7

Socioeconomic inequalities in body composition at age 5-6 explained by pre- and postnatal factors

Gerrit van den Berg, Manon van Eijsden, Francisca Galindo-Garre,
Tanja G. M. Vrijkotte, Reinoud J.B.J. Gemke

Submitted

ABSTRACT

Background

Low socioeconomic status has convincingly been associated with childhood obesity. The risk factors for childhood obesity appear to originate in the earliest stages of life.

Objective

We aimed to examine the role of early life factors such as maternal prepregnancy BMI, maternal smoking during pregnancy, standardized birth weight, infant feeding practice and infant first year weight gain in the association between socioeconomic status and childhood body composition.

Design

This is a prospective study in 1725 5-6 yr old Dutch children from the Amsterdam Born Children and their Development Study, a population-based pregnancy cohort.

Results

Using path-analysis, the higher BMI in children with low-educated mothers can be explained by maternal smoking during pregnancy (27%), maternal prepregnancy BMI (20%), infant weight gain between 0 and 3 months (14%), infant weight gain between 3 and 6 months (5%) and infant weight gain between 6 and 12 months (2%). The latter two act through a shorter breastfeeding duration. Regarding fat mass index (FMI), the results are comparable, though maternal smoking did not contribute to the association between maternal education and childhood FMI. BMI and FMI differences by family's income adequacy are explained by maternal prepregnancy BMI only (~30%).

Conclusion

Prevention programs to reduce socioeconomic inequalities in childhood body composition should start at pre- and postnatal care as these inequalities are partly explained by pre- and postnatal factors.

INTRODUCTION

Childhood obesity shows a clear socioeconomic gradient. Research carried out within the past 15 years indicates more adiposity in lower socioeconomic status (SES) groups.[35] Particularly children whose mothers have a low level of education are vulnerable to the development of obesity.

The higher prevalence of obesity amongst lower SES children reinforces the need to understand the mechanisms through which parental SES influences childhood obesity. Although childhood obesity has been largely attributed to an energy imbalance between calories consumed and calories expended,[220] there is increasing evidence that many risk factors for obesity originate in the earliest stages of life. These early environmental conditions in utero and during infancy may differ between socioeconomic groups and may lead to the socioeconomic gradient in obesity. One potential mechanism through which parental SES influences childhood obesity is rapid growth in infancy as rapid growth in infancy is strongly correlated with the child's obesity risk[221] and infants from lower SES families appear to have increased weight gain during infancy.[158] This might reflect catch-up growth because lower SES infants were born smaller for their gestational age.[30] A second potential mechanism for inequalities in childhood obesity is breastfeeding duration. Shorter breastfeeding is associated with more obesity[222] and lower SES children were breastfed for a shorter period of time.[168,223] This may act through rapid growth since formula fed children grow more rapidly compared to breastfed children.[36] Maternal prepregnancy BMI is the third potential mechanism as maternal BMI is strongly correlated with the child's BMI[224,225] and lower SES mothers had on average a higher BMI.[178] A higher prevalence of childhood obesity in lower SES groups could therefore be the consequence of a higher maternal BMI in these groups. Other determinants of childhood obesity, such as maternal age and smoking during pregnancy might also partly explain the socioeconomic gradient in childhood obesity. Although it can be hypothesized that the socioeconomic gradient in childhood obesity can partly be explained by early life risk factors, it remains unclear since there are no studies that have examined these pathways. Examining these pathways should be done simultaneously as it is likely that early life risk factors are highly correlated.

The aim of this study was to investigate the contribution of early life risk factors to the association of parental socioeconomic status with body mass index and fat mass index in the offspring at age 5-6 analysing data from a large population-based pregnancy cohort study (ABCD study) with a structural equation modelling approach.

SUBJECTS AND METHODS

Study design

This study was part of the Amsterdam Born Children and their Development (ABCD) study, a prospective cohort study. Details of this study were described previously.[43] Approval was obtained from the medical ethical committee of the Academic Medical Center Amsterdam, and the Registration Committee of Amsterdam. All participants gave written informed consent for themselves and their children.

Study population

In 2003-2004, 12 373 pregnant women were invited to participate at their first antenatal visit. 8266 women returned the pregnancy questionnaire including sociodemographic data, obstetric history, family history and lifestyle. Of the mothers with a singleton life birth (n= 7863), 6735 gave permission for follow-up (86%). When the children turned five, 6161 mothers received a questionnaire, including an informed consent sheet for a health check of their child. Attrition in follow-up number was largely due to untraceable changes in address or migration. 4488 questionnaires were returned (73%) and 4158 gave permission for the health check. The health check itself consisted of various health measurements in 3321 children aged five-six.

The present study excluded mother-child pairs of which growth data in infancy were not available (n=983). Furthermore, we excluded those of which bioelectrical impedance measurement was not available (n=23), those who had not filled out their educational level (n=47), or income adequacy (n=23). Our analyses leave out participants with missing data on confounders or mediators as well (breastfeeding duration n=10; complementary feeding n=25; birth weight n=2). Finally, 2208 mother-child pairs had complete data. Between this sample and the sample with health measurements (n=3321), differences are found in BMI (15.5 vs 15.6; $p = 0.004$), maternal BMI (22.9 vs 23.2 $p = 0.05$), childhood age (5.7 vs 5.9; $p < 0.001$), and years of maternal education after primary school based on pregnancy questionnaire (10.0 vs 9.0; $p < 0.001$). Thus, our sample seemed slightly healthier, younger, and higher educated than the health-check sample. As ethnicity was associated with both maternal education and body composition, we decided to include only children with a Dutch mother (n=1725) to limit confounding bias based on ethnicity.

Socioeconomic status

Socioeconomic status (SES) was first indexed using maternal education, as education level is the most consistent SES predictor of adiposity.[35] Maternal education was reported in the childhood questionnaire and was categorized as follows: low (no education, or primary school; lower vocational secondary education or technical secondary education); mid (higher vocational secondary education, intermediate vocational education); high (higher vocational education, university education).

Analyses were repeated with family income adequacy as indicator of SES to get more insight in the broad construct of SES. Family income adequacy was requested in the childhood questionnaire and was categorized into four categories: inadequate – scored if the mother filled out either "overdraft or in debt" or "using up my savings"; adequate – scored if the mother filled out "can just make ends meet"; bit more than adequate – scored if "can make ends meet and a bit more" was filled out ; and a lot more than adequate – scored if "can make ends meet and a lot more" was filled out.

Body composition

To calculate BMI, height (HT) was measured to the nearest millimetre using a Leicester height measure (Seca), and weight (WT) was measured to the nearest 100 gram using a Marsden weighing scale, model MS-4102. Arm-to-leg bioelectrical impedance analysis (BIA) was measured using the Bodystat 1500MDD system (Bodystat Inc, Douglas, UK) to obtain fat mass (FM).[46] FMI was calculated as FM/HT^5. As described by Wells et al,[226] this variable is an independent index of FM adjusted for body size.

Covariates

Literature on the determinants of body composition was used to select confounders and mediators in the relation of SES to BMI and FMI. Confounders were childhood age (continuous), and sex. Potential mediators were standardized birth weight (continuous), smoking during pregnancy (no/yes), maternal age (continuous), maternal prepregnancy BMI (continuous), breastfeeding duration (< 1 month, 1 - 3 months, 3 - 6 months, and > 6 months), age at introduction of solid foods (< 4 months, 4-6 months, and > 6 months), standardized weight gain 0-3 months (continuous), standardized weight gain 3-6 months (continuous), standardized weight gain 6-12 months (continuous). Birth weight, sex and gestational age were obtained from the Youth Health Care Registration. Dutch reference curves by gestational age, parity, and sex were used to standardize birth weight.[60] Smoking during pregnancy and maternal age were self-reported in the pregnancy questionnaire. Self-reported prepregnancy weight and height were used to calculate maternal BMI. Information on breastfeeding duration was available from the infancy questionnaire received when the child was 3 months, and from the Youth Health Care registration. In 19.9% of the cases, the prospectively collected information was combined with retrospective information of the 5-year questionnaire to complete the data. It was described that breastfeeding can be reliable reported retrospectively.[227] Information on introduction of solid foods was obtained from the Youth Health Care registration and the 5-year questionnaire (96%). Weight and height were routinely collected at the Youth Health Care Center of the Public Health Service by well trained nurses and Youth Health Care medical doctors. Weight gain was calculated by subtracting the standardized weight at the earlier moment in time from the standardized weight at the later moment in time, resulting in delta standard deviation scores (Δ sds). If weight 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 3, 6 and 12 months were respectively 2-4 months, 4-8 months and 9-15 months.

Statistical analyses

Differences between socioeconomic groups were examined using a Chi-square test for categorical variables and ANOVA analysis for continuous variables. Socioeconomic differences in BMI and FMI were assessed with a linear regression analysis adjusted for child's age and sex. Path-analysis mediation models were used to identify potential determinants of body composition that may explain the association of SES with body composition. The path-model consists of the regression equations that describe the relationship between body composition and potential mediators (adjusted for all mediators), and the regression equations describing the relationship between each mediator and socioeconomic status.[64] We hypothesized that the influence of birth weight and infant feeding practice on body composition acts through increased weight gain; hence weight gain within three time periods was placed after feeding practice in the model. Associations between socioeconomic status 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 the dichotomous variable (i.e 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 body composition and socioeconomic status originating from 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

The children's mean age was 5.7 years (SD 0.5). Most children had a high-educated mother and these mothers less often smoked during pregnancy, had a lower BMI, and are older than the low-educated mothers. Children with low-educated mothers had more weight gain in the first 6 months of life, had shorter breastfeeding duration, and an earlier age at introduction of solid foods (Table 7.1).

Table 7.1. Sample characteristics by maternal education

	Maternal education				
	Total	High	Mid	Low	p-value*
	(n=1725)	(n=1292)	(n=314)	(n=119)	
Age, yr (SD)	5.7 (0.5)	5.7 (0.5)	5.8 (0.5)	5.8 (0.5)	<.01
Sex (% boys)	51.1	51.5	50.6	47.9	.73
Income adequacy (%)					<.001
Lot more	28.8	33.8	17.2	5.0	
Bit more	42.1	42.9	40.4	37.8	
Adequate	20.5	16.7	28.7	39.5	
Inadequate	8.6	6.6	13.7	17.6	
Standardized birth weight (SD)	1.02 (0.12)	1.02 (0.12)	1.01 (0.13)	1.01 (0.15)	.26
Smoking during pregnancy (% yes)	6.1	2.7	12.7	25.2	<.001
Maternal prepregnancy BMI, kg/m^2 (SD)	22.7 (3.5)	22.3 (3.1)	23.3 (4.1)	24.5 (4.8)	<.001
Maternal age during pregnancy, yr (SD)	32.7 (4.0)	33.2 (3.5)	31.4 (4.8)	30.5 (5.6)	<.001
Delta sds weight 0-3 mo (SD)	-0.16 (0.84)	-0.19 (0.83)	-0.12 (0.86)	0.03 (0.89)	.01
Delta sds weight 3-6 mo (SD)	-0.15 (0.48)	-0.17 (0.47)	-0.08 (0.53)	-0.09 (0.48)	<.01
Delta sds weight 6-12 mo (SD)	0.12 (0.50)	0.12 (0.51)	0.11 (0.48)	0.09 (0.48)	.72
Breastfeeding duration (%)					<.001
> 6 mo	17.8	19.3	12.7	14.3	
3 – 6 mo	32.9	36.0	27.4	13.4	
1 – 3 mo	26.6	26.1	29.3	25.2	
< 1 mo	22.7	18.6	30.6	47.1	
Age at introduction of solids (%)					<.001
> 6 mo	57.1	59.2	51.6	48.7	
4 – 6 mo	37.3	36.9	40.8	32.8	
< 4 mo	5.6	3.9	7.6	18.5	
Fat mass index, kg/m^5 (SD)	1.93 (0.67)	1.89 (0.60)	1.99 (0.78)	2.11 (0.93)	<.01
BMI, kg/m^2 (SD)	15.37 (1.31)	15.31 (1.17)	15.50 (1.57)	15.65 (1.80)	<.001

*p-values are based on ANOVA's for continuous variables and Chi-square test for categorical variables.

7

Overall, mean BMI was 15.37 kg/m^2 (SD 1.31). In linear regression analyses, children with mid-educated mothers had a 0.19 kg/m^2 (95% CI 0.03 – 0.35) higher BMI and children with low-educated mothers had a 0.35 (95% CI 0.10-0.60) higher BMI compared to children with high-educated mothers. The path-analysis model was used to explain the associations of maternal education with BMI (Figure 7.1) estimating the regression equations simultaneously. The left part of figure 7.1 shows that compared to high-education, a low-education was associated with higher odds of being exposed to maternal smoking during pregnancy, higher maternal BMI, younger maternal age, shorter breastfeeding duration, and earlier age at introduction of solids. Subsequently, longer breastfeeding duration was associated with lower weight gain between 3 and 6 months and between 6 and 12 months of age. A higher weight gain between 0 and 3 months of age was associated with low maternal education and lower standardized birth weight. Standardized birth weight was also associated with weight gain between 3 and 6 and between 6 and 12 months of age. The right part of figure 7.1 shows that BMI at age 5-6 was associated with maternal smoking during pregnancy, increased maternal BMI, increased

standardized birth weight, and increased weight gain in the first year of life. There was no association of age at introduction of solids with weight gain and childhood BMI. From table 7.2 we infer that BMI-difference between children with low-educated mothers and children with high-educated mothers could be explained by maternal smoking (26%), maternal prepregnancy BMI (20%), weight gain between 0-3 months (13%), weight gain between 3 and 6 months (4%), and weight gain between 6 and 12 months (2%) (Table 7.2). The indirect effects of weight gain between 3 and 6 and weight gain between 6 and 12 months of age are

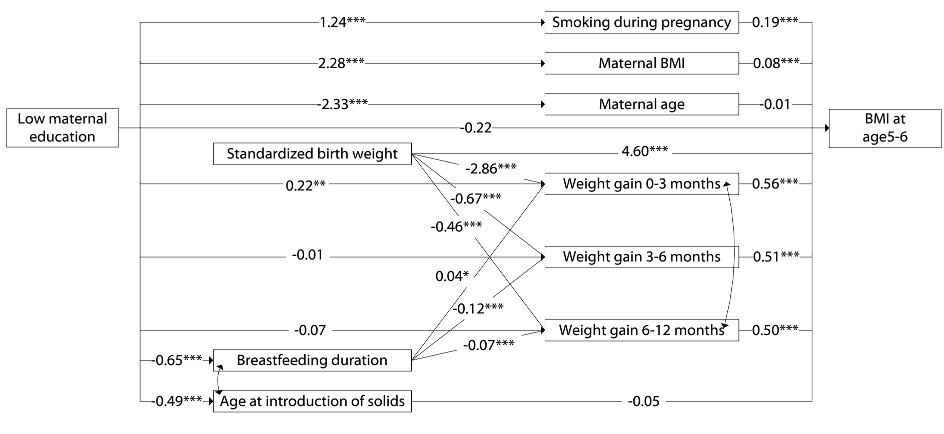

Figure 7.1. Graphic display of the estimated path-analysis model with maternal education. The regression equations with BMI and the potential mediators as the outcomes are represented by single-headed arrows. Double-headed arrows refer to the correlation between potential mediators. This model is corrected for childhood age and sex. Reference group: high education. * p < .05, ** p <.01, *** p < .001

Table 7.2. Indirect effects (95% CI) of the association between low maternal education and BMI and FMI

	BMI		FMI	
Potential mediators	Indirect effects (95% CI)	Percentage mediated	Indirect effects (95% CI)	Percentage mediated
Smoking during pregnancy	0.24 (0.10 – 0.40)	26%	0.05 (-0.03 – 0.12)	
Maternal BMI	0.18 (0.10 – 0.29)	20%	0.08 (0.04 – 0.13)	31%
Maternal age	0.03 (-0.01 – 0.08)		0.02 (0.00 – 0.05)	
Weight gain 0-3 mo	0.12 (0.04 – 0.23)	13%	0.04 (0.01 – 0.08)	15%
Weight gain 3-6 mo	0.00 (-0.05 – 0.05)		0.00 (-0.02 – 0.02)	
Weight gain 6-12 mo	-0.04 (-0.08 – 0.01)		-0.01 (-0.03 – 0.00)	
Breastfeeding à weight gain 0-3 mo	-0.02 (-0.03 – 0.00)		0.00 (-0.01 – 0.00)	
Breastfeeding à weight gain 3-6 mo	0.04 (0.02 – 0.06)	4%	0.02 (0.01 – 0.03)	8%
Breastfeeding à weight gain 6-12 mo	0.02 (0.01 – 0.04)	2%	0.01 (0.00 – 0.01)	4%
Age at introduction of solids	0.03 (0.00 – 0.06)		0.02 (0.00 – 0.05)	
Total indirect effect*	0.71 (0.50 – 0.96)	76%	0.25 (0.17 – 0.36)	92%
Direct effect	-0.20 (-0.50 – 0.10)		0.02 (-0.14 – 0.21)	
Total effect**	0.93 (0.60 – 1.40)		0.26 (0.21 – 0.48)	

Indirect effects are determined with path analysis
*Total indirect effect= sum of abs(indirect effects)
**Total effect=Total indirect effect + abs(direct effect)

acting through shorter breastfeeding duration in the low-educated group compared to the high-educated group.

The results regarding FMI are comparable to BMI with a mean FMI of 1.93 kg/m^5 (SD 0.67). The low-educated group (β 0.22; 95% CI 0.09 – 0.34), as well as the mid-educated group (β 0.11; 95% CI 0.03 – 0.19) had a higher FMI in comparison to the high-educated group. An increasing maternal prepregnancy BMI, decreasing maternal age, increasing birth weight and infant weight gain are associated with an increasing FMI at age five-six. The association between low educational level and childhood FMI could be explained by maternal BMI (31%), weight gain 0-3 months (15%), the breastfeeding-weight gain 3-6 months path (8%) and the breastfeeding-weight gain 6-12 months path (4%) (Table 7.2).

Children with inadequate family income had a 0.45 kg/m^2 (95% CI; 0.21 – 0.69) higher BMI and a 0.26 kg/m^5 (95% CI; 0.14 – 0.38) higher FMI compared to children from lot more than adequate income families. Income adequacy was not associated with potential mediators other than depicted in figure 7.2. The higher BMI and FMI of children from inadequate income families compared with children from a lot more than adequate income families can be explained by maternal BMI (~30%) only.

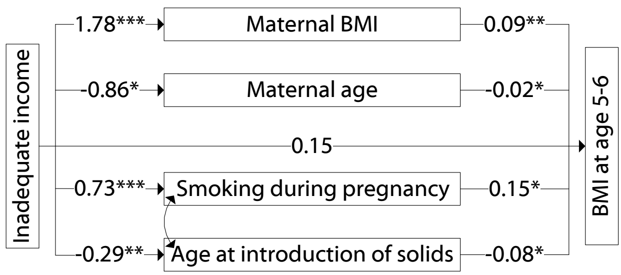

Figure 7.2. Graphic display of the estimated path-analysis model with family income adequacy. The regression equations with BMI and the potential mediators as the outcomes are represented by single-headed arrows. Double-headed arrows refer to the correlation between potential mediators. This model is corrected for childhood age and sex. Reference group: lot more than adequate income. * p < .05, ** p <.01, *** p < .001

DISCUSSION

We demonstrated that children with low-educated mothers have a higher BMI and also a higher FMI compared to children with high-educated mothers. This study provides novel evidence for the explanation of this association by maternal smoking during pregnancy, maternal prepregnancy BMI, and higher weight gain in the first year of life. The contribution of first year weight gain acts partly through a shorter duration of breastfeeding in the low-educated group. The association between income adequacy and body composition can partly be explained by maternal prepregnancy BMI.

Many studies have examined socioeconomic inequalities in childhood BMI,[35] but there are no studies that examined the contribution of early (gestational and infant age) life factors to the association of SES to childhood BMI. We showed that the half of this association can be explained by maternal smoking during pregnancy and maternal prepregnancy BMI. These factors can reflect an unhealthier family lifestyle and may also program the foetus in utero for a higher BMI in later life. Smoking during pregnancy may affect the offspring's appetite control,[228] food preferences,[229] and may lead to postnatal catch-up growth augmented by smoking related fetal growth restriction.[230] As the path-analysis model accounts for standardized birth weight and infant growth, the contribution of smoking in the association of maternal education with childhood BMI appears not to operate via catch-up growth. Besides reflecting an unhealthier lifestyle, the contribution of maternal prepregnancy BMI may be the result of a genetic mechanism[231] or may decrease insulin sensitivity altering placental function as well as fetoplacental availability of nutrients.[232] The association between low maternal education and childhood BMI can be further explained by increased weight gain in the first year of life by 15%. Increased weight gain between 3 and 12 months of age operates via breastfeeding duration, but the contribution of weight gain between 0 and 3 months is independent of standardized birth weight and breastfeeding duration. Several studies have linked childhood BMI with breastfeeding in infancy, though the mechanism is not clear. Breastfeeding might have a preventing effect due to its bioactive factors[233] or the on average lower energy density compared to formula feeding. In the first three months of age, breastfeeding duration does not play a role in the relationship between maternal education and childhood BMI. Thus, not only the duration of breastfeeding, but also the content and amount of feeding between educational groups might play a role. However, there is no evidence that the content of breast milk or formula differ between socioeconomic groups. Nevertheless, infant weight gain, maternal smoking during pregnancy and maternal prepregnancy BMI explain the relation between maternal education and childhood BMI to a substantial extent. Future research should make a difference between pre- and postnatal maternal BMI and smoking. There should also be more research into why children from less educated mothers had increased weight gain as this does not operate entirely through breastfeeding duration and age at introduction of solids.

The role of explanatory early life factors in the relation of socioeconomic status to BMI is comparable to the role of these factors regarding FMI, except the influence of maternal smoking during pregnancy. Maternal smoking during pregnancy partly explains the association between maternal education and BMI, but it does not partly explain the association between maternal education and FMI. Similar to earlier research,[234] also in our cohort maternal smoking during pregnancy is associated with both more fat mass and more lean mass. Hence, maternal smoking is less strongly related to FMI compared to BMI which includes fat mass as well as lean mass. Although the long-term consequences of this difference are uncertain, it highlights the importance of studying more refined measures of body composition than BMI alone.

Early life risk factors play a larger role in the association of body composition with educational level than in the association of body composition with income adequacy. Hence, it is likely that socioeconomic differences in early life risk factors are not due to availability of economic and material resources, but rather to knowledge and beliefs.[35]

The current study was conducted as part of a large prospective cohort study. Unfortunately, as in most cohort studies, selection bias is present. The current subgroup tends to be a slightly healthier (i.e. lower offspring's BMI and maternal BMI) and higher SES (i.e. higher educational level) selection of the total cohort. Thus, both the proportion of women in the low-educated group and the average BMI is higher in the total cohort. As it is likely that the educational level within the low-educated group is also higher in our cohort compared to the population, our result might be an underestimation of the actual associations, but we can think of no reason why this influences mediated proportions by risk factors. To minimize confounding bias we excluded non-Dutch mother-child pairs which reduce generalizability. An extension of our research in ethnic minority groups is planned. Furthermore, the BIA-measurements have some limitations as BIA uses an algorithm based on height, whole-body impedance, and hydration constants to predict body fatness. Since BIA has been validated in this age range and appears to be an accurate tool for epidemiologic studies,[46] we do not suppose that the prediction of fat mass systematically differs between educational groups. Finally a methodological limitation has to be mentioned as causal assumptions about the explanatory factors are not verifiable. On the other hand, the identified explanatory factors are based on plausible pathophysiologic mechanisms, which suggest that improving these factors might reduce BMI and FMI in childhood.

CONCLUSIONS

In conclusion, the higher BMI and FMI in children with low socioeconomic status can partly be explained by maternal prepregnancy BMI and first year weight gain that partly acts through a shorter breastfeeding duration in the low socioeconomic groups. Socioeconomic differences in childhood BMI can also be explained by maternal smoking during pregnancy. This indicates that interventions to reduce the socioeconomic gradient in childhood obesity might be too late at age five. Tackling socioeconomic inequalities in overweight should start with programs including prenatal and infant care.