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Explaining Socioeconomic Inequalities in Childhood Blood Pressure and Prehypertension : The ABCD Study

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Explaining Socioeconomic Inequalities in Childhood Blood Pressure and Prehypertension

The ABCD Study

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

Abstract—Much remains to be understood about the socioeconomic inequalities in hypertension that continue to exist. We investigated the association of socioeconomic status with blood pressure and prehypertension in childhood. In a prospective cohort, 3024 five- to six-year–old children had blood pressure measurements and available information on potential explanatory factors, namely birth weight, gestational age, smoking during pregnancy, pregnancy-induced hypertension, familial hypertension, maternal body mass index, breastfeeding duration, domestic tobacco exposure, and body mass index. The systolic and diastolic blood pressures of children from mid-educated women were 1.0-mm Hg higher (95% CI, 0.4–1.7) and 0.9-mm Hg higher (95% CI, 0.3–1.4), and the blood pressures of children with low-educated women were 2.2-mm Hg higher (95% CI, 1.4–3.0) and 1.7-mm Hg higher (95% CI, 1.1–2.4) compared with children with high-educated women. Children with mid- (odds ratio, 1.50; 95% CI, 1.18–1.92) or low-educated mothers (odds ratio, 1.80; 95% CI, 1.35–2.42) were more likely to have prehypertension compared with children with high-educated mothers. Using path analyses, birth weight, breastfeeding duration, and body mass index were determined as having a role in the association of maternal education with offspring blood pressure and prehypertension. The socioeconomic gradient in hypertension appears to emerge from childhood as the results show a higher blood pressure and more prehypertension in children from lower socioeconomic status families. Socioeconomic disparities could be reduced by improving 3 factors in particular, namely birth weight, breastfeeding duration, and body mass index, but other factors might also play a role. (***Hypertension*****. 2013;61:35-41.)** ● **Online Data Supplement**

Key Words: socioeconomic status ■ blood pressure ■ hypertension ■ obesity ■ breastfeeding ■ birth weight

Hypertension has convincingly been established to begin in childhood. Children with elevated blood pressure have structural and functional cardiovascular changes, and are more likely to have hypertension in adulthood,[1] so hypertension in childhood contributes to the early development of cardiovascular disease.[2,3] Therefore, identifying children with elevated blood pressure and hypertension and early treatment or prevention of hypertension may have an important impact on long-term outcome of cardiovascular disease.

Some recent studies have identified socioeconomic disadvantaged adults as being at risk of elevated blood pressure.[4–6] However, it is unclear whether these social inequalities may already exist in childhood. Available studies in childhood have suboptimal designs,[7,8] produced inconsistent results,[9,10] and have included adolescents as well.[11] In an Australian cohort, low maternal education was identified as a risk factor for elevated blood pressure at age 5 years, but no attention was devoted to the explanatory factors.[12] Identifying the mechanisms underlying the association of socioeconomic status (SES) with blood pressure is useful for public health action to forestall the ongoing social patterning of hypertension in later life.

The literature on the modifiable factors of childhood hypertension has identified many variables, such as low birth weight,[13] smoking during pregnancy,[12] breastfeeding duration,[12,14] maternal body mass index (BMI),[12] and childhood obesity,[15,16] as predictors of elevated blood pressure. To establish the role of these factors in explaining the socioeconomic inequalities relative to blood pressure we used a birth cohort study, with extensive data on blood pressure and other relevant characteristics at ages 5 to 6 years.

The aims of study were to assess whether socioeconomic characteristics influence blood pressure and prehypertension, and to investigate to which extent these potential relations are explained by acknowledged risk factors of cardiovascular disease.

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Methods

Study Design

This study was nested in the Amsterdam Born Children and their Development (ABCD) study, a prospective cohort study from fetal life onward. Details of this study were described previously.[17] This study was approved by the institutional review committee of the Academic Medical Center, and the Registration Committee of Amsterdam. All of the participants gave written informed consent for themselves and their children. The present study was conducted according to the guidelines laid down in the Declaration of Helsinki.

Study Population

From 2003 to 2004, 12 373 Amsterdam women who first attended antenatal care were approached to participate. A total of 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 5, 6161 mothers received a questionnaire, including an informed consent sheet for a health check of their child. Attrition in follow-up number was largely attributable to untraceable changes in address or migration. A total of 4488 questionnaires were returned (response 73%) and 4158 gave permission for the health check. The health check itself consisted of various health measurements in 3321 children aged 5 to 6 years.[18] Measurements took place only in Amsterdam; therefore children who lived outside the measurement area were not included.

The present study excluded mother-child pairs of which the mother had not filled out their educational level (n=68) or income adequacy (n=43) in the childhood questionnaire and of which the blood pressure measurement was not available (n=111). Our analyses also leave out participants with missing data on covariates (breastfeeding duration, n=41; birth weight, n=16; ethnicity, n=2) and participants with diseases that might influence blood pressure (ie, congenital cardiac abnormalities [n=12], kidney failure [n=1], Beckwith Wiedeman syndrome [n=2], and hypertension treated with antihypertensive therapy [n=1]). Finally, 3024 mother-child pairs with completed data were included in the analyses.

Socioeconomic Status

SES was indexed using maternal education, because education level is often seen as the most consistent SES predictor of cardiovascular disease and its risk factors.[19] 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); middle (higher vocational secondary education, intermediate vocational education); or high (higher vocational education, university education). In the questionnaire there were 4 categories, but only 96 women reported no education or primary school; therefore, this category was combined with lower vocational secondary education or technical secondary education.

Analyses were repeated with family income adequacy as an 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 4 categories: (1) inadequate—scored if the mother filled out either overdraft or in debt or using up my savings; (2) adequate—scored if the mother filled out can just make ends meet; (3) bit more than adequate—scored if can make ends meet and a bit more was filled out; and (4) a lot more than adequate—scored if can make ends meet and a lot more was filled out.

Blood Pressure

Blood pressure was measured twice in the supine position after 5 minutes of rest with the Omron 705 IT (Omron Healthcare Inc, Cannockburn, IL) with its small cuff (arm circumference, 17–22 cm), that is validated in children.[20] If these measurements differed by >10 mm Hg (systolic blood pressure [SBP] or diastolic blood pressure [DBP]) a third measurement was taken. The SBP and DBP were determined by averaging the 2 closest measurements per child. Prehypertension (SBP or DBP >90th percentile or both) was based on sex-, age-, and height-specific percentiles.[21]

Covariates

Literature on the determinants of hypertension in childhood was used to select potential confounders and mediators for the association of SES with blood pressure and prehypertension (categories are in parentheses). Potential confounders were ethnicity, based on the country of birth of the pregnant woman her mother (Dutch native, African descent, Turkish, Moroccan, or other),[16,22] childhood height (continuous),[12] childhood age (continuous),[12] and sex.[21] Potential mediators were birth weight (continuous),[23] gestational age (continuous),[24] smoking during pregnancy (no or yes),[12] pregnancy-induced hypertension (no or yes),[25] familial hypertension (no or yes),[26,27] maternal BMI (continuous),[12] breastfeeding duration (none, <1 month, 1–3 months, and >3 months),[14] domestic tobacco exposure (no or yes),[26] and childhood BMI (in kilograms per meter squared continuous).[16,28,29] Birth weight and gestational age were obtained from the Youth Health Care Registration and the Dutch Perinatal Registration. Smoking habits and familial hypertension (yes if father, mother, or the family of father or mother had hypertension) were self-reported in the childhood questionnaire. Pregnancy-induced hypertension was the case if pregnancy-related hypertension, eclampsia, or preeclampsia was recorded in the perinatal registration or if women without pre-existent hypertension reported high blood pressure or the use of medicines against high blood pressure during pregnancy or both. Self-reported prepregnancy weight and height were used to calculate maternal BMI. Information on duration of breastfeeding was available from the infancy questionnaire received when the child was 3 months, and from the Youth Health Care Registration. This prospectively collected information was combined with retrospective information of the 5-year questionnaire to complete the data (19.9% of completed data was from 5-year questionnaire). To calculate BMI, height was measured to the nearest millimeter using a Leicester height measure (Seca), and weight to the nearest 100 g using a Marsden weighing scale, model MS-4102.

Statistical Analyses

Univariate associations of SES to all covariates and blood pressure were explored using χ^2 tests and ANOVAs. To explore the associations of covariates with blood pressure and prehypertension, linear and logistic regression analyses were performed. Path analysis mediation models were used to identify potential determinants of childhood hypertension that may explain the association of blood pressure and prehypertension with SES. Each path model consists of the following regression equations: a regression equation that describes the relationship between blood pressure/prehypertension and SES (adjusted for all the mediators), and the regression equations describing the relationship between each mediator and SES.[30] All of the regression equations involved in the path model were adjusted for ethnicity, sex, age, height, and income adequacy. A linear regression model was estimated for blood pressure, and a logistic regression model was estimated for prehypertension. Associations between SES and the mediating risk factors were modeled with the maximum likelihood algorithm implemented in M-PLUS.[31] A linear regression model was used for the continuous mediators, and a logistic regression model was used for dichotomous mediators. A logistic link function was used instead of a probit link function because this model achieves better results when the categories of the dependent variables are rare, which was the case in our study.[31] Breastfeeding duration, familial hypertension, smoking during pregnancy, and domestic tobacco exposure were replaced by underlying continuous latent variables in the regression of the outcomes on the categorical mediators. The indirect effects of the mediating risk factors were determined by calculating the product of the coefficients along a path. The indirect effects on prehypertension were based on the standardized coefficients, as recommended for binary outcomes.[30] The proportion of the relationship between blood pressure and SES mediated by each of the mediators was determined by dividing

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Table. Sample Characteristics by Maternal Education

		Maternal Education			
Confounders/Potential Mediators	Total	High	Mid	Low	*P* Value*
N	3024	2008	614	402	
Age (SD), y	5.7 (0.5)	5.7 (0.5)	5.8 (0.5)	5.9 (0.5)	<0.001
Sex (% boys)	50.3	50.3	48.7	52.2	NS
Height (SD), cm	116.7 (5.7)	116.4 (5.5)	117.1 (5.9)	118.0 (6.2)	<0.001
Ethnicity, %					<0.001
Dutch	63.3	74.0	51.6	27.9	
African descent	8.3	3.9	16.0	18.4	
Turkish	3.0	0.7	4.2	12.9	
Moroccan	5.1	1.2	8.1	19.9	
Others	20.2	20.1	20.0	20.9	
Birth weight (SD), kg	3.48 (0.54)	3.52 (0.51)	3.43 (0.56)	3.32 (0.60)	<0.001
Gestational age (SD), wk	39.87 (1.67)	39.98 (1.59)	39.82 (1.66)	39.40 (1.96)	<0.001
Smoking during pregnancy (% yes)	6.1	2.8	10.6	15.4	<0.001
Pregnancy-induced hypertension (% yes)	8.5	8.8	8.6	7.0	NS
Familial hypertension (% yes)	7.3	5.4	11.4	10.2	<0.001
Maternal prepregnancy BMI	22.9 (3.8)	22.3 (3.2)	23.8 (4.4)	24.6 (4.8)	<0.001
Breastfeeding duration, %					<0.001
None	17.6	14.4	23.1	24.6	
<1 mo	7.2	5.7	9.3	11.7	
1–3 mo	26.6	24.6	30.8	30.1	
>3 mo	48.6	55.3	36.8	33.6	
Domestic tobacco exposure (% yes)	8.0	4.9	12.4	17.4	<0.001
Childhood BMI (SD), kg/m^2	15.5 (1.5)	15.3 (1.2)	15.6 (1.7)	16.1 (2.0)	<0.001
Outcome					
Systolic blood pressure (SD), mm Hg	99.4 (7.3)	98.7 (6.8)	100.0 (7.6)	101.8 (8.3)	<0.001
Linear regression coefficient (95% CI)†		Reference	0.95 (0.28–1.61)	2.20 (1.33–3.07)	
Diastolic blood pressure (SD), mm Hg	57.1 (6.0)	56.5 (5.7)	57.9 (6.5)	59.2 (6.4)	<0.001
Linear regression coefficient (95% CI)†		Reference	0.82 (0.27–1.38)	1.72 (1.00–2.44)	
Prehypertension, %	16.5	13.2	20.8	26.6	<0.001
Odds ratio (95% CI)‡		Reference	1.50 (1.18–1.92)	1.80 (1.35–2.42)	

BMI indicates body mass index; NS, nonsignificant.

**P* value was based on ANOVA for continuous variables and χ^2 test for categorical variables.

†Linear regression analyses were adjusted for income adequacy (categorical), age (continuous), sex, height (continuous), and ethnicity (categorical).

‡Logistic regression analysis was adjusted for ethnicity (categorical).

each of the corresponding indirect effects by the total effect.[32] The assumptions required to test mediation hypotheses were met, although we cannot assert that associations were not confounded.[4] Maternal education was included as categorical variable with high education as reference group. SPSS 15.0 (SPSS Inc, Chicago, IL) was used for the univariate analyses and M-PLUS (Muthen and Muthen) was used for the path analysis mediation models. A $P<0.05$ was considered as significant.

Results

The children's mean age was 5.7 (SD, 0.5). Most children had a Dutch ethnicity (63%), and these children had on average a higher SES compared with children of ethnic minorities. All of the potential confounders in the Table, except sex, were associated with maternal education. In addition, all of the potential mediators, except pregnancy-induced hypertension, were associated with maternal education, to the detriment of the low-educated group.

Overall, mean SBP was 99.4 (SD, 7.3) and mean DBP was 57.1 (SD, 6.0). More than 16% of the children were indexed as prehypertensive. Multivariable linear regression analyses showed that children of mid- and low-educated mothers had a higher blood pressure compared with children of high-educated mothers. Although 13.2% of children with high-educated mothers were considered as prehypertensive, 20.8% and 26.6% of the children with mid- and low-educated mothers were prehypertensive, respectively (Table). For the relations of other covariates to SBP, DBP,

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and prehypertension, see Table S1 in the online-only Data Supplement.

The path analysis model was used to explain the associations of maternal education with blood pressure and prehypertension (the SBP model is depicted in Figure 1). The left part of Figure 1 shows that a low education was associated with lower birth weight, shorter gestation, higher maternal and childhood BMI, shorter breastfeeding duration, and higher odds of being exposed to tobacco during pregnancy and at home. The right part of Figure 1 shows that SBP increased with decreasing child's birth weight. In addition, a higher SBP was observed among individuals with familial hypertension and among those with a higher childhood BMI, maternal BMI, and shorter breastfeeding duration.

The indirect effects of low education on SBP through mediating risk factors involved birth weight, maternal BMI, breastfeeding duration, and childhood BMI (Figure 2). For example, the indirect effect of birth weight on the association between low maternal education and SBP was 0.28 (95% CI, 0.13–0.48), which is the product of the regression equations along that path (−0.17×−1.66). The indirect effects of education level on DBP were comparable to the effects on SBP. However, longer gestation increased (rather than decreased) DBP, and tended therefore to mask rather than explain the association between maternal education and DBP. Domestic tobacco exposure played also a role in explaining the association between DBP and maternal education. Birth weight, breastfeeding duration, and childhood BMI explained the association between maternal education and prehypertension (Figure 2). The explanatory factors in the association of mid-education with blood pressure and prehypertension were comparable to the association of low education with blood pressure and prehypertension, but the effects are smaller. After adjustment for mediators, the associations of maternal education with SBP, DBP, and hypertension disappeared.

Regarding the proportion of explanation by each of the risk factors (Figure 2), childhood BMI was the most important contributor to the association of maternal education to blood pressure, and prehypertension, with indirect effects representing 19% to 25%. Secondly, birth weight contributed to the association between maternal education and blood pressure accounting for 13% to 17%. Breastfeeding duration was the third important contributor to the education–blood pressure association, representing 9% to 20%. In addition, there was a small indirect effect of maternal BMI on the association between maternal education and SBP/DBP and an indirect effect of domestic tobacco exposure on the association between maternal education and DBP. Overall, 61% of the association of low maternal education with SBP and DBP was explained by included mediators.

Replicating the Analyses Using Income Adequacy as an Indicator of SES

Because income adequacy may operate in a qualitatively different way in determining blood pressure compared with maternal education, the analyses were repeated using income adequacy as an indicator of SES. Income adequacy was associated with all of the potential mediators, except gestational age and pregnancy-induced hypertension (Table S2). Children from families with inadequate financial situation had on

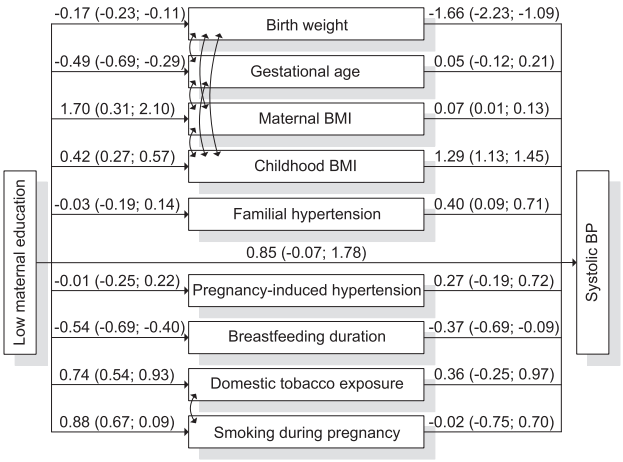

Figure 1. Graphic display of the estimated path analysis model. The 9 regression equations, with systolic blood pressure (**right part**) and each of the potential mediators as the outcomes (**left part**), are represented by single-headed arrows. Double-headed arrows refer to the correlation between potential mediators. The product of the coefficients reflects the weight of the path with the 95% CIs in parentheses. The path analysis model is corrected for height, age, sex, income adequacy, and ethnicity. Total effect was 1.35 (95% CI, 0.68–1.95), total indirect effect 1.04 (0.58–1.51). BMI indicates body mass index; BP, blood pressure.

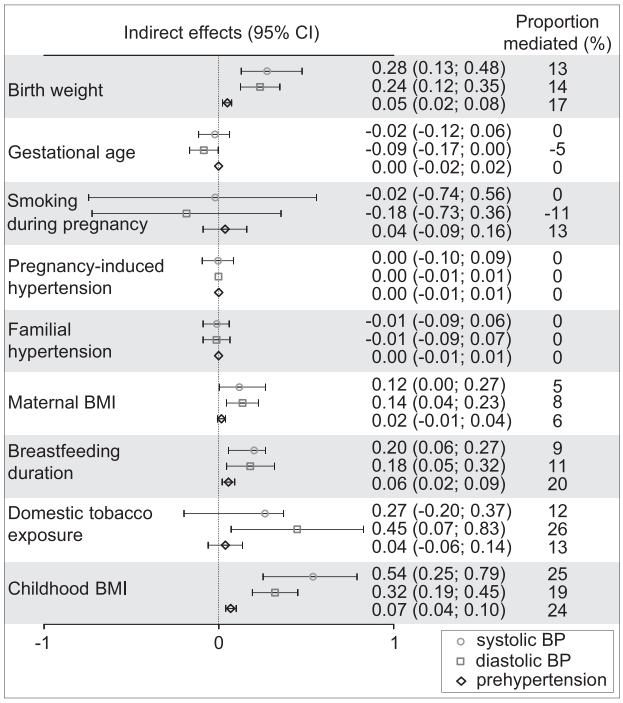

Figure 2. Indirect effects of mediators on the associations of low maternal education with systolic blood pressure (SBP; 95% CI), diastolic blood pressure (DBP; 95% CI), and prehypertension (95% CI). The **right column** shows the proportion mediated by mediators. The SBP model and DBP model were corrected for income adequacy (categorical), age (continuous), sex, height (continuous), and ethnicity (categorical). The prehypertension model was corrected for income adequacy (categorical) and ethnicity (categorical). BMI indicates body mass index.

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average a higher blood pressure compared with children from families that had a lot more than adequate (99.9 versus 98.7 mm Hg). However, after adjustment for height, age, sex, and ethnicity, the association was no longer significant. In addition, these children from inadequate income families were more likely to have prehypertension compared with children from families that had a lot more than adequate (odds ratio, 1.37; 95% CI, 1.03–1.84). Indirect effects of income adequacy on blood pressure involved maternal and childhood BMI for SBP, maternal BMI, childhood BMI, breastfeeding duration, and domestic tobacco exposure for DBP, and breastfeeding duration and childhood BMI for prehypertension (Table S3).

Discussion

We demonstrated that the socioeconomic gradient in blood pressure and hypertension originate in childhood. Children from lower SES families had on average a higher blood pressure and were more likely to have prehypertension, which is highly predictive for hypertension in later life.[33] This study provides novel evidence for the explanatory role of higher BMI, shorter breastfeeding duration, and lower birth weight in these relationships.

Few studies have examined socioeconomic inequalities in childhood blood pressure. At the univariate level, our findings agree with previous literature demonstrating a higher blood pressure in children of lower educated parents.[12,26,34] We are, however, not aware of any study that addressed the potential explanatory factors in this relationship. Studies in adulthood showed a stronger association of SES to blood pressure,[4,6] and in these studies biobehavioral factors like BMI, current smoking, and physical activity are indicated as having a part in blood pressure differences.

Underlying Mechanisms

Three possible explanations for the SES difference in blood pressure and prehypertension were indicated in particular, namely BMI, breastfeeding duration, and birth weight. The relation between elevated blood pressure and an increasing BMI has been demonstrated extensively[15,16,35] and has been shown to begin in early childhood.[28,36] A brief review reported 3 main pathophysiological mechanisms of hypertension attributable to obesity in childhood, disturbances in autonomic function, insulin resistance, and abnormalities in vascular structure and function.[29] Childhood BMI was showed previously to be strongly related to SES,[37] and we showed that socioeconomic differences in blood pressure and prehypertension are largely explained by an increasing BMI. Other than BMI, socioeconomic disparities in prehypertension are attributable to a lower birth weight and a shorter duration of breastfeeding. Numerous studies have demonstrated that lower birth weight is associated with higher blood pressure in children,[36,38] and evidence suggests that low birth weight affects blood pressure because of a decreased number of nephrons that results in compensatory hypertrophy and intraglomerular hypertension.[39] Despite this evidence regarding birth weight, the rapid postnatal growth in low-birth weight infants may contribute to a greater extent.[36,40] Because of its lower caloric content and lower initial (demand) volume, breastfeeding usually results in slower early growth,[14] and might be beneficial to later blood pressure, whereas formula feeding is usually fixed volume and more energy dense, which may increase the risk of later obesity, insulin resistance, and endothelial dysfunction.[14] The low sodium content and high long-chain polyunsaturated acids content in breast milk might play a role in these programming effects.[41] Although it was demonstrated earlier that birth weight and breastfeeding affect blood pressure, we showed that these factors also play a role in the association of SES with blood pressure and prehypertension in childhood.

Socioeconomic disparities in blood pressure and prehypertension can be partly explained by aforementioned variables, but there is a small indirect effect of maternal BMI on the education-SBP, income-SBP, and income-DBP associations additionally. Furthermore, domestic tobacco exposure plays a role in the association of DBP with maternal education and income adequacy. Therefore, maternal BMI and domestic tobacco exposure might partly explain the SES gradient in blood pressure and prehypertension. Because we found no evidence for the explanatory role of many other factors, it is unclear which factors may play a role in the relationship between SES and blood pressure additionally. Because salt intake was associated with both hypertension and SES,[42] this factor may explain socioeconomic disparities in childhood blood pressure. Therefore, more research is needed regarding the influence of salt intake on childhood blood pressure, especially in socioeconomic disadvantaged children.

Socioeconomic Status

This study used the mother's educational level and family's income adequacy as indicators of SES. Education reflects knowledge and beliefs, whereas income adequacy is likely to reflect the availability of economic and material resources. In addition, income adequacy is a subjective indicator of SES, so an adequate income could interpret differential by participants and might be less stable over time than maternal education. This might be the reason that birth weight does not explain the association between blood pressure and income adequacy, whereas it partly explains the association between blood pressure and education. In addition, maternal occupational level and neighborhood income were explored as indicators of SES in a subsample, but had no effect on blood pressure (results not shown).

Limitations

The current study was conducted in a large prospective cohort study. Unfortunately, selective loss to follow-up is present, as in most cohort studies. The current subgroup tends to be a slightly healthier (ie, lower maternal BMI) and higher SES reflection (ie, higher educational level) of the population. Thus, both the proportion of women in the lowest SES group and the average blood pressure might be higher at the population level. This may result in an underestimation of the actual associations, but we can think of no reason why this influences the magnitude mediated by risk factors. Prehypertension was classified on widely used references that were based on sitting blood pressure recorded by auscultation. However, we assessed blood pressure in supine position with an automatic oscillometric device because of their ease of use and decrease in observer bias. As compared with auscultated blood

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pressure measurements, mean SBP readings with our device were 4.6±4.9-mm Hg higher, and mean DBP readings were 3.3±5.4-mm Hg lower.[20] Because systolic hypertension in children is far more common than diastolic hypertension, the prevalence of prehypertension might be overestimated, but this will probably not affect the investigated association. Finally, a statistical limitation has to be mentioned. Some categorical variables have a low proportion of people answering one of the categories and the estimated SEs are large, so the indirect effects may therefore not reach significance. Moreover, causal assumptions about the explanatory factors are not verifiable. On the other hand, the identified explanatory factors are based on plausible pathophysiological mechanisms, which suggest that improving these factors might reduce blood pressure and prehypertension in childhood.

Perspectives

Given the elevated blood pressure and ~2-fold higher risk of prehypertension among children from low SES families and the associated consequences in later life and track into adulthood, it is important to invest in policies aimed at reducing socioeconomic inequalities in blood pressure and prehypertension. This elevated risk could partly be explained by birth weight, breastfeeding duration, and BMI and is therefore modifiable by interventions. Initiation of preventive interventions at age 5 years may be too late. The most effective strategy for tackling socioeconomic inequalities in cardiovascular disease risk of future generations is probably by nesting these programs in prenatal care.

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Disclosures

None.

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Novelty and Significance

What Is New?

- Extensive study on the relation of socioeconomic status to blood pressure and prehypertension at age 5 to 6 years.
- Special attention to explanatory potentially modifiable early life factors in this relationship.

What Is Relevant?

- The socioeconomic gradient in blood pressure and hypertension originate in early childhood.
- Childhood body mass index, breastfeeding duration, and birth weight are important mediators in the relation of socioeconomic status to blood pressure and prehypertension.

Summary

Interventions that improve body mass index, breastfeeding duration, and birth weight have the potential to reduce the socioeconomic gradient in blood pressure and prehypertension at age 5 to 6 years and related cardiovascular disease in later life.

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Explaining Socioeconomic Inequalities in Childhood Blood Pressure and prehypertension. The ABCD-Study

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S1. Associations of blood pressure and prehypertension with determinants

Determinants	Beta-coefficients (95% CI) for SBP and determinants adjusted for all presented determinants	Beta-coefficients (95% CI) for DBP and determinants adjusted for all presented determinants	Odds ratio (95% CI) for prehypertension and determinants adjusted for all presented determinants
Maternal education			
High	Reference	Reference	Reference
Mid	0.44 (-0.22 – 1.09)	0.43 (-0.13 – 0.98)	1.18 (0.85 – 1.64)
Low	1.13 (0.27 – 1.99)	0.95 (0.22 – 1.68)	1.18 (0.90 – 1.53)
Income adequacy			
Lot more	Reference	Reference	Reference
Bit more	0.13 (-0.75 – 1.00)	0.30 (-0.24 – 0.83)	1.37 (1.03 – 1.84)
adequate	0.11 (-0.63 – 0.84)	0.08 (-0.54 – 0.70)	1.44 (1.04 – 1.99)
Inadequate	0.34 (-0.29 – 0.97)	0.10 (-0.65 – 0.84)	1.35 (0.93 – 1.97)
Potential confounders			
Age yr	-0.27 (-0.88 – 0.34)	0.04 (-0.48 – 0.56)	
Sex			
Boys	Reference	Reference	
Girls	0.13 (-0.36 – 0.61)	1.85 (1.44 – 2.26)	
Height cm	0.31 (0.26 – 0.37)	0.09 (0.04 – 0.13)	
Ethnicity			
Dutch	Reference	Reference	Reference
African descent	-0.59 (-1.53 – 0.36)	1.79 (0.99 – 2.60)	1.32 (0.93 – 1.88)
Turkish	-0.17 (-1.67 – 1.32)	0.98 (-0.29 – 2.25)	2.09 (1.26 – 3.46)
Moroccan	-0.25 (-1.45 – 0.95)	0.74 (-0.28 – 1.76)	0.97 (0.62 – 1.53)
Others	-0.04 (-0.67 – 0.58)	0.22 (-0.31 – 0.75)	1.14 (0.87 – 1.47)
Potential mediators			
Birth weight	-1.60 (-2.17 – -1.02)	-1.41 (-1.90 – -0.92)	0.61 (0.49 – 0.78)
Gestational age	0.05 (-0.13 – 0.23)	0.18 (0.03 – 0.33)	1.00 (0.93 – 1.07)
Smoking during pregnancy			
No	Reference	Reference	Reference
Yes	-0.41 (-1.48 – 0.66)	-0.61 (-1.51 – 0.30)	0.97 (0.65 – 1.45)
Pregnancy-induced hypertension			
No	Reference	Reference	Reference
Yes	0.07 (-0.79 – 0.93)	-0.24 (-0.97 – 0.49)	0.96 (0.67 – 1.38)
Familial hypertension			
No	Reference	Reference	Reference
Yes	1.13 (0.20 – 2.06)	0.31 (-0.48 – 1.10)	1.20 (0.84 – 1.70)
Maternal prepregnancy BMI	0.06 (-0.01 – 0.13)	0.08 (0.02 – 0.13)	1.01 (0.99 – 1.04)
Breastfeeding duration			
> 3 months	Reference	Reference	Reference
1-3 months	0.47 (-0.11 – 1.06)	0.55 (0.05 – 1.04)	1.40 (1.10 – 1.79)
< 1 months	0.87 (-0.10 – 1.83)	1.19 (0.37 – 2.00)	1.81 (1.27 – 2.59)
None	0.48 (-0.20 – 1.17)	0.43 (-0.15 – 1.01)	1.27 (0.96 – 1.69)
Domestic tobacco exposure			
No	Reference	Reference	Reference
Yes	0.51 (-0.42 – 1.43)	0.88 (0.09 – 1.67	1.15 (0.80 – 1.63)
BMI	1.28 (1.11 – 1.46)	0.76 (0.61 – 0.91)	1.32 (1.24 – 1.42)

Values are based on linear regression analyses for SBP and DBP, and on logistic regression analyses for prehypertension

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S2. Sample characteristics by income adequacy

Characteristics	Income adequacy				
	Lot more	Bit more	Adequate	Inadequate	p-value *
n	721	1179	731	393	
Age yr (sd)	5.7 (0.5)	5.7 (0.5)	5.8 (0.5)	5.8 (0.5)	<.001
Sex (% boys)	50.1	49.8	51.0	51.4	Ns
Height, cm (sd)	116.9 (5.6)	116.6 (5.6)	116.8 (5.9)	116.7 (6.0)	Ns
Ethnicity (%)					<.001
Dutch	63.1	74.0	51.6	27.2	
African descent	8.4	3.9	16.1	18.4	
Turkish	3.1	0.7	4.2	12.9	
Moroccan	5.3	1.2	8.2	20.8	
Others	20.2	20.1	19.9	20.8	
Birth weight, kg (sd)	3.52 (0.54)	3.48 (0.52)	3.45 (0.57)	3.43 (0.55)	.04
Gestational age, weeks (sd)	39.94 (1.74)	39.92 (1.59)	39.83 (1.70)	39.69 (1.67)	Ns
Smoking during pregnancy (% yes)	2.2	5.0	9.4	9.9	<.001
Pregnancy-induced hypertension (% yes)	9.0	8.7	7.4	9.2	Ns
Familial hypertension (% yes)	4.9	6.3	9.4	10.7	<.001
Maternal prepregnancy BMI	22.3 (3.1)	22.7 (3.5)	23.4 (4.1)	24.0 (4.6)	<.001
Breastfeeding duration (%)					<.001
None	14.4	18.2	18.3	20.1	
< 1 month	5.7	6.1	10.3	7.92	
1-3 months	26.2	25.8	26.1	30.3	
> 3 months	53.7	50.0	45.3	41.7	
Domestic tobacco exposure (% yes)	4.3	7.9	8.3	14.5	<.001
Childhood BMI (kg/m^2) (sd)	15.3 (1.3)	15.4 (1.3)	15.7 (1.7)	15.7 (1.7)	<.001
Outcome					
Systolic blood pressure mmHg (sd)	98.7 (6.6)	99.3 (7.2)	99.8 (7.8)	99.9 (7.8)	.01
Diastolic blood pressure mmHg (sd)	56.4 (5.8)	57.1 (6.0)	57.6 (6.2)	57.8 (6.1)	<.001
Prehypertension (%)	11.1	15.9	20.8	20.4	<.001
SES					
Maternal education					<.001
High	88.3	71.7	48.4	43.8	
Mid	10.4	20.1	26.7	27.2	
Low	1.2	8.2	24.0	29.0	

* P-value based on ANOVAs for continuous variables and Chi-square test for categorical variables.
Ns indicates non significant.

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S3. Indirect effects (95% CI) of the association between inadequate income and SBP, DBP, and prehypertension.

Potential mediating risk factors	Indirect effect (95% CI) of the association between inadequate income and SBP attributable to each mediator	Indirect effect (95% CI) of the association between inadequate income and DBP attributable to each mediator	Indirect effect (95% CI) of the association between inadequate income and prehypertension attributable to each mediator
Birth weight	0.00 (-0.11 – 0.11)	0.00 (-0.11 – 0.10)	0.00 (-0.02 – 0.02)
Gestational age	-0.01 (-0.03 – 0.02)	-0.02 (-0.07 – 0.03)	0.00 (0.00 – 0.00)
Smoking during pregnancy	-0.07 (-0.63 – 0.50)	-0.23 (-0.73 – 0.27)	0.00 (-0.01 – 0.01)
Pregnancy-induced hypertension	0.02 (-0.05 – 0.08)	0.00 (-0.02 – 0.03)	0.02 (-0.10 – 0.13)
Familial hypertension	0.03 (-0.04 – 0.10)	0.04 (-0.04 – 0.12)	0.00 (-0.01 – 0.01)
Maternal prepregnancy BMI	0.11 (0.02 – 0.20)	0.12 (0.04 – 0.20)	0.02 (-0.00 – 0.03)
Breastfeeding duration	0.08 (-0.00 – 0.166)	0.08 (0.00 – 0.15)	0.02 (0.00 – 0.04)
Domestic tobacco exposure	0.36 (-0.08 – 0.81)	0.49 (0.10 – 0.89)	0.05 (-0.04 – 0.15)
Childhood BMI	0.29 (0.06 – 0.52)	18.7 (14.3 – 23.1)	0.04 (0.01 – 0.07)
Total effect	0.90 (0.00 – 1.80)	0.70 (-0.07 – 1.46)	0.26 (0.07 – 0.46)
Total indirect effect	0.82 (0.35 – 1.30)	0.66 (0.24 – 1.08)	0.15 (0.05 – 0.24)

Indirect effects were determined with path-analysis corrected for height, age, sex, and ethnicity

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