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# Low maternal education is associated with increased growth velocity in the first year of life and in early childhood

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## ABSTRACT

### Objective

The objective of this study is first to examine educational disparities in growth velocity during the first year of life and early childhood (1 year – 5 years) and second, to determine the potential explanatory role of standardized birth weight, maternal smoking during pregnancy, maternal prepregnancy body mass index (BMI), and infant feeding practice.

### Methods

We used longitudinal growth data of 1684 participants with Dutch ethnicity participating in a population-based cohort study (ABCD study). Growth velocity of weight and of weight-for-length were calculated by subtracting the weight and weight-for-length standard deviation scores (sds), respectively of two time periods.

### Results

In the first year of life, children with low-educated mothers had an increase in sds of 0.26 (95% CI 0.08 – 0.45) for weight compared to children with high-educated mothers. In early childhood, children with low-educated mothers had a 0.27 sds (95% CI 0.11 – 0.42) increase for weight-for-length, compared to children with high-educated mothers. Using path-analysis, these inequalities could partly be explained by maternal smoking, duration of breastfeeding, maternal age, and maternal BMI.

### Conclusions

Educational inequalities in childhood obesity may have an origin in the first years of life as children with less educated mothers had an increased weight gain during the first year of life and an increased weight-for-length gain in early childhood. Although underlying mechanisms were not completely clarified, an optimal duration of breastfeeding, cessation of maternal smoking and reduction of maternal BMI seem to reduce educational inequalities in early growth and possible adverse consequences of accelerated growth.

## INTRODUCTION

Accelerated growth in infancy and early childhood has been associated with increased risk of childhood obesity,[148-150] adult obesity,[151,152] hypertension,[10] insulin resistance,[153,154] and other determinants of cardiovascular disease.[152,154,155] Barker et al[153] described that the risk of coronary heart disease in adults was more strongly related to the increase of body mass index (BMI) in childhood than to the BMI attained at any particular age. Therefore, optimisation of infant and child growth could lead to substantial reductions in adverse health outcomes in later life.[156]

As there is compelling evidence that low birth weight,[157] and higher weight in childhood[35] are associated with lower socioeconomic status (SES), on average children of lower SES may have increased growth velocity in the first years of life. Previous observed determinants of growth velocity are: infant feeding practices,[36,37] maternal smoking,[37,158] parental overweight,[37,159,160] and ethnicity.[37] A few studies addressed socioeconomic differences in growth velocity,[37,158,159,161] which were limited to weight or length alone. However, measures of size that include length in addition to weight reflect body size and adiposity better than weight alone[162,163] and it was observed that weight-for-length gain had a greater effect on later obesity than weight gain.[162] To our knowledge only Wijlaars et al.[158] focussed on the underlying factors of socioeconomic differences in weight gain in a twin cohort. This study showed that infant feeding practices partly explained the diverging weight trajectories in the first few months. Yet, it is not known to what extent the determinants of growth velocity explain the socioeconomic gradient in growth velocity throughout the first years of life.

Using data from a population-based birth cohort, we tested the hypothesis that growth velocity in the first year of life as well as in early childhood is higher in children with low-educated mothers. Maternal education is chosen as indicator of SES, because socioeconomic disparities in the prevalence of obesity have typically been observed by education.[35] Growth velocity was determined using longitudinal measurements of weight, and weight-for-length. In addition, this study aimed to explain educational inequalities in growth velocity in these two time periods by potential mediators such as standardized birth weight, maternal smoking during pregnancy, duration of breastfeeding, age at introduction of solid foods, and maternal prepregnancy body mass index.

## METHODS

### Study Design and Population

The present study is part of the Amsterdam Born Children and their Development (ABCD) study, a prospective cohort study from fetal life onward which started in 2003-2004 with 8266 women that returned pregnancy questionnaire (mean gestational age: 15.7 weeks (SD

3.5)). The cohort has been described in detail elsewhere.[43] The sampling procedure of the current study is shown in Figure 5.1.

Growth data were routinely collected at the Youth Health Care registration of the Public Health Service in Amsterdam by well trained nurses and Youth Health Care medical doctors. Data at 1 month of age were used because neonates may lose up to 10 percent of their birth weight in the first days of life and length is not measured routinely at birth. If a certain measurement did not take place at the exact time, we derived this value by interpolating between the nearest measurements. The allowed age ranges for the interpolation at 1 and 12 months were 0-2 months and 9-15 months respectively. Physical measurement at age

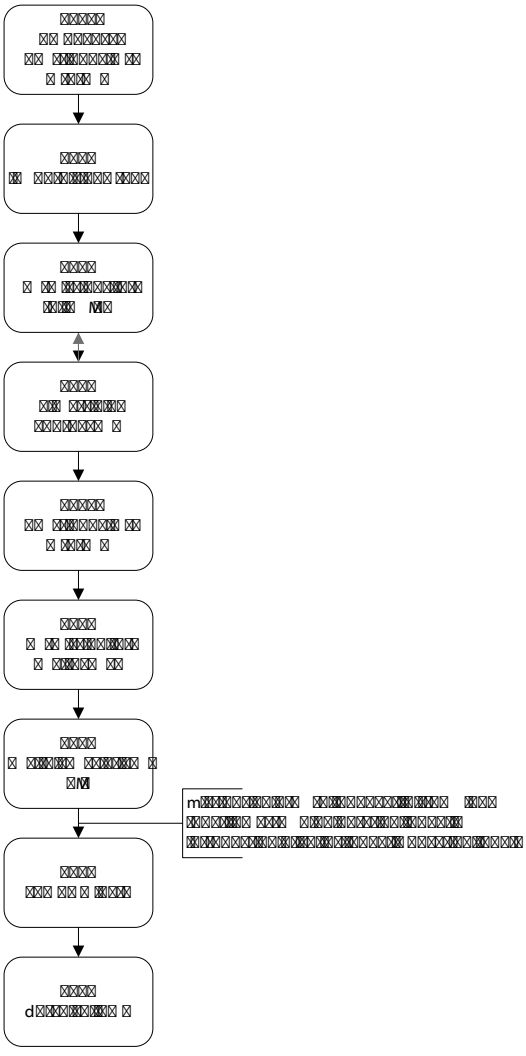

**Figure 5.1.** sampling procedure

5-6 included weight and height (vertical position) assessment. Height was measured to the nearest 0.1 cm using a Leicester portable height measure (Seca), and weight was measured to the nearest 0.1 kg using a Marsden weighing scale, model MS-4102. More details about the age 5 health check were described previously.[164] Attrition in follow-up was largely due to untraceable changes in address or migration. As weight gain and educational level differs by ethnicity,[36] only children with a Dutch mother were selected for the present study. Finally, 1684 mother–child pairs with complete data were included in the analyses.

This final sample of 1684 cases was comparable to the Dutch sample that underwent the health check (n = 2464) in terms of BMI at age 5 (p = 0.93) or maternal BMI (p = 0.51). In contrast, the final sample is breastfed for a shorter duration (p < .001) and these mothers were higher educated (10.7 yr vs 10.3 yr; p = 0.02) compared to the Dutch sample that underwent the health check. Approval for the ABCD study was obtained by the institutional review board of Academic Medical Centre Amsterdam. All participating mothers gave written informed consent.



### Growth velocity

The outcomes were growth velocity in the first year of life and early childhood (between 1 year and 5 years). For each time period the average standardized growth velocity was calculated by subtracting the standardized weight or weight-for-length, at the earlier moment in time from the standardized weight or weight-for-length at the later moment in time, resulting in delta standard deviation scores (Δ sds). Sex specific Dutch reference standards were used in expressing weight and weight-for-length as standard deviation scores.[165]

### Maternal education

Although socioeconomic status can also be measured by income and occupation, socioeconomic disparities in the prevalence of obesity have typically been observed by educational levels and less consistently by income.[35] Therefore, we focussed on educational level as a measure of SES. Maternal educational level was reported in the pregnancy questionnaire by years of education after primary school. Educational level was categorized as low (< 6 years), mid (6 – 10 years), and high (> 10 years).

### Covariables

Literature on the determinants of growth velocity[37,158,159] was used to select potential mediators for the association between maternal educational level and growth velocity. Birth weight (continuous), gestational age and sex were obtained from the Youth Health Care Registration at the Public Health Service in Amsterdam. Dutch reference standards were used to standardize birth weight.[60] Maternal cigarette smoking during pregnancy (yes/no), maternal weight, maternal height, and maternal age were self-reported in the pregnancy questionnaire. Prepregnancy BMI was calculated (kg/m$^2$, continuous). Information about infant feeding was

obtained from the Youth Health Care centre, and by infant questionnaire. To complete our feeding data, 19.9% of feeding data was assessed retrospectively from the 5-years questionnaire. As described previously, infant feeding can reliably be reported retrospectively after 5 years.[166] Exclusive breastfeeding (< 1 month, 1 - 3 months, 3 - 6 months, > 6 months) and age at introduction of solid foods (< 4 months, 4 – 6 months, > 6 months) were categorized.

### Statistical analyses

Differences between educational groups were examined using a Chi-square test for categorical variables and ANOVA analysis for continuous variables. Educational differences in growth velocity were assessed with a linear regression analysis. Path-analysis mediation models were used to identify potential determinants of growth velocity that may explain the association of growth velocity with maternal education. Each path model consists of the following linear regression equations: A regression equation that describes the relationship between growth velocity and maternal education (adjusted for all the mediators), and the regression equations describing the relationship between each mediator and maternal education.[64] Associations between maternal education and the mediating risk factors were modelled with the weighted least squares algorithm implemented in M-PLUS.[167] A linear regression model was used for the continuous mediators, and a probit regression model was used for smoking. In the regression of categorical mediators on the outcomes, the mediators were replaced by underlying continuous latent variables. The indirect effects of the mediating risk factors were determined by calculating the product of the coefficients along a path. The proportion of the relationship between growth velocity and maternal education mediated by each of the mediators was determined by dividing each of the corresponding absolute indirect effects by the absolute total effect.[66] The assumptions required to test mediation hypotheses were met, although we cannot assert that associations were not confounded. SPSS 20.0 was used for the univariate analyses and M-PLUS (Muthen and Muthen) was used for the path-analysis mediation models. A p-value <0.05 was considered as significant.

## RESULTS

Table 5.1 presents the sample characteristics by maternal education. In general, low-educated mothers were younger, had a higher prevalence of smoking during pregnancy, and had a higher BMI. In addition, children of low-educated mothers were breastfed for a shorter duration and had an earlier age of introduction of solids. Five year olds with low-educated mothers had on average a higher weight, and weight-for-length ratio than children with high-educated mothers.

Linear regression analyses showed that children with mid- and low-educated mothers had an increased weight gain in the first year of life (β 0.12; 95% CI 0.02 – 0.22 and β 0.26; 95%

CI 0.08 – 0.45) and weight-for-length gain in childhood (β 0.10; 95% CI 0.02 – 0.18 and β 0.27; 95% CI 0.11 – 0.42) compared to children with high-educated mothers. There was no educational gradient in weight-for-length gain in the first year of life and weight gain in early childhood.

The path-analysis model was used to explain the associations of maternal education with infant weight gain and child weight-for-length gain. The left part of Figure 5.2 shows that a low-education was associated with higher odds of being exposed to tobacco during

**Table 5.1.** Baseline characteristics by maternal education

| | Maternal education | | | | |
|---|---|---|---|---|---|
| | Total | High | Mid | Low | p-value* |
| | (n=1684) | (n=1018) | (n=554) | (n=112) | |
| Infant characteristics | | | | | |
| Boys (%) | 52.0 | 53.2 | 49.3 | 54.5 | .28 |
| Standardized birth weight (SD) | 1.02 (0.12) | 1.02 (0.12) | 1.02 (0.13) | 1.01 (0.13) | .30 |
| Maternal characteristics | | | | | |
| Age, yr (SD) | 32.6 (4.0) | 33.2 (3.3) | 32.0 (4.5) | 30.1 (6.4) | <.001 |
| Smoking during pregnancy (%) | 6.1 | 2.3 | 8.7 | 28.6 | <.001 |
| Body mass index, kg/m$^2$ (SD) | 22.7 (3.5) | 22.3 (2.9) | 23.1 (3.9) | 24.2 (4.8) | <.001 |
| Feeding practice | | | | | |
| Exclusive breast feeding duration (%) | | | | | <.001 |
| < 1 month | 22.4 | 18.8 | 24.4 | 46.4 | |
| 1 – 3 months | 26.6 | 25.4 | 28.3 | 28.6 | |
| 3 – 6 months | 33.2 | 36.8 | 30.9 | 11.6 | |
| > 6 months | 17.8 | 19.0 | 16.4 | 13.4 | |
| Solid food introduction (%) | | | | | <.001 |
| < 4 months | 5.2 | 3.2 | 7.2 | 12.5 | |
| 4 – 6 months | 37.4 | 37.6 | 36.8 | 38.4 | |
| > 6 months | 57.4 | 59.1 | 56.0 | 49.1 | |
| Childhood characteristics | | | | | |
| Age, yr (SD) | 5.7 (0.5) | 5.7 (0.5) | 5.8 (0.5) | 5.8 (0.5) | .16 |
| Weight, kg (SD) | 21.0 (3.0) | 20.8 (2.8) | 21.1 (3.2) | 21.6 (3.9) | <.01 |
| Weight-for-length sds (SD) | -0.16 (0.88) | -0.21 (0.8) | -0.12 (0.9) | 0.08 (1.1) | <.01 |
| Outcomes | | | | | |
| Δ sds first year weight (SD) | -0.47 (0.95) | -0.52 (0.91) | -0.41 (0.99) | -0.27 (1.07) | <.01 |
| Δ sds first year weight-for-length (SD) | 0.08 (1.06) | 0.09 (1.05) | 0.07 (1.08) | -0.03 (1.13) | .54 |
| Δ sds early childhood weight (SD) | -0.18 (0.76) | -0.20 (0.73) | -0.17 (0.78) | -0.08 (0.90) | .27 |
| Δ sds early childhood weight-for-length (SD) | -0.26 (0.81) | -0.31 (0.79) | -0.21 (0.83) | -0.04 (0.90) | <.01 |

*p-values are based on One way ANOVA analysis and Chi square tests. Sds indicates standard deviation scores

pregnancy, a higher maternal BMI, a younger maternal age, an earlier introduction of solids, and shorter breastfeeding duration. The right part of Figure 5.2 shows that first year weight gain increased with decreasing child's standardized birth weight, decreasing maternal age, shorter breastfeeding duration, and if the mother has smoked during pregnancy.

The indirect effects of low-education on first year weight gain through mediating risk factors involved smoking during pregnancy, maternal age, and breastfeeding duration (Table 5.2). For example, the indirect effect of breastfeeding duration on the association between low maternal education and first year weight gain was 0.17 (95% CI 0.11 – 0.24), which is the product of the regression equations along that path (-0.72 * -0.24). After adjustment for mediators, the associations of maternal education with first year weight gain disappeared. Regarding the proportion of explanation by each of the risk factors (Table 5.2), smoking during pregnancy explained 28%, breastfeeding duration 27%, and maternal age 7% of the association between low maternal education and infant weight gain. Overall, 72% of the association of low maternal education with first year weight gain was explained by included mediators.

In early childhood, weight-for-length gain was associated with standardized birth weight and maternal BMI (Figure 5.3). The educational gradient in weight-for-length gain was mainly explained by maternal prepregnancy BMI (18%). Standardized birth weight, smoking during pregnancy, maternal age, age at introduction of solids, and breastfeeding duration did not play a role in the association between maternal education and childhood weight-for-length gain.

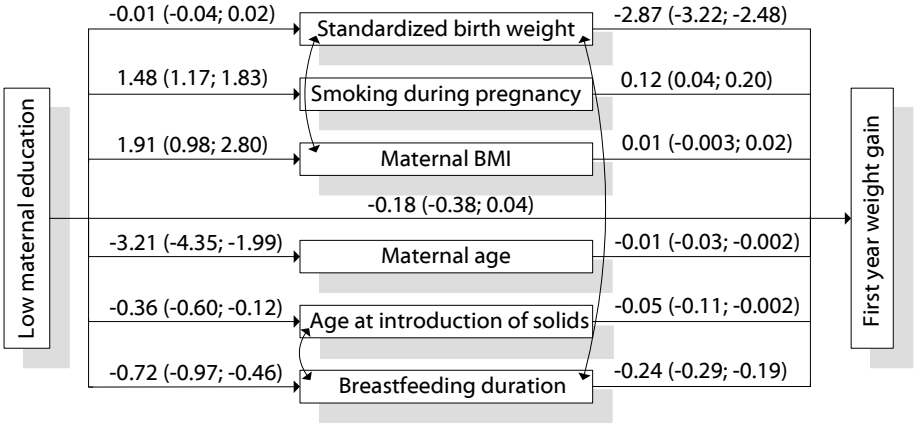


**Figure 5.2.** path analysis model with first year weight gain

**Table 5.2.** Indirect effects of the association between maternal education and growth velocity.

| | Mediation of the association between low maternal education and first year weight gain | | Mediation of the association between low maternal education and weight-for-length gain in early childhood | |
|---|---|---|---|---|
| Potential mediating risk factors | Indirect effect (95% CI) | Proportion mediated (%) | Indirect effect (95% CI) | Proportion mediated (%) |
| Standardized birth weight | 0.03 (-0.05 – 0.10) | 4 | 0.00 (-0.01 – 0.02) | 1 |
| Smoking during pregnancy | 0.18 (0.06 – 0.31) | 28 | 0.03 (-0.10 – 0.16) | 7 |
| Maternal prepregnancy BMI | 0.02 (-0.01 – 0.05) | 3 | 0.06 (0.03 – 0.11) | 18 |
| Maternal age | 0.05 (0.01 – 0.09) | 7 | 0.02 (-0.01 – 0.06) | 6 |
| Age at introduction of solids | 0.02 (0.00 – 0.05) | 3 | 0.01 (-0.01 – 0.03) | 2 |
| Breastfeeding duration | 0.17 (0.11 – 0.24) | 27 | -0.03 (-0.07 – 0.01) | -7 |
| Total effect | 0.63 (0.34 – 0.97)* | | 0.34 (0.21 – 0.62)** | |
| Total indirect effect*** | 0.46 (0.28 – 0.65) | 72 | 0.14 (0.10 – 0.30) | 41 |

Indirect effects were determined with path-analysis
*Total effect=Total indirect effect + abs(-0.18)
**Total effect=Total indirect effect + abs(0.20)
***Total indirect effect= sum of abs(indirect effects)

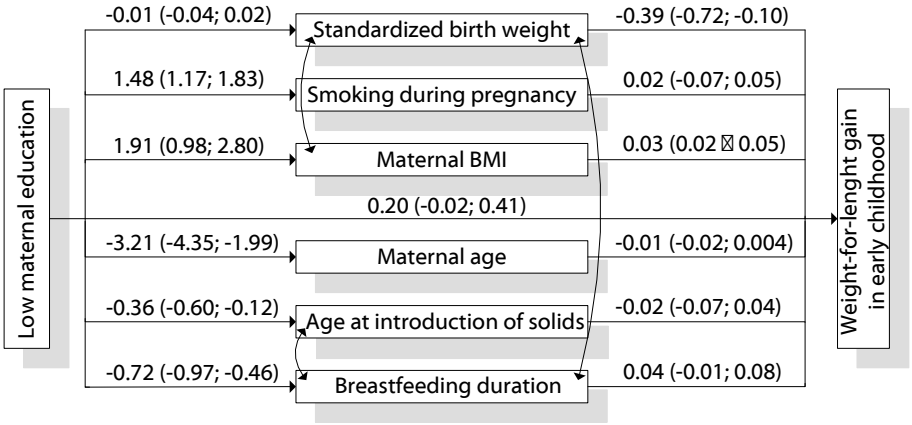


**Figure 5.3.** path analysis model with early childhood weight-for-length gain

## DISCUSSION

Children with low-educated mothers had increased weight gain in the first year of life and increased weight-for-length gain in early childhood compared to children with high-educated mothers. First year weight gain difference by maternal education was partly explained by maternal smoking, breastfeeding duration, and maternal age. The association between maternal education and childhood weight-for-length gain was partly explained by maternal BMI.

Our results regarding first year weight gain are comparable to the results of Wijlaars et al.[158] who found evidence to a SES gradient in a twin cohort. Although in this twin cohort only weight gain in the first 3 months of age was assessed, breastfeeding attenuated the association between SES and growth velocity mostly, followed by maternal smoking. Griffiths et al.[37] indicated low maternal education as a risk factor for increased weight gain in early childhood, but did not unravel explanatory factors. We found that children with low-educated mothers had increased weight-for-length gain in childhood compared to children with high-educated mothers, but there were no differences in weight-for-length gain in the first year of life. Proportionate growth in infancy seems to render in disproportionate growth in early childhood in children with low-educated mothers.

Duration of breastfeeding was identified as one of the explanatory factors in the relation of first year weight gain to maternal education. Various studies, like ours, have found that socially disadvantaged mothers start breastfeeding less often and also breastfeed for a shorter period.[168-170] Non breastfed children gain weight more quickly than breastfed children from birth to 1 year[171] and to 3 years.[36] A number of possible behavioural and biological explanations have been proposed, such as an early programming of appetite regulation and satiety.[36] In addition, lower protein and energy content of breast milk compared with formula, resulting in a diminished insulin release and thereby lower fat storage, may play a role. Besides duration of breastfeeding, maternal smoking and maternal age partly explain the association between maternal education and first year weight gain. As birth weight of smokers' offspring is lower, growth in smokers' offspring is suggested to 'catch-up' in infancy.[172] Moreover, smoking could lead to increased weight gain due to lower amounts (and duration) of breastfeeding among smokers.[168] Therefore, smokers' offspring more common were formula fed. In the present study, standardized birth weight and duration of breastfeeding seem not to explain the contribution of smoking in the association between maternal education and first year weight gain, since these factors were included in the model simultaneously. Maternal smoking possibly reflects an unhealthier lifestyle as smoking is associated with a poorer diet quality.[173] Additionally, control of appetitive behaviours may be decreased due to deficits in impulse-control in smokers' offspring.[174] Maternal BMI was previously associated with accelerated weight gain in early childhood,[37] but the contribution of maternal BMI in the relation of growth velocity to SES is not clear. Maternal BMI plays a role in the association

between maternal education and weight-for-length gain in early childhood and not in first year weight gain. As children are likely to be fully engaged the family lifestyle and eating habits after one year of age,[175] maternal BMI as a proxy of an unhealthy lifestyle rather affects weight-for-length gain in childhood instead of growth velocity in the first year of life.

The present study has a number of strengths. First, we assessed weight and weight-for-length, whereas previous studies assessed weight only, enabling an enhanced assessment of growth velocity in infancy and early childhood. Second, educational inequalities were not obscured by ethnicity as we used a homogeneous Dutch sample. Third, we used path-analysis accounting for the correlation between mediators. Some limitations have to be mentioned as well. Missing data and loss to follow-up has reduced the number of participants. Selective participation and loss to follow-up is present, as in most cohort studies. The current sample was longer breastfed and the mothers were more educated. Thus, the proportion of women in the low-educated group is probably higher at population level, implying an underestimation of the actual associations. Furthermore, duration of breastfeeding, smoking, and BMI were self-reported, which could result in inaccuracy. Unfortunately, we could not examine the influence of paternal overweight/obesity. Future studies should investigate the role of diet and physical activity and in the relation between growth velocity and socioeconomic status in childhood.



## CONCLUSION

In conclusion, our findings indicate that the educational gradient in obesity and cardiovascular disease may have an origin in the first years of life. Although causality remains to be established, promotion of optimal duration of breastfeeding, cessation of maternal smoking during pregnancy and reduction of maternal BMI, in particular in least educated women, is expected to contribute to normalizing infant and childhood growth and reduce adverse consequences of increased growth velocity, both in childhood and in later adult life.