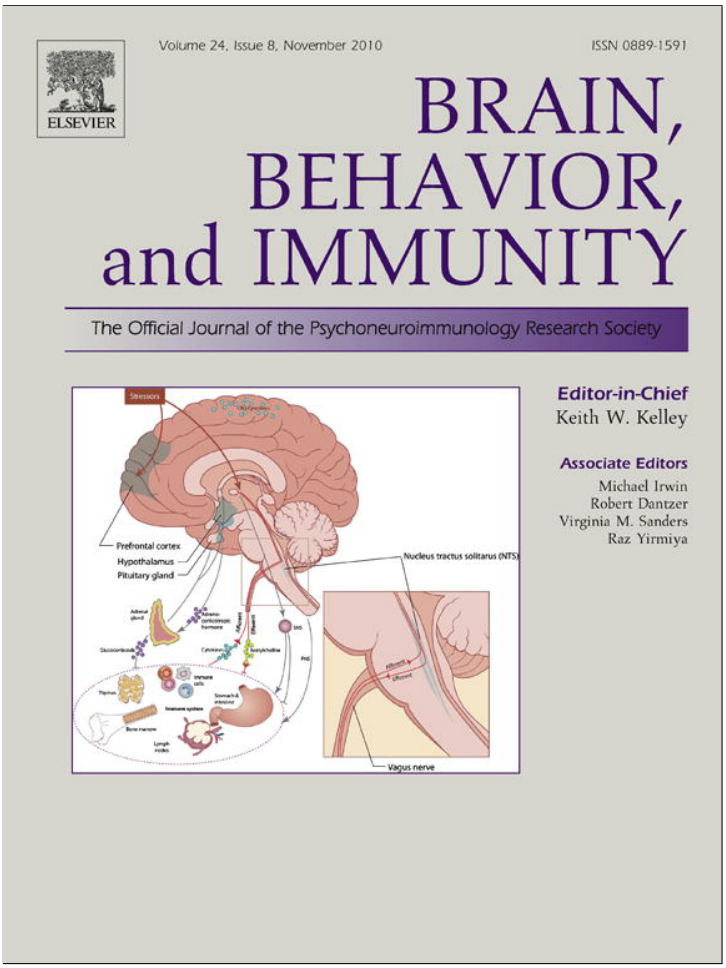

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Socioeconomic status in one's childhood predicts offspring cardiovascular risk ☆

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ABSTRACT

Objective: To test whether effects of socioeconomic environments can persist across generations, we examined whether parents' childhood socioeconomic status (SES) could predict blood pressure (BP) trajectories in their youth across a 12-month study period and C-reactive protein (CRP) levels at one year follow-up. *Methods:* BP was assessed in 88 healthy youth (M age = 13 ± 2.4) at three study visits, each 6 months apart. CRP was also assessed in youth at baseline and one year follow-up. Parents reported on current and their own childhood SES (education and crowding). *Results:* If parents' childhood SES was lower, their children displayed increasing SBP and CRP over the 12-month period, or conversely, the higher parents' childhood SES, the greater the decrease in SBP and CRP in their youth over time. These effects persisted even after controlling for current SES. A number of other factors, including child health behaviors, parent psychosocial characteristics, general family functioning, and parent physiology could not explain these effects. *Conclusion:* Our study suggests that the SES environment parents grow up in may influence physical health across generations, here, SBP and CRP in their children, and hence that intergenerational histories are important to consider in predicting cardiovascular health in youth.

1. Introduction

A vast body of literature has demonstrated that living in a low socioeconomic status (SES) environment is linked to poorer health (Adler et al., 1994; Adler and Newman, 2002). A robust relationship has been demonstrated between low SES and increased mortality (Anderson et al., 1997; Lynch et al., 1994), as well as between low SES and specific diseases or risk factors for disease, such as cancer (Conway et al., 2008; Funch, 1986; Shackley and Clarke, 2005; Ward et al., 2004), diabetes (Everson et al., 2002), and allostatic load (Szanton et al., 2005).

One of the most consistent associations of SES with a disease outcome has been with cardiovascular disease (CVD). Lower SES is associated with an increased risk of stroke, (Cox et al., 2006) cardiovascular disease (Kaplan and Keil, 1993; Pollitt et al., 2005), and cardiovascular risk factors such as blood pressure (BP), cholesterol, and subclinical CVD (BP; Appel et al., 2002; Colhoun et al., 1998; Grotto et al., 2008; Nordstrom et al., 2004). In adolescence as well, lower SES is associated with risk factors such as metabolic syndrome, higher insulin and glucose levels, higher low-density lipoprotein and lower high-density lipoprotein cholesterol, and greater body mass index (BMI; Goodman et al., 2005), and in boys greater crowding in the home has been linked to higher BP (Evans et al., 1998).

However, the majority of research in this area has focused on effects of current SES. While current context is certainly important, researchers have also argued that social environments throughout one's lifetime may be important to consider (Lynch et al., 1997; Murasko, 2007). Different models of lifetime SES have been proposed in the literature (Pollitt et al., 2005). The most common ones among these include critical period models, which argue that there are particular stages of life, for example, early childhood, during which SES is especially influential; social mobility models, which posit that changes in SES across the life course are most important; and finally accumulation models, which suggest that the effects of SES environments accumulate over time and that, hence, the longer a person lives in a low SES environment the more detrimental the associated costs.

Studies have shown that SES early in life predicts CV health in adulthood (Poulton et al., 2002). For example, one study found that medical school graduates who grew up in a low SES environment were more likely to develop CHD before age 50, even after controlling for a number of potential risk factors (Kittleson et al., 2006). Because all of these graduates had similar current high SES occupations, SES-related differences were attributed to differences in childhood SES. Similarly, a Finnish study that followed children for 34 years, concluded that childhood SES predicted adult BP over and above current, adult SES (Kivimaki et al., 2004).

Abbreviations: BP, blood pressure; SBP, systolic blood pressure; DBP, diastolic blood pressure; CRP, C-reactive protein; SES, socioeconomic status.

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These studies document the importance of considering SES at various points throughout the lifespan. In the present study, we ask the question of whether influences of the social environment might even persist across generations. That is, we tested whether the childhood circumstances that parents grew up in might have implications for their children's cardiovascular health, in terms of BP and C-reactive protein (CRP). CRP is typically released in response to acute inflammation and used as a marker of systemic inflammation. Chronically elevated levels of CRP have been linked to an increased risk of CVD (Danesh et al., 2000) and high levels of CRP are also more likely to be found among people from currently low SES backgrounds (Alley et al., 2006; Muennig et al., 2007; Nazmi and Victora, 2007).

Previous research provides some empirical evidence suggestive of the possibility of intergenerational transmission – that is, the idea that childhood experiences might get transmitted from one generation to the next, with consequences for the younger generation's health. For example, Chassin et al. (2008) investigated the intergenerational transmission of smoking behaviors and found that adults who were exposed to smoking during their childhood were more likely to have children who smoked. Perhaps of most interest is that effects on children's smoking were not driven by parents' current smoking habits, but rather by parents' childhood exposure to smoke, suggesting that the effects on child smoking are not just a function of role modeling based on current parent behavior, but instead stem at least in part from parents' childhood experiences. Another study found evidence that the SES that parents grew up with affects the birthweight of their children (Astone et al., 2007). Specifically, childhood poverty among mothers predicted low birthweight in their infants, over and above the effects of current SES, suggesting that parents' childhood experiences are able to affect outcomes in the next generation.

The animal literature also provides evidence of intergenerational effects on behavioral and physical health outcomes. For example, among rats, female offspring born to dams who are high in licking/grooming behaviors exhibited higher licking/grooming themselves, whereas offspring of low licking/grooming dams later engaged in fewer such behaviors (Champagne et al., 2003). Cross-fostering studies confirm that this is a consequence of having been exposed to either high or low licking and grooming behaviors in early life and not genetic predispositions. Experiencing more licking and grooming in early life has in turn been linked to reduced stress reactivity among adult offspring (Francis et al., 1999), suggesting a mechanism through which maternal behavior can come to influence the physical health of their offspring. Similarly, research on both plants and animals suggests a strong influence of the maternal environment on offspring defence mechanisms (Agrawal et al., 1999). Both wild radish plants and waterfleas exhibit defensive behavioral phenotypes (e.g., growing helmets on their heads in the case of waterfleas) after being exposed to predators, and these phenotypes serve to protect them from further predation. If radish plants or waterfleas are exposed to predators, their offspring will display the same behavioral phenotypes, even in the absence of a predator environment, suggesting that the environment of the parent generation can have persistent effects on the offspring generation, even if the offspring environment differs from that of the parents.

There are a number of pathways through which the childhood social environment of parents may come to influence health outcomes among their children. For example, it is possible that biological pathways are involved in the intergenerational transmission of effects of the social environment. Specifically, early life experiences can result in epigenetic modifications (stable changes in the activity, rather than sequence, of genes) which have been shown to be heritable and consequently are able to exert long-term biological influences (Jirtle and Skinner, 2007). There are also a number of psychosocial pathways through which a low SES environment may be transmitted from one generation to the next. Generally speaking, low SES families are less likely to be warm and supportive (Bradley et al., 2001), and parents who grow up in low SES environments may be more likely to engage in punitive and inconsistent behaviors themselves as adults (Wahler, 1990), which would then impact the subsequent generation. Alternatively, adverse health behaviors, such as smoking, may be shaped by childhood SES for parents, carried out into adulthood, and then impact the health of their children (Harwood et al., 2007). Finally, the psychosocial characteristics of parents may represent another possible pathway. Childhood SES may shape the mental health characteristics of parents into adulthood, which in turn may have effects on parenting behaviors; for example, parents struggling with depression may be less available for their children, which in turn may influence physical health outcomes among youth.

In the present study, we conducted a preliminary empirical investigation of the intergenerational hypothesis, aimed at (1) testing whether intergenerational transmission of SES effects onto BP and CRP can occur via parents' childhood socioeconomic environments predicting their children's BP and CRP, and, if so, (2) testing several mechanisms through which this process may occur. We also tested whether any intergenerational effects occur above and beyond effects of current SES. We followed youth across a 12 month study period, assessing BP at baseline, 6- and 12-month follow-ups, and CRP at baseline and 12 months, which allowed us to investigate more persistent changes over time in markers of cardiovascular risk. In addition we conducted a preliminary investigation of the pathways connecting parent early life experience and child CV risk. Specifically, we conducted an indirect evaluation of the possibility of genetic explanations by assessing parents' BP. We also considered the possible influence of parent mental health characteristics (parent perceived stress and parent depressive symptoms) and general family functioning to address the possibility that childhood environments affect parents' mental health and family relationship quality, which in turn influence child BP and CRP. Finally, we assessed child health behaviors (smoking and exercise behaviors) to address the possibility that parent childhood environments affect child BP and CRP via effects on children's health behaviors.

2. Methods

2.1. Participants

Participants were 88 healthy youth between the ages of 9 and 18 years ($M = 13.0 \pm 2.4$ years; 56.8% male), with one parent participating (83% of whom were mothers). They were recruited from the larger Vancouver, BC area through advertisements at schools and in newspapers. Participants were primarily Caucasian (51.1%) and Asian (35.3%), the remainder being of Hispanic (8.0%) and of First Nations (5.7%) descent. Twenty-one percent of the families were single-parent households. Exclusion criteria included not being English-speaking and having any chronic physical illness. Parents reported an average of 18.04 ± 3.14 years of education for themselves and an average of 13.19 ± 4.68 years for their parents. Parents reported an average of 1.83 people per room during their own youth, and 1.28 people per room currently. See Table 1 for participant characteristics.

2.2. Measures

2.2.1. SES

A number of measures have been used in the literature to assess SES. Some researchers have focused on prestige-based measures,

Table 1
Participant characteristics.

	n (%)	M ± SD (range)
N = 88		
Male	50 (57%)	
Female	38 (43%)	
Age		13.00 ± 2.40
Child systolic blood pressure (mmHg)		
Baseline		103.21 ± 8.65 (81.67–129.67)
6-Month follow-up		101.80 ± 9.28 (73.67–124.00)
12-Month follow-up		103.61 ± 9.85 (83.00–131.00)
Child diastolic blood pressure (mmHg)		
Baseline		64.70 ± 7.76 (50.00–91.00)
6-Month follow-up		65.54 ± 9.63 (47.00–101.67)
12-Month follow-up		63.76 ± 9.09 (51.00–103.00)
Child C-reactive protein (mg/L)		
Baseline		.58 ± 1.02 (.19–7.95)
12-Month follow-up		.57 ± 1.02 (.19–7.95)
Parent systolic blood pressure (mmHg)		
Baseline		112.65 ± 15.32 (81.67–170.00)
Parent diastolic blood pressure (mmHg)		
Baseline		73.51 ± 9.98 (52.67–106.33)
Education (years)		
Current (parent education)		18.04 ± 3.14
Parents' childhood (parents' parent's education)		13.19 ± 4.68
Crowding (people/room)		
Current		1.28 ± .59
Parents' childhood		1.83 ± .86
Parent questionnaires		
CESD		9.94 ± 8.47
PSS		14.59 ± 6.89
FAD-GFS		24.07 ± 6.01
Child health behaviors		
Exercise days (out of 14)		8.91 ± 10.10
% Smoked in past 6 months	7 (8.0%)	

CESD, Center for Epidemiological Studies Depression Scale; PSS, Perceived Stress Scale; FAD-GFS, Family Assessment Device–General Functioning Scale.

such as years of education and occupation, others on resource-based measures focusing on material assets, for example, family income and savings (Krieger et al., 1997; Winkleby et al., 1992). As different measures of SES could indicate different pathways to health, we included one SES measure belonging to each category in order to test whether differential effects exist across different SES measures. In addition, we chose measures that parents would be able to easily recall for their own childhood. Consequently we chose a prestige-based SES measure, years of education, as well as a resource-based measure, family crowding.

Years of education. (1) Parent's childhood SES: number of years of education completed by the participating parent's parents. For 2-parent families, the higher of the two was used. (2) Current SES: number of years of education completed by the child's parents. For 2-parent families, the higher of the two was used.

Crowding. (1) Parents' childhood SES: the ratio of people to bedrooms in the family house for each year of childhood, averaged across the 18 years of the parents' childhood. (2) Current SES: the ratio of people to bedrooms in the family house during the most recent year of the child's life.

2.2.2. BP

At every visit, youth were seated in separate rooms and rested for 15 min after which one test BP reading was taken and discarded. Following this, three BP readings, each 2 min apart, were taken using a BPM-100 automated BP monitor (BP Tru Medical Devices; Coquitlam, BC, Canada) with a standard occluding cuff. For values of systolic BP (SBP) and diastolic BP (DBP) the averages of the three SBP and DBP readings at each visit were calculated. Parent BP was assessed at the first visit, following the same procedure. See Table 1 for information regarding our participants' blood pressure values and other participant characteristics.

2.2.3. C-reactive protein (CRP)

To investigate inflammatory markers implicated in CVD, baseline and one year follow-up CRP levels were assessed in youth. At both occasions, whole blood was drawn into BD Vacutainer serum separation tubes and spun at 1200 rpm for 10 min. Serum was stored at −30 °C until further assaying. Serum levels of CRP were then measured using a high-sensitivity chemiluminescence technique (Immulite, 2000, Diagnostic Products Corporation, Los Angeles, California) with a detection threshold of 0.20 mg/L. One participant had an outlying CRP value >10 mg/L at one measurement occasion. This score was capped at the highest score in the rest of the sample. See Table 1 for information regarding youth's CRP values and other participant characteristics.

2.2.4. Body mass index (BMI)

Youth's height and weight were measured at baseline and BMI was computed as kg/m^2. This variable was included as a covariate in analyses.

2.2.5. Youth puberty status

At every visit, youth completed the Physical Development Scale, which is comprised of five questions (Petersen et al., 1988). Based on their responses youth are categorized as being in pre-, early, mid-, or late puberty. This variable was included as a covariate in analyses.

2.2.6. Psychosocial parental and general family characteristics

A number of psychosocial parent and general family characteristics that could provide explanations for the associations between parents' childhood SES and BP outcomes among youth were assessed through self-report questionnaires.

Parental depressive symptoms. Parental depressive symptoms were assessed through the Center for Epidemiological Studies Depression Scale (CES-D; Radloff, 1977), a widely used depression scale consisting of 20 items assessing the frequency of a number of behaviors, such as feeling hopeful, lonely, or sad, over the past week (from 0 = 'less than one day' to 3 = '5–7 days'), that has been tested in clinical as well as general populations. Parents completed the CES-D as part of the first study visit. Internal consistency reliability of the CES-D was alpha = .91 in the present sample.

Parental perceived stress. Parental perceived stress was assessed through the perceived stress scale (PSS; Cohen et al., 1983). The PSS is a 14-item scale asking people about their feelings and thoughts during the past month, such as feeling 'on top of things'. It is answered on a 0–4 scale, ranging from 0 = 'never' to 4 = 'very often'. Parents completed the PSS during the first study visit. Internal consistency reliability of the PSS was alpha = .91 in the present sample.

General family functioning. Parents completed the general functioning scale of the Family Assessment Device (FAD-GFS). The FAD-GFS (Epstein et al., 1983) consists of 13 items answered on a 4-point Likert scale ranging from 1 = 'strongly agree' to 4 = 'strongly disagree'. This measure discriminates between nonclinical and psychiatric families (Byles et al., 1988). Moreover, it was significantly correlated with variables such as family structure (single-parent versus two parent family), marital violence and disharmony, and whether parents had been arrested at any point in time (Byles et al., 1988). Higher scores indicate unhealthier family functioning. Parents completed the GFS as part of their second study

visit. Internal consistency reliability of the FAD-GFS was alpha = .84 in the present sample.

2.2.7. Child health behaviors

Youth indicated the number of cigarettes they had smoked during the past 6 months. Because of the overall low prevalence of smoking ($n = 7$; 8%) this variable was dichotomized and participants characterized either as smokers or nonsmokers.

Youth's exercise levels were assessed using the Child Physical Activity and Exercise questionnaire (Aaron et al., 1993). Responses to the first question, on how many days during the past two weeks youth had performed hard exercise (e.g., cycling) for at least 20 min were used. Higher scores indicate higher activity levels.

2.3. Procedure

Written assent and consent was obtained from participating youth and their parents, respectively. Participating youth visited the lab three times over 12 months, on average once every 6 months, together with a parent. At the first visit, youth and their parents completed computer questionnaires (younger participants were given the option of having questions read out to them by their research assistant); BP measurements were taken from youth at each visit and from parents at the baseline visit. Youth also underwent a blood draw at the baseline and one year follow-up visit. As this was a study focused on youth, health-related assessments in parents were minimal. Participants were reimbursed for their time as well as transportation to the study site. The study was approved by the Research Ethics Board of the University of British Columbia.

2.4. Statistical analyses

Hierarchical linear modeling (HLM) was used to predict youth's BP trajectories over the 12 month study period, given the three time points of measurements. HLM is a multi-level modeling technique that can be used to assess both within-person and between-person factors predicting changes in a dependent variable (e.g., BP) over time.

We examined the influence of the between-person model (level 2) on the slopes and intercepts of the within-person model at level 1 (BP) as a function of factors varying across people. We first added youth age, sex, and ethnicity into our model as covariates. We next entered our primary variable of interest, parents' childhood SES. To investigate the effect of a number of alternative explanations, we entered each variable (i.e., current SES, parent BP, parent psychosocial characteristics, and child health behaviors) simultaneously with parents' childhood SES to test whether the relationship between parent early life SES and youth BP remained significant.

All relationships were estimated using full maximum likelihood and robust standard errors.

For CRP, we tested whether SES could predict CRP levels at one year follow-up. Standard linear regression was used to examine relationships between parent childhood SES and youth CRP levels at 12-month follow-up. All analyses controlled for baseline levels.

3. Results

3.1. Preliminary analyses

We first tested associations between parents' childhood and current SES measures. Early childhood crowding and current crowding were positively correlated ($r = .457$, $p < .01$), as were parents' education and parents' parents' education ($r = .235$, $p < .05$). In addition, parents' childhood SES (in terms of education of their parents) was associated with parents' childhood crowding ($r = -.334$, $p < .01$). In addition, youth's SBP aggregated across the three study visits, that is, their mean value across the three visits, was positively correlated with parent SBP at baseline ($r = .25$, $p < .05$), but the same was not true for child and parent DBP ($r = .01$, $p > .50$). Associations of BP and CRP with traditional covariates (demographics, BMI, puberty) are presented in Tables 2 and 3.

In addition, linear regression analyses predicting baseline youth SBP, DBP, and CRP from parent childhood crowding and educational attainment in the parent's childhood home were conducted. Parent's childhood crowding did not predict youth baseline CRP ($B = -.047$, $p > .05$). Educational attainment in the parent's home

Table 3
Parent childhood SES and covariates predicting CRP aggregate scores and 12-month follow-up values.

	Aggregate CRP			Time 2 CRP[a]		
	B	SE	*p*	*B*	SE	*p*
Age	−.048	.034	.16	−.005	.058	.93
Sex	−.144	.163	.38	−.113	.284	.69
Ethnicity	.098	.042	.02	−.004	.072	.95
Youth BMI	.049	.023	.04	.082	.039	.04
Youth Puberty Status	−.170	.071	.02	−.089	.124	.48
Parent Education	−.025	.026	.33	−.018	.045	.70
Parents' Parent's Education	−.018	.017	.30	−.060	.032	.06
Current Crowding	.122	.137	.38	.115	.257	.65
Parent Childhood Crowding	.234	.091	.01	.602	.124	.00

Note: Coefficients in this table are unadjusted associations of the target variable with youth C-reactive protein levels. SES, Socioeconomic Status; CRP, C-reactive protein; BMI, Body Mass Index.

[a] Time 2 CRP results are controlling for time 1.

Table 2
Parent childhood SES and covariates predicting 12-month SBP trajectories and aggregate scores.

	Aggregate SBP			Trajectories of SBP		
	B	SE	*p*	*B*	SE	*p*
Age	.758	.309	.02	.254	.299	.40
Sex	−2.643	1.508	.08	1.715	1.405	.23
Ethnicity	−.306	.407	.46	−.082	.622	.90
Parent SBP	.119	.047	.013	−.236	.643	.71
Youth BMI	.562	.213	.01	−.357	.713	.62
Youth Puberty Status	.53	.64	.41	−.130	.795	.87
Parent Education	−.010	.243	.97	.185	.187	.33
Parents' Parent's Education	.201	.162	.22	−.402	.144	.01
Current Crowding	−.674	1.290	.60	−.321	1.204	.79
Parent Childhood Crowding	−.807	.880	.36	2.294	.920	.02

Note: Coefficients in this table are unadjusted associations of the target variable with youth systolic blood pressure.
SES, Socioeconomic Status; SBP, Systolic Blood Pressure; BMI, Body Mass Index.

did not predict youth baseline CRP ($B = .025$, $p > .05$). Parent's childhood crowding did not predict youth baseline SBP ($B = -1.883$, $p > .05$). However, parent childhood SES (education) did predict youth SBP ($B = .441$, $p < .05$). Finally, parent's childhood crowding did not predict youth baseline DBP ($B = .048$, $p > .05$) and educational attainment in the parent's childhood home did not predict youth baseline DBP ($B = .105$, $p > .05$).

3.2. Does SES predict averaged cardiovascular risk across the year?

Given the aggregated scores, we first tested whether SES predicted average BP or CRP – that is, whether SES variables are associated with a multiply-assessed indicator of basal levels of BP or CRP. Using linear regression analyses that controlled for child age, sex, and ethnicity, parent education, educational attainment in parent's home, current crowding, and parent childhood crowding were not associated with aggregated child SBP (all $ps > .10$). Current parent education, educational attainment in parent's home, and crowding also were not associated with CRP (all $ps > .50$). Parent childhood crowding predicted averaged CRP ($B = .233$, SE = .089, $p = .01$, respectively).

3.3. Does SES predict change over time in youth's BP?

We next tested whether SES would predict change over time, rather than averaged values, in children's CV risk scores. HLM analyses revealed that parents' childhood SES predicted changes in youth's SBP across the 12 month study period ($B = -.434$, SE = .1468, $p < .01$), such that parents who grew up in less educated households had youth whose SBP increased over time, or stated another way, parents who grew up in more educated households had youth whose SBP decreased over time. Current SES (parents' own education) was not associated with youth's SBP over time ($B = .099$, SE = .2451, $p > .50$). Parents' childhood SES continued to predict youth's BP trajectories after current SES was taken into account ($B = -.457$, SE = .1465, $p < .01$).

Similarly, SES in terms of crowding in the parents' childhood home predicted changes in youth's SBP across the 12-month study period ($B = 2.413$, SE = 1.0075, $p < .05$), such that more crowding was related to greater increases in SBP over time, or stated another way, less crowding was related to greater decreases in SBP over time. Current crowding did not predict changes in youth's SBP over time ($B = .087$, SE = 1.2224, $p > .50$). The effect of parents' childhood crowding remained significant after controlling current crowding ($B = 2.4178$, SE = 1.0002, $p < .05$). Finally, when simultaneously including both indicators of parent childhood SES, education and crowding, in the analyses, parent childhood SES in the form of education remained a significant predictor of youth SBP trajectories across the one year study period ($B = -.346$, SE = .131, $p < .05$) whereas the effect of parent childhood crowding was reduced to marginal significance ($B = 1.6421$, SE = .8702, $p < .10$). See Table 2 for unadjusted coefficients of variables predicting youth's SBP.

SES variables did not predict youth's DBP (all $ps > .10$).

3.4. Does SES predict youth's CRP levels over time?

Using linear regression analyses, parent childhood SES marginally predicted youth's CRP at one year follow-up ($B = -.065$, $p < .10$) controlling for baseline, such that parents who grew up in less educated households had youth whose CRP levels at one year follow-up, controlling for baseline levels, were marginally higher, or conversely that parents who grew up in more educated households had youth whose CRP levels at follow-up were marginally lower. Current household education was not associated with youth's CRP levels at follow-up ($B = -.025$, $p > .60$). Parent's childhood SES continued to marginally predict youth's CRP levels after current education was taken into account ($B = -.064$, $p < .10$).

Linear regression analyses also revealed that parents' childhood SES in the form of crowding predicted youth's CRP at time 2 controlling for time 1 ($B = .612$, $p < .001$), such that greater parent childhood crowding predicted greater CRP one year later, controlling for baseline values, among youth. Current crowding did not predict youth's CRP at follow-up ($B = .155$, $p > .50$). Parent's childhood crowding continued to predict youth's CRP levels at follow-up after current crowding was taken into account ($B = .619$, $p < 001$). Finally, when simultaneously including both indicators of parent childhood SES, education and crowding, in the analyses, parent childhood SES in the form of parent childhood crowding remained a significant predictor of youth CRP at one year follow-up ($B = .590$, SE = .139, $p < .01$) whereas the effect of education was reduced and no longer significant ($B = -.015$, SE = .032, $p > .10$). See Table 3 for unadjusted coefficients of variables predicting youth's CRP levels.

3.5. Possible pathways connecting parents' early life environment to youth's CV trajectories

As one test of the possibility of genetic transmission, we included parent SBP in our models. The associations between parents' childhood SES (both education, $B = -.4600$, SE = .1483, $p < .01$ and crowding, $B = 2.4900$, SE = 1.0708, $p < .05$) persisted over and above the contribution of parent SBP to child SBP. Because we did not obtain blood samples on parents, we were not able to conduct comparable analyses for CRP.

Including other traditional covariates such as BMI (education: $B = -.375$, SE = .173, $p < .05$; crowding: $B = 2.396$, SE = 1.087, $p < .05$) and puberty status[1] (education: $B = -.122$, SE = .049, $p < .05$; crowding: $B = 3.654$, SE = 0.982, $p < .01$) did not affect youth's SBP trajectories. Similarly, there were no effects on youth's CRP at follow-up controlling for baseline when adding BMI (education: $B = -.082$, SE = .033, $p < .05$; crowding: $B = .634$, SE = .118, $p < .001$) or puberty status (education: $B = -.073$, SE = .038, $p < .10$; crowding: $B = .608$, SE = 0.143, $p < .001$).

Including youth health behaviors did not change the relationship between parents' childhood SES and youth's SBP trajectories. Parent childhood SES remained significant after controlling for smoking (education: $B = -.427$, SE = .1587, $p < .01$; crowding: $B = 2.463$, SE = 1.0292, $p < .05$) and youth exercise behaviors (education: $B = -.429$, SE = .1537, $p < .01$; crowding: $B = 2.636$, SE = 1.1191, $p < .05$). The impact of parent childhood SES on youth's CRP at follow-up controlling for baseline was not altered by the inclusion of youth smoking (education: $B = -.064$, SE = .036, $p < .10$; crowding: $B = .617$, SE = .135, $p < .001$) and exercise behaviors (education: $B = -.063$, SE = .036, $p < .10$; crowding: $B = .639$, SE = .133, $p < .001$).

Finally, the effects of parents' childhood SES on youth SBP also remained significant after controlling for psychosocial characteristics such as parent depressive symptoms (education: $B = -.423$, SE = .1497, $p < .01$; crowding: $B = 2.667$, SE = 1.1030, $p < .05$), parent perceived stress (education: $B = -.425$, SE = .1494, $p < .01$; crowding: $B = 2.601$, SE = 1.0589, $p < .05$), and general family functioning (education: $B = -.412$, SE = .1420, $p < .01$; crowding: $B = 2.323$, SE = 1.002 $p < .05$). The impact of parent childhood SES on youth's CRP levels at follow-up controlling for baseline was not altered when controlling for parent depressive symptoms (education: $B = -.063$, SE = .036, $p < .10$; crowding: $B = .601$, SE = .137, $p < .001$), parent perceived stress (education: $B = -.063$, SE = .035, $p < .10$; crowding: $B = .594$, SE = .137, $p < .001$), and gen-

[1] Findings remain unchanged if puberty status is substituted for age.

eral family functioning (education: $B = -.066$, SE = .036, $p < .10$; crowding: $B = .679$, SE = .133, $p < .001$).

3.6. Parent childhood SES predicting parent BP

Lastly, given that we also obtained BP at baseline on parents, we tested whether childhood or current SES predicted parents' BP. Linear regression analyses revealed that parent childhood SES (education) predicted parents' current SBP ($B = -.695$, $p < .05$), such that parents who came from less educated households during childhood had higher current SBP. Similarly, current education predicted parents' current SBP on its own ($B = -1.393$, $p < .01$) and above parent childhood SES (education; $B = -1.217$, $p < .05$). However, crowding did not predict parents' SBP. Parent DBP was predicted only by current crowding ($B = -4.500$, $p < .05$).

4. Discussion

The present study provides novel data supporting the notion that the effects of childhood environments can persist into the next generation's physiological health. Lower childhood SES in parents predicted increases over a 12-month period in youth's SBP and lower CRP levels at follow-up (or conversely, higher parental childhood SES predicted decreases in youth's SBP and CRP over time). Decreases over time in physiological indicators could possibly reflect an accumulation of positive, buffering effects from high resource families. These effects persisted even after controlling for current SES, and, in the case of BP, parent BP. These findings suggest the importance of considering not just the immediate environment that children and adolescents live in, but also the context that their families grew up in for understanding physiological health.

In addition, it is important to note that while our results are statistically significant, the changes in BP and CRP values found as part of this study do not represent clinically meaningful changes among this sample of healthy adolescents. Rather, we expect these trajectories to perhaps represent the beginnings of longer, ongoing trajectories of patterns that may persist throughout the adolescent years and into adulthood, eventually affecting cardiovascular health later in life.

Our findings have implications for conceptual models of SES and cardiovascular health. First, they suggest that perhaps life-course models should be broadened to include environments from previous generations in understanding influences on current health. This intergenerational approach may represent a variant on the critical period notion, that SES during particular life periods has the most potent effects (Rosvall et al., 2006). Second, these findings may help explain the mixed findings on SES and BP during childhood. Unlike other life periods, the relationship between SES and BP during childhood has been inconsistently documented (Chen et al., 2002). One reason for this may be that during this formative period, the SES of parent's own childhood environment may play a stronger role in shaping CV risk in children. This would be consistent with the fact that in adulthood, current SES is associated with BP, but that this association with current SES is not clearly present in adolescence (Chen et al., 2002; Colhoun et al., 1998).

Our findings furthermore suggest that there is value to using both prestige- and resource-based SES measures. On its own, each was associated with CRP and SBP over time. When including the two types of measures simultaneously, prestige SES (education) was found to predict SBP, whereas resources (crowding) predicted CRP. Future research is warranted to better understand why different SES measures may have different relationships with different physiological outcomes.

Why would one's childhood social environment have effects on the next generation's physiological health? Evolutionarily, the intergenerational transmission of physiological states could be adaptive in serving as one mechanism through which parents can prepare their offspring for an environment similar to the one in which they grew up. If true, it provides for the opportunity to let events that happened before the parent gave birth to their child nonetheless influence the development of the child, with the idea of better preparing the child for dealing with his/her own life circumstances. This is in line with the broader framework of evolutionary developmental biology (Lickliter and Schneider, 2006), which holds that other factors, such as behavioral modifications brought about by changes in the environment (e.g., the presence of predators; Agrawal et al., 1999) can lead to variations in current and subsequent generations.

In the present study, we tested several psychosocial pathways, including effects on parent mental health, family functioning, and child health behaviors, but did not find mediational support for any of these pathways. Below we speculate on some additional possibilities that we were not able to test in the present study.

One possibility is via biological pathways, such as epigenetic transmission, from parent to child. Epigenetic programming occurs in different ways that affect a cell's ability to transcribe a particular gene into RNA, which in turn affects how much of the gene's protein is ultimately synthesized. For example, the methylation of DNA by certain enzymes prevents regulatory molecules from binding to the promoter, which in turn can suppress the rate of transcription of a gene (Bird, 2001; Whitelaw et al., 2006). Chromatin remodeling involves chemicals attaching to the histone proteins that package DNA, which can cause DNA to become more tightly coiled, also limiting gene transcription.

Research suggests that the social environment can have effects on genomic function in a manner that persists over long periods of time. Although not directly addressing epigenetic effects per se, one study found that adolescents who were in low SES environments during their first 2–3 years of life showed greater expression of genes encoding the toll-like receptor 4, which signals inflammatory responses in the presence of bacteria, and reduced expression of genes encoding the glucocorticoid receptor, which conveys anti-inflammatory signals from the hormone cortisol, and that these effects of SES at age 2–3 persisted into adolescence (Miller and Chen, 2007). More direct evidence of epigenetic effects comes from the animal literature (Weaver et al., 2004). This work shows that positive social environments (high levels of nurturing of rat pups in their first week of life by their mothers) can create epigenetic modifications, such as demethylation of DNA and acetylation of histone proteins, that facilitate expression of the glucocorticoid receptor in hippocampal tissue, and that these modifications persist into adulthood.

Another possible explanation that is more social relates to parenting style. For example, parents who grow up themselves in a low SES environment may have experienced harsh and inconsistent parenting that they then model when they become parents themselves. Low SES families are less likely to be warm and supportive (Bradley et al., 2001) and parents from low SES environments are more likely to engage in punitive and inconsistent behaviors (Wahler, 1990). If parents adopt negative parenting styles that they were exposed to as children, their own children may experience more stressful home lives, leading to physiological consequences over time, such as increased SBP and CRP.

Our results indicate that parent childhood SES most strongly predicts youth's change trajectories, more so than average levels. This may mean that as children develop, their CV risk profiles begin to diverge over time, and hence that it is important to study dynamic, rather than static, physiological indicators of CV risk in youth. Studies have shown that adolescents' BP and CRP values

change over time (Matthews et al., 2003; Miller and Wrosch, 2007); understanding the predictors of these changes may provide important clues regarding who may be at risk for CV problems later in life. We failed to find a relationship between parent's childhood SES and youth's DBP. It is unclear why parental SES predicted youth's SBP but not DBP, however, it has been previously suggested that SBP and DBP tap different aspects of cardiovascular health (with SBP relating to myocardial and DBP to vascular functioning; Tomaka et al., 1993, 1997).

Parent childhood SES was largely unrelated to baseline cardiovascular risk variables, with one exception (high education in parent's childhood home associated with high baseline SBP in offspring). The reason for this pattern is unclear, and warrants further research to determine if it is a replicable finding.

This study has a number of strengths. First of all, we were able to follow our participants longitudinally over a period of 12 months and reassess their blood pressure at three separate time points as well as assess changes in CRP levels across the 12 month period. We furthermore took advantage of a powerful statistical technique, hierarchical linear modeling, allowing us to examine trajectories of change over time in youth's BP. Lastly, we conducted extensive SES assessments to measure both parents' childhood SES as well as children's SES throughout their lifetime. Short of conducting a prospective, intergenerational study, the approach in this study represents one of the only ways to investigate the influence of the SES environment of one generation on health outcomes in a subsequent generation. While the SES assessments were conducted retrospectively, we focused on concrete variables more likely to be remembered accurately over time by our participants (e.g., years of education, rather than income). Nonetheless, retrospective SES assessments remain a limitation of the present study.

In addition to the retrospective SES measures, another limitation was that our assessment of BP was conducted in the laboratory. Future studies that also utilize ambulatory BP measurements would provide a better indication of BP experienced in every day life. Including measures of parent physical health other than BP together with measures of other possible psychosocial mediators may also be important as parents' health status may have impacted their children's physical health through other biological and psychosocial pathways. Finally, other variables likely to influence youth's health outcomes, such as family diet, should also be investigated in the future.

In conclusion, our study showed that parents' childhood SES influences trajectories of SBP and one year follow-up levels of CRP among their children, even after parental SBP had been taken into account. Our findings draw attention to the need to broaden current models of the impact of lifecourse SES to take into account how the early life environments of parents may also shape children's health. Although speculative, this may be due to a mechanism that serves to prepare future generations for the environment they will likely encounter throughout their lives, based on the environment their parents grew up in. These findings are unique in demonstrating that it is possible to transmit physiological effects of the social environment from parent generation to children, with implications for the physical health of future generations.

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