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Early Socioeconomic Position and Blood Pressure in Childhood and Adulthood

The Cardiovascular Risk in Young Finns Study

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Abstract—Studies have found an association between low socioeconomic position in childhood and high adult blood pressure. It is unclear whether this association is explained by a pathway directly linking disadvantage to elevated blood pressure in childhood and adolescence, which then tracks into adulthood. We assessed parental socioeconomic position and systolic blood pressure in 1807 children and adolescents ages 3 to 18 years at baseline. Adult systolic blood pressure was measured 21 years later at ages 24 to 39 years. There was strong tracking of blood pressure from childhood to adulthood. Lower parental socioeconomic position was associated with higher blood pressure in childhood, adolescence ($P<0.01$), and adulthood ($P<0.0001$), with the mean age- and sex-adjusted systolic pressure differences between the highest and lowest socioeconomic groups varying between 2.9 and 4.3 mm Hg. With adjustment for blood pressure in childhood and adolescence, the regression coefficient between parental socioeconomic position and adult blood pressure attenuated by 32%. A similar level of attenuation (28%) occurred with adjustment for adult body mass index (BMI). With adjustment for both preadult blood pressure and adult BMI, the association between parental socioeconomic position and adult blood pressure was attenuated by 45%. Other factors, including birth weight and BMI in childhood and adolescence, had little impact on the association between parental socioeconomic position and adult blood pressure. These data suggest that early socioeconomic disadvantage influences later blood pressure in part through an effect on blood pressure in early life, which tracks into adulthood, and in part through an effect on BMI. (*Hypertension*. 2006; 47:39-44.)

Key Words: blood pressure ■ socioeconomic factors ■ life style ■ epidemiology

Many,[1–6] although not all,[7] studies have found an association between childhood socioeconomic position (SEP) and adult blood pressure. The mechanisms underlying this association remain unclear. Life course models suggest that early socioeconomic disadvantage could affect future blood pressure through several mechanisms. First, early disadvantage is strongly associated with adult disadvantage,[8] which is, in turn, related to adult behavioral risk factors, such as low levels of physical activity, high dietary sodium intake, and obesity, which adversely affect blood pressure.[9–13] Second, early disadvantage may have a direct effect on these lifestyle factors, which are often learned in childhood or adolescence and persist into adulthood.[14] Third, early disadvantage is related to fetal and childhood growth and development and may have a direct effect on future blood pressure via a programming effect.[15] This direct effect on blood pressure in childhood might then lead to increased adult blood pressure, because blood pressure tracks from childhood to adulthood, with those in the highest end of the distribution in childhood being in the highest end of the distribution in adulthood.[16] These mechanisms are not mutually exclusive.

From a public health policy perspective it is important to establish which mechanism predominates but, in particular, whether the association of early disadvantage with childhood blood pressure mediates its effect on adult blood pressure. If true, this would suggest that policies to reduce inequalities should focus on childhood disadvantage, because an association between childhood disadvantage and high childhood blood pressure that then tracks into adulthood implies a trajectory of increased risk among those from more disadvantaged backgrounds that begins in childhood.

Several studies have examined the association between childhood SEP and childhood blood pressure and reported inconsistent results.[15,17–19] These inconsistencies may be because of variation in sample size; the confounding factors taken into account in each study; and the methods used to

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assess exposure, covariates, and outcomes in each study. To our knowledge, no previous study has examined whether the association between childhood SEP and adult blood pressure is mediated via a direct association between childhood SEP and childhood blood pressure and then tracking into adulthood. Thus, we studied this issue in the Cardiovascular Risk in Young Finns Study. For comparison, we examined whether fetal effects, indicated by birth weight, and body mass index (BMI) in later life would mediate the association between childhood SEP and adult blood pressure.

Methods

Participants

The Cardiovascular Risk in Young Finns Study is an ongoing 5-center follow-up study of atherosclerosis risk factors of Finnish children and adolescents.[20,21] The first cross-sectional survey was conducted in 1980. The original sample size was 4320 children and adolescents at 3, 6, 9, 12, 15, and 18 years of age. The individuals were randomly chosen from the national register. There were 3596 participants (83% of those invited) who participated in the baseline study in 1980 (phase 1). The first follow-up study was conducted in 1983 and is referred to as phase 2. In phase 3 in 2001, the participants, who had then reached 24 to 39 years of age, were reexamined (Table 1). The study was approved by local ethics committees and conducted in accordance with the guidelines of the declaration of Helsinki. All of the participants gave written informed consent.

Measurements

The main components of SEP are income, occupational status, and education. We assessed parental SEP components by parent's household income at phase 1 (1=bottom quartile, 2=2 middle quartiles, 3=top quartile), parental occupational status at phase 1 (1=manual, 2=lower grade nonmanual, 3=higher grade nonmanual), and parental education at phase 2 (1=comprehensive school; 2=secondary education, not academic; 3=academic). For parental occupational status and education, data on the parent with a higher occupational status or higher education were used. In analyses, we used a composite parental SEP measure: sum scores of household income, parental occupational status and parental education (range, 3 to 9). Cronbach's α reliability coefficient for the composite score is $r=0.78$, indicating that the score is internally consistent ($\alpha>0.7$ indicates internal consistency).

Repeated measurements of blood pressure in phases 1 to 3 have been described previously.[4,21–23] In brief, measurements were made in sitting position after 5 minutes rest with a mercury sphygmomanometer at phases 1 and 2 (for the 3-year-olds, with ultrasound device, Arteriosonde 1020, Roche) and with a random zero sphygmomanometer (Hawksley & Sons Ltd) at phase 3. Cuff size for measurement was chosen to cover two thirds of arm length. Korot-

koff's first phase was used as the sign of systolic blood pressure. Readings to the nearest even number of millimeters of mercury were performed ≥3 times on each subject. The average of systolic blood pressure measurements was used in the analysis.

Birth weight (grams) was reported by the mothers. The mothers/participants were also asked to bring with them the booklet from the well-baby center in which the in-birth weights were recorded. Physical measurements of height (millimeters) and weight (kilograms) were obtained to calculate BMI (weight in kilograms/height^2 in meters) at phases 1, 2, and 3. In addition, waist circumference (millimeters, measured in duplicate at the level of the twelfth rib or the level with the navel in thin subjects at phase 3) was measured at phase 3.

Statistical Methods

The current analysis was limited to those participants with full data on parental SEP and blood pressure at all 3 of the phases. We calculated 3-year and 21-year tracking correlations in measurements of blood pressure by using linear regression analysis. The analysis was stratified by age groups.[16,24] We used the regression dilution method to estimate the effect of measurement error on these correlations.[25,26] The regression/dilution ratio was calculated by comparing the first and third blood pressure measurements at phases 1 and 2. We used the inverse of this ratio to estimate the extent that corrected tracking correlations differ from observed tracking correlations.

We used various multivariable linear regression models to examine associations between parental SEP score and blood pressure. Although causal chains, such as mediated effects, cannot be inferred from observational epidemiological data, such data can be used to test whether the observed associations are consistent with what one would expect to see if a causal path from parental SEP→childhood blood pressure→adult blood pressure is true. Operationally, a mediated effect of preadulthood blood pressure is demonstrated if 3 criteria are met.[27] First, one should document socioeconomic differences in blood pressure in childhood or adolescence. Second, parental SEP (exposure) should predict adult blood pressure (outcome), and this association should attenuate after adjustment for blood pressure in childhood and adolescence (possible mediator). Third, the association of blood pressure in childhood and adolescence with adulthood blood pressure should be of similar direction and magnitude in all of the strata of parental SEP. Failure to meet these criteria suggests that the effects on childhood blood pressure do not explain the association between parental SEP and adult blood pressure.

We ran similar models to determine whether birth weight, preadult and adult BMI, and waist circumference also mediated the association between parental SEP and adult blood pressure. In addition to sex and birth year, adjustments were made for place of measurement and, when studying childhood blood pressure, for height. All of the tests were performed with Statistical Analysis System (SAS Institute).

Results

Of the 3596 participants at baseline, 1807 (50%) had full data on systolic blood pressure at all 3 of the phases and information on all of the indicators of parental SEP available. They did not differ from the population at baseline in terms of SEP and age group (discrepancy in any category of the variables was <3%), but women were slightly overrepresented (55% in the study cohort versus 51% in the baseline population). Based on earlier observations, lack of time, absence from the place of residence at the time of examination, and unwillingness to participate were the main reasons for nonparticipation.[21]

Tracking in Blood Pressure Measurements

The 3-year tracking correlation was 0.43 to 0.62 at ages 6, 9, 12, 15, and 18 (all *P* values <0.0001), but only 0.07 at age 3

TABLE 1.   Study Design

Birth Year	No.	Age at Phase 1 (1980)	Age at Phase 2 (1983)	Age at Phase 3 (2001)
1977	270	(3)	6	24
1974	318	6	9	27
1971	352	9	12	30
1968	345	12	15	33
1965	291	15	18	36
1962	231	18	(21)	39

Parental socioeconomic position was measured at phases 1 and 2, and blood pressure and body mass index were measured at all phases. Childhood and adolescent blood pressure related to measurements at ages 6 to 18 and adult blood pressure to a measurement at age 24 to 39.

(P=0.15). Correspondingly, the 21-year tracking correlation varied between 0.41 and 0.56 in all of the age groups (P values <0.0001) except among the 3-year-old participants for whom it was 0.06 (P=0.27). The regression dilution analysis suggested that the tracking correlations may be 10% higher than those that we observed (the regression/dilution ratio was 0.89 at phase 1 and 0.91 at Phase 2).

Early Blood Pressure as a Mediator Between Parental SEP and Adult Blood Pressure

Because of low tracking of blood pressure measurements in the 3-year-olds, the mediator analyses on childhood and adolescent blood pressure were limited to ages 6 to 18. As shown in Table 2, there was an inverse association between parental SEP and blood pressure in childhood and adolescence across the age groups (P for trend in the total sample was 0.009 at phase 1 and P<0.0001 at phase 2), and adjustment for place of measurement had little effect on this association. The average age- and sex-adjusted difference in blood pressure levels between the lowest and highest parental SEP category was 2.9 mm Hg at phase 1 and 3.8 mm Hg at phase 2.

Table 3 shows that there was also an association between parental SEP and blood pressure in adulthood (P<0.0001). The average age- and sex-adjusted difference in blood pressure levels between the lowest and highest parental SEP category was 4.3 mm Hg. The regression coefficient between parental SEP and adult blood pressure attenuated by 15% to 48% after adjustment for blood pressure in childhood and adolescence. This attenuation was greater after adjustment for adolescent blood pressure than childhood blood pressure.

The stratified analysis in Table 4 shows that there was a strong association between blood pressure in childhood and adolescence and blood pressure in adulthood at each of the 7 parental SEP levels. Thus, all of the findings presented in Tables 1 to 3 support the mediated effect status of blood pressure in childhood and adolescence on the association between parental SEP and adult blood pressure.

Birth Weight and BMI as Potential Mediators

We performed the corresponding tests related to a potential mediator effect of birth weight and BMI on the association between parental SEP and adult blood pressure (data not

TABLE 3. Regression Coefficient Between Parental Socioeconomic Position Score and Adult Blood Pressure

Outcome Adjustment	No.	B	(95% CI)	Change in B
Blood pressure at age 24 to 27				
Birth year and sex	588	−0.66	(−1.19 to −0.13)	Reference
+Blood pressure at age 6	588	−0.54	(−1.04 to −0.04)	−18%
Blood pressure at age 27 to 30				
Birth year and sex	670	−0.80	(−1.30 to −0.30)	Reference
+Blood pressure at age 9	670	−0.68	(−1.15 to −0.22)	−15%
Blood pressure at age 30 to 33				
Birth year and sex	697	−0.64	(−1.22 to −0.07)	Reference
+Blood pressure at age 12	697	−0.49	(−1.03 to 0.05)	−23%
Blood pressure at age 33 to 36				
Birth year and sex	636	−0.71	(−1.30 to −0.11)	Reference
+Blood pressure at age 15	636	−0.54	(−1.10 to 0.02)	−24%
Blood pressure at age 36 to 39				
Birth year and sex	522	−0.80	(−1.42 to −0.19)	Reference
+Blood pressure at age 18	522	−0.42	(−1.01 to 0.17)	−48%

All regression models are adjusted for birth year and sex, and some models are additionally adjusted for early blood pressure.

shown). The regression coefficients between parental SEP and adult blood pressure were attenuated <5% by adjustment for birth weight and BMI at ages 3 to 18, suggesting that these factors are unlikely mediators between parental SEP and blood pressure.

In contrast, adjustment for adult BMI reduced the regression coefficient between parental SEP and adult blood pressure by 31% from B=−0.69 (95% CI, −1.02 to −0.35) to B=−0.47 (95% CI, −0.79 to −0.16; an additional adjustment for waist circumference at adulthood had little extra effect). There was a consistent association between adult BMI

TABLE 2. Regression Coefficient* Between Parental Socioeconomic Position Score† and Blood Pressure at Various Ages in Childhood and Adolescence

Age for Measurement of Blood Pressure, y	No.	B	(95% CI)
6	587	−0.42	(−0.80 to −0.04)
9	669	−0.39	(−0.79 to 0.01)
12	695	−0.43	(−0.85 to −0.02)
15	635	−0.47	(−0.95 to 0.00)
18	521	−1.13	(−1.66 to −0.59)

*Regression coefficient (B) and 95% CIs are adjusted for birth year, sex, and height.

†Sum of parental income, parental occupation, and parental education (range, 3 to 9).

TABLE 4. Regression Coefficients for Tracking of Blood Pressure Levels From Ages 6 to 18 to Ages 24 to 39 by Parental Socioeconomic Position

	18-Year Tracking		21-Year Tracking	
Parental SEP Score	No.	B (95% CI)	No.	B (95% CI)
3 (lowest)	223	0.48 (0.35 to 0.61)	245	0.35 (0.22 to 0.48)
4	409	0.40 (0.30 to 0.50)	412	0.45 (0.33 to 0.57)
5	384	0.36 (0.24 to 0.47)	374	0.37 (0.25 to 0.48)
6	212	0.45 (0.30 to 0.61)	188	0.45 (0.29 to 0.61)
7	111	0.38 (0.10 to 0.66)	101	0.34 (0.07 to 0.61)
8	119	0.28 (0.08 to 0.49)	105	0.39 (0.18 to 0.59)
9 (highest)	118	0.54 (0.33 to 0.76)	112	0.39 (0.18 to 0.60)

SEP indicates socioeconomic position.

and adult blood pressure across all of the levels of parental SEP (all age- and sex-adjusted Bs=0.78; 95% CI, 0.21 to 1.34 or higher), and parental SEP predicted adult BMI (age- and sex-adjusted B=−0.23; 95% CI, −0.35 to −0.12). These findings support adult BMI, but not birth weight or preadulthood BMI, as a mediating factor between parental SEP and adult BP.

Combined Mediated Effects on Parental SEP and Adult Blood Pressure

Table 5 presents the effects of blood pressure in childhood and adolescence and BMI in adulthood on the association between parental SEP and adult blood pressure. Simultaneous adjustment for blood pressure in childhood and adolescence and adult BMI attenuated this association by 45%. Early blood pressure and adult BMI appeared to have equally strong effects.

Discussion

In this prospective 21-year follow-up of Finnish children and adolescents, we found strong tracking between blood pressure measured in childhood/adolescence and blood pressure measured 21 years later in adulthood. Furthermore, early socioeconomic disadvantage was associated with higher blood pressure in childhood, adolescence, and early adulthood. With adjustment for socioeconomic differences in blood pressure at ages 6 to 18 years, the association between early disadvantage and adult blood pressure was markedly attenuated, with additional attenuation resulting in additional adjustment for adult BMI. Our findings suggest that early disadvantage influences adult blood pressure not only through its link with adult risk factors, in particular adult BMI, but also by causing long-lasting vascular damage in early life.

Some,[18] but not all,[15] previous studies have failed to observe an association between socioeconomic indicators and blood pressure in childhood and adolescence. We assessed early disadvantage with a composite measure of family income, parent's education, and occupational status, thus simultaneously covering all of the major indicators of SEP.[28] In earlier studies, analyses have usually been based on single socioeconomic indicators, which may explain some of the null results in some previous studies. Furthermore, earlier studies have typically not controlled for height, and it has

been suggested that negative confounding by height may explain any null findings.[17] This is because taller status relates to higher SEP,[29,30] and there is also a normal height-related increase in blood pressure in childhood with taller stature being associated with higher pressures.[15,31] Indeed, adjustment for height strengthened the association between parental SEP and blood pressure in childhood in this study.

A number of studies across various populations have demonstrated tracking of blood pressure from childhood to adulthood.[16] Consistent with earlier studies, we found that the tracking correlations strengthened after early childhood.[19,24,32–34] Our findings of an inverse association between parental SEP and adult BMI is also consistent with findings from other studies.[2,3,19] One previous study also found that the association between parental SEP and adult blood pressure was attenuated by adjustment for adult BMI.[3]

We found relatively little overlap between early blood pressure and adult BMI as 2 mediating mechanisms to the association between early disadvantage and adult blood pressure. These results suggest that early blood pressure and adult BMI are distinct and additive mediators in this association. Our cohort may have been leaner in childhood than the current children are, and it is known that the tracking of BMI from childhood to adulthood is largely driven by the upper end of the distribution (ie, obese children are more likely to become obese adults). If the increases in childhood and adult obesity that have occurred in recent decades have affected all of the socioeconomic groups equally, then the role of childhood or adult BMI as a mediator of the association we have examined here will not differ markedly in contemporary populations to what we have found here. However, if there has been a greater increase in obesity in the lower socioeconomic groups over time, then the role of obesity in mediating the relationship between early SEP and later blood pressure may increase over time and result in a widening of health inequalities.

In the present study, birth weight and BMI in childhood and adolescence had little effect on the association between early disadvantage and adult blood pressure. This suggests that whereas preadulthood blood pressure and adult adiposity partially mediates the influence of adverse childhood SEP on adult blood pressure, fetal programming[35] or early developments in BMI are unlikely to do so. Low birth weight is a possible surrogate for poor fetal nutrition,[35] and it is associated with lower childhood SEP[36] and higher adult blood pressure.[3] However, birth weight seems not to be a causal link between early disadvantage and adult blood pressure. To our knowledge, only 1 investigation, the 1946 British birth cohort study,[3] has previously tested the effect of birth weight on the association between parental social class and later blood pressure, and the results were similar to those in the present study.

Limitations

Our results should be interpreted in light of some limitations. Measurement error in childhood blood pressure reduces the ability to detect mediated effects of childhood blood pressure on the association between early SEP and adult blood pressure. Indeed, a regression dilution analysis suggests that

TABLE 5. Effect of Adjustment for Early Blood Pressure at Ages 6 to 18 and Adult BMI on the Regression Coefficient Between Parental Socioeconomic Position and Adult Blood Pressure

Parental SEP Score	B	95% CI	Change in B
Adjusted for birth year and sex	−0.75	−1.14 to −0.36	Reference
Adjusted for birth year, sex, and early BP	−0.51	−0.87 to −0.16	−32%
Adjusted for birth year, sex, and adult BMI	−0.52	−0.90 to −0.16	−29%
Adjusted for birth year, sex, early BP, and adult BMI	−0.37	−0.71 to −0.02	−45%

BP indicates blood pressure. N=1285 in all models.

tracking in blood pressure from childhood to adulthood was underestimated in our data. Although estimates of blood pressure levels were based on 3 blood pressure measurements at a single visit, a more precise estimate would be obtained by averaging multiple blood pressure measurements obtained during several visits. Moreover, studies suggest that ambulatory blood pressure measures are superior to casual measures as predictors of cardiovascular morbidity and mortality.[37,38] It would be beneficial to repeat the current assessments with portable blood pressure monitors and multiple readings to better control for "white coat hypertension" and the nuisance effect of within-person variability in blood pressure.[39]

Other drawbacks of our study include homogenous sample (all white Finns) and the restricted follow-up period (ie, the participants had only reached their late 30s and, thus, we are unable to currently assess the effect of normal ageing on blood pressure trajectories and on the relationships examined here). Moreover, 50% of the original cohort was lost during the follow-up. Although dropout analysis indicated no major selection bias, smaller numbers inevitably reduced the statistical power of our analyses. Future research with larger and more diverse samples, including other ethnic groups and populations from developing countries, is needed to evaluate the generalizability of our findings. Additional studies should also more closely explore sex differences in mediating factors for the association between early SEP and adult blood pressure.

Perspectives

We have demonstrated with prospective data that adverse early SEP may influence later blood pressure in part by long-lasting damage in childhood or early adulthood. Mean levels of blood pressure in children and adults have been declining in most industrialized countries for at least the last 50 years, but the important drivers of these trends are unclear.[40] Childhood and adult obesity, important determinants of high blood pressure have increased over this time and so cannot explain these favorable trends. Whereas socioeconomic inequalities remain in most developed, as well as developing, countries, socioeconomic circumstances in early life have improved in developed countries over the last years, and the reductions in absolute levels of childhood poverty in these countries may have been important in influencing the secular trends of reductions in population levels of blood pressure in all ages over time.[18] Our results suggest that future reductions in cardiovascular disease inequalities within countries should focus on reducing inequalities in early life and identifying the factors that link socioeconomic disadvantage to increased blood pressure in childhood.

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