1.7) years.

Relationship of Birth Size with Blood Pressure and cIMT in Young Adulthood

Multiple regression (MR) models showed that male gender, alcohol use, and lower SES were associated with higher SBP (table 2). Birth length SDS and birthweight SDS were not associated with SBP (model A). In the second model, heart rate was added, which was significantly positively associated with SBP (model B). In the third model, birthweight SDS was replaced by adult weight SDS to investigate the relationship between adult size and SBP

(model C). Adult weight SDS was a strong positive determinant of SBP in young adulthood. Birthweight SDS was added to model B to determine whether weight gain from birth to young adulthood was related to SBP (model D). Adult weight SDS remained significant, while birthweight SDS was not, indicating that more weight gain during childhood was related to a higher SBP in young adulthood, independent of birthweight. Finally, adult weight was specified by fat mass and lean body mass (model E). Fat mass was positively related to SBP, thereby indirectly showing that fat accumulation from birth to young adulthood was positively related to SBP. Furthermore, fat mass explained the gender difference in SBP. In the final model, alcohol use, heart rate and SES remained significantly positively associated with SBP, while SES was inversely related.

DBP was not associated with birth size, while age, gender, SES, smoking and alcohol use were significant determinants (model A). Heart rate was positively associated with DBP (model B). Adult weight was a strong positive determinant of DBP (model C), and after adding birthweight SDS to the model, the weight gain during childhood proved to be determinant of DBP, independent of birthweight SDS. After specifying adult weight in fat mass and lean body mass, fat mass appeared to be a positive determinant of DBP in young adulthood, next to alcohol use and heart rate. This indirectly indicates that fat accumulation from birth to young adulthood is positively related to DBP.

Table 3 shows the relationship between cIMT and birth size. Birth length SDS and birthweight SDS were not significantly associated with cIMT in young adulthood. Female gender and artery diameter were significant positive determinants, whereas adult height SDS was inversely related to cIMT (model B). Birthweight SDS was replaced by adult weight SDS to investigate whether weight in young adulthood influenced cIMT, but its effect was not significant (model C). Weight gain from birth to young adulthood did not influence cIMT, while female gender, SBP and artery diameter remained positive determinants (model B). Specification of adult weight SDS in fat mass and lean body mass did not change the results (data not shown).

Notably, women had a greater cIMT than men in the final model. When analyzing this gender difference without corrections, men had greater cIMT and larger artery diameter than women (0.53 vs. 0.51 mm, $p = 0.006$, and 6.94 vs. 6.48 mm, $p < 0.001$, respectively). Nevertheless, the cIMT/artery diameter ratio was higher in women than in men (0.079 vs. 0.077, $p = 0.006$), indicating that

Table 2. Multiple regression of SBP and DBP in young adulthood

Variables	SBP, mm Hg						DBP, mm Hg					
	model A		model B		model C		model D		model E		model F	
	β	p	β	p	β	p	β	p	β	p	β	p
Age, years	0.18	0.615	-0.01	0.989	-0.03	0.929	-0.01	0.970	-0.24	0.485	0.55	0.034
Gender	-6.13	<0.001	-7.85	<0.001	-7.68	<0.001	-7.61	<0.001	-3.56	0.660	-0.51	0.577
SES	-3.49	0.001	-2.65	0.011	-2.53	0.009	-2.39	0.014	-2.32	0.021	-4.18	0.001
Smoking	-1.72	0.230	-1.18	0.399	-0.26	0.844	-0.33	0.803	-0.21	0.874	-2.75	0.008
Alcohol use	3.55	0.017	3.81	0.008	3.26	0.014	3.38	0.011	3.54	0.009	-6.45	0.089
Adult height SDS	0.54	0.358	0.63	0.267	-0.68	0.248	-0.70	0.230	-1.06	0.138	-0.28	0.506
Birth length SDS	0.43	0.573	0.66	0.376	0.19	0.683	0.79	0.257	0.79	0.263	0.22	0.690
Birth weight SDS	-0.55	0.423	-0.78	0.240			-0.71	0.248	-0.83	0.184	-0.07	0.886
Heart rate, beats/min			0.29	<0.001	0.27	<0.001	0.28	<0.001	0.29	<0.001	0.235	<0.001
Adult weight SDS					2.36	<0.001	2.35	<0.001			1.247	<0.001
Fat mass, kg									0.22	0.003		
LBM, kg									0.39	0.130		
Overall	<0.001		<0.001		<0.001		<0.001		<0.001		<0.001	
R ² adjusted	0.18		0.24		0.34		0.34		0.33		0.25	

Gender: 1 = men, 2 = women; LBM = lean body mass. All models include the interaction term birth length SDS $\times$ adult height SDS, gender $\times$ lean body mass is included in model E, and SES $\times$ alcohol use is included in the models with DBP as dependent variable.

Table 3. Multiple regression models for cIMT in young adulthood

Variables	cIMT, mm							
	model A		model B		model C		model D	
	β	p	β	p	β	p	β	p
Age, years	0.001	0.557	0.001	0.646	0.001	0.718	0.001	0.658
Gender	−0.006	0.414	0.022	0.004	0.023	0.003	0.024	0.003
SES	−0.002	0.782	−0.003	0.536	−0.004	0.500	−0.003	0.545
Smoking	0.013	0.144	0.013	0.075	0.013	0.087	0.012	0.095
Alcohol use	−0.006	0.489	−0.001	0.947	−0.001	0.910	0.000	0.960
Systolic BP, mm Hg	0.001	0.002	0.001	0.044	0.001	0.023	0.001	0.026
Adult height SDS	−0.003	0.400	−0.007	0.033	−0.005	0.134	−0.005	0.126
Birth length SDS	−0.001	0.791	0.000	0.957	−0.002	0.461	0.000	0.993
Birth weight SDS	−0.001	0.869	−0.002	0.506			−0.002	0.498
Artery diameter, mm			0.061	<0.001	0.062	<0.001	0.062	<0.001
Adult weight SDS					−0.003	0.316	−0.003	0.313
Overall		0.027		<0.001		<0.001		<0.001
R ² adjusted		0.05		0.29		0.29		0.29

Gender: 1 = men, 2 = women. All models include the interaction term birth length SDS × adult height SDS.

Table 4. Subgroup analyses of SBP and DBP

Variables	SBP, mm Hg				DBP, mm Hg			
	model 1		model 2		model 1		model 2	
	β	p	β	p	β	p	β	p
SGA-S	−0.006	0.998	−0.203	0.927	0.734	0.644	0.061	0.969
SGA-CU	1.729	0.390	−0.370	0.838	0.506	0.723	−0.402	0.757
ISS	−2.911	0.197	−0.618	0.788	−0.662	0.678	0.435	0.793
Overall		0.001		<0.001		0.776		<0.001
R ² adjusted		0.12		0.34		−0.02		0.21

Reference group is controls. Model 1: corrected for age and gender. Model 2: corrected for age, gender, heart rate, SES, smoking, alcohol use and adult weight SDS.

women have a relatively thicker carotid arterial wall than men.

Comparison of Subgroups

The clinical characteristics of the four subgroups are shown in table 1. Differences between the subgroups with regard to anthropometric measurements were in agreement with inclusion criteria. There were no differences in SBP and DBP between the subgroups. Carotid IMT was significantly higher in SGA-CU subjects compared to SGA-S and ISS subjects. Carotid artery diameter was sig-

nificantly smaller in the subgroups with short adult height than those with normal adult height. There was no difference in cIMT/artery diameter ratio between the subgroups. Heart rate was significantly higher in SGA-S subjects than in SGA-CU and controls.

After correction for age and gender, and additional correction for heart rate, SES, smoking, alcohol use and adult weight SDS, there were no significant differences between the subgroups with regard to SBP and DBP (table 4).

SGA-CU subjects also had a higher cIMT than SGA-S and ISS subjects after correction for age and gender (table 5). Additional correction for artery diameter resulted in a similar cIMT in subjects with short adult stature and those with normal adult stature, indicating that subjects with short stature have smaller carotid arteries and therefore a lower cIMT. SGA-CU subjects had also a significantly higher cIMT than controls after correction for artery diameter ($p = 0.025$), which became borderline significant after correction for variables with a p value <0.1 in the MR analyses.

Discussion

This study shows that birthweight and birth length SDS had no significant influence on blood pressure and cIMT in young adulthood. Fat mass was a positive determinant for both SBP and DBP in young adulthood, next to heart rate, while SES was inversely related. Alcohol use was positively related to SBP and inversely related with DBP. For cIMT, female gender, SBP and artery diameter were positive determinants. Subgroup analyses showed that there was no difference in blood pressure between the subgroups, but cIMT was significantly higher in SGA-CU subjects than controls.

The relationship between birth size and subsequent blood pressure has been previously investigated [12–18], with controversial results. This is likely due to different study designs, publication bias and different statistical approaches [19]. In order to investigate the relationship between birth size and blood pressure, we used the statistical model of Leeson et al. [31], and adjusted for several confounding factors. Our results indicate that increased fat accumulation during childhood is a risk factor for higher blood pressure, independent of birth size. However, because our results comprise data at birth and in early adulthood, we cannot draw conclusions on the time window during childhood, in which fat accumulation is most important. Our results are in line with previous studies reporting a positive association between weight gain or fat accumulation in childhood and later blood pressure [7, 8, 32, 33].

Infant nutrition is an important determinant of weight gain in early life [32]. SGA infants who received nutrient-enriched formulas during early life showed more catch-up in weight, which has been associated with increased blood pressure in childhood [34]. In view of these and our results, nutritional intervention studies should be undertaken to investigate which feeding regimen provides the

Table 5. Subgroup analyses of cIMT

Variables	cIMT, mm					
	model 1		model 2		model 3	
	β	p	β	p	β	p
SGA-S	−0.023	0.060	0.000	1.000	0.001	0.920
SGA-CU	0.018	0.084*	0.022	0.025	0.018	0.062
ISS	−0.020	0.086	0.001	0.920	0.000	0.965
Overall		0.002		<0.001		<0.001
R ² adjusted		0.09		0.25		0.28

Reference group is controls. Model 1: corrected for age, gender. Model 2: corrected for age, gender, and artery diameter. Model 3: corrected for age, gender, artery diameter, smoking and SBP.

* $p < 0.01$ compared with SGA-S and ISS.

optimal growth in early life with regard to blood pressure in later life. Awaiting these results, it seems best to avoid an exaggerated catch-up in weight-for-height during early life.

Birth size had no influence on cIMT in young adulthood. Adult weight or fat mass were no determinants of cIMT either, which was in contrast to our expectations. Previous reports showed that an increase in body mass index (BMI) during childhood was associated with cIMT in adulthood [2, 35]. However, we could not confirm such a relation with either BMI or fat mass, which might be explained by the small range of BMI and the relatively young age of the participants in our study.

Although men had greater cIMT than women when no corrections were made, women had a higher cIMT/artery diameter ratio, and female gender was positively related to cIMT in the MR analyses. This indicates that women have a relatively thicker carotid arterial wall. One explanation might be a different composition of the arterial wall in women which might preclude a different plaque phenotype in later life than men [36]. Our data warrant further investigation.

The MR analyses and subgroup analyses showed that artery diameter is a significant factor for cIMT. Subjects with short adult height had smaller cIMT and smaller artery diameter. This indicates that they had smaller carotid arteries, which was confirmed by a similar cIMT/artery diameter ratio as controls. These results might suggest that future studies investigating cIMT need to adjust for artery diameter, otherwise cIMT will be underestimated in short subjects.

Subgroup comparison showed no differences in blood pressure between the four clinically relevant subgroups. We expected that SGA-CU subjects would have a higher blood pressure than controls, because they had more fat mass than controls [37]. Although the MR analyses showed a positive relationship between fat mass and blood pressure, blood pressure was not significantly different between the subgroups. This is probably due to the relatively small differences in fat mass between the subgroups. Both subgroups born SGA did not have a higher blood pressure, which is in line with the MR analyses showing that a lower birthweight does not increase the risk for higher blood pressure.

SGA-CU subjects had a greater cIMT than controls. This became borderline significant after correction for smoking and SBP. In contrast, SGA subjects without catch-up growth had a similar cIMT as controls. Our results are in line with Oren et al. [38], who previously reported that young adults with the lowest birthweight and the highest postnatal growth had a greater cIMT than young adults with normal birth size and postnatal growth. However, they were unable to make a distinction whether prenatal or postnatal growth influenced cIMT in later life. We show that not prenatal growth but postnatal growth influenced cIMT in adulthood, as SGA-CU subjects and not SGA-S subjects had a greater cIMT in young adulthood.

Our study population consisted of a relatively high percentage of subjects born SGA and of short adults. This created greater contrast in the study population, which contributed to a model with more statistical power, in which relationships between various factors could be de-

tected. In addition, this study population allowed comparison between clinically relevant subgroups.

In conclusion, our study shows that catch-up growth in weight during childhood, specified as fat accumulation, influences blood pressure in later life. Birth size did not influence blood pressure and cIMT in young adulthood. Subgroup comparison showed that subjects born SGA with catch-up growth had a greater cIMT in young adulthood than controls. Therefore, fat accumulation during childhood should be prevented to attain normal blood pressure in young adulthood, and more research is warranted to investigate whether subjects born SGA with catch-up in weight have a higher risk for cardiovascular events in later life.

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Disclosure Statement

The authors have nothing to disclose.

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