What Do Trajectories of Childhood Socioeconomic Status Tell Us About Markers of Cardiovascular Health in Adolescence?

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Objective: The current study examined trajectories of socioeconomic status (SES) throughout childhood and their relationship to markers of cardiovascular health in adolescence. The goal was to determine whether early-life SES, current SES, cumulative SES, and/or social mobility best explained the relationship between SES experiences across an adolescent's life span and current blood pressure (BP), heart rate (HR), and body mass index (BMI). **Design:** One hundred two adolescents completed cardiovascular health assessments including systolic blood pressure, diastolic blood pressure, HR, and BMI. Parents reported on family SES, indicating the number of bedrooms in the family home for each year of the child's life. **Results:** Using Jones, Nagin, and Roeder's semiparametric group-based method, four distinct trajectories of childhood SES were identified. Trajectory groups were differentially related to adolescents' systolic blood pressure and diastolic blood pressure. A trajectory showing low early-life SES that increased through childhood was associated with the highest BP in adolescence. Partial correlation analyses specifically examining the various life-course scenarios similarly indicated that early-life SES was the strongest predictor of adolescents' BP. Trajectories of childhood SES were unrelated to HR and BMI. **Conclusions:** Of the life-course models that we tested, an early-life SES model best explained adolescents' current BP. These findings point toward early-life developmental processes as potential candidates for explaining the relationship between SES and risk factors related to cardiovascular disease. They suggest that interventions designed to reduce SES health disparities should take place early in a child's life. **Key words:** socioeconomic status, life-course models, blood pressure, adolescents.

BIC = Bayesian information criterion; **BMI** = body mass index; **BP** = blood pressure; **CVD** = cardiovascular disease; **CV** = cardiovascular; **DBP** = diastolic blood pressure; **HR** = heart rate; **SES** = socioeconomic status; **SBP** = systolic blood pressure.

INTRODUCTION

Socioeconomic status (SES) is an important determinant of health status at each phase of the life cycle. Thus, among children, adults, and the elderly, individuals in lower SES groups experience higher rates of morbidity and mortality due to a wide range of medical conditions (1–3). Life-course models of SES have proposed various pathways through which SES at different stages can influence health. At least four major life-course models exist: critical period, current SES, cumulative SES, and mobility. The critical period and current SES models emphasize the importance of timing of SES exposure, whereas cumulative SES and mobility models emphasize dynamic aspects of SES across time.

According to critical period models, there is a window of time in which SES exerts its most profound effects on the body. One example of a critical period is thought to be in early childhood, when important developmental processes are underway. Early-life environmental conditions may program a pattern of biological and behavioral responses that have a long-term impact (4,5). In fact, past research has shown that early childhood environments predict adult cardiovascular disease (CVD), stomach cancer, and hemorrhagic stroke (see Refs. 6 and 7 for reviews). Importantly, these associations persist after accounting for adult SES.

In contrast, current SES models specify paths through which socioeconomic experiences at any point in the life cycle can influence concurrent health status. Such models suggest that the current living environment can have a relatively immediate impact on health. Although few studies have examined the impact of current socioeconomic circumstances independent of the childhood environment, research evidence suggests that current SES is an important predictor of self-reported health (8) and CVD mortality (9).

Models of cumulative risk focus on the additive effects of SES experience. Thus, individuals who are exposed to low SES for longer durations are thought to be at greater risk. Indeed, research evidence has shown that the amount of time spent in low SES is an important predictor of mortality (10,11) and young adults' self-reported health (12).

Finally, mobility models propose that changes in SES over the life course will affect health. Markers of SES like family income can fluctuate from year to year (13). Thus, over any given period of time, a person may experience upward mobility or downward mobility, and these changes in social status may affect health. A few studies have examined these types of relationships. For instance, downward mobility in adulthood has been associated with increased risk of hypertension (14) and poorer self-reported health (15). Conversely, upward mobility in adulthood has been associated with cardiovascular risk reduction (16).

Previous research on the longitudinal relationship between SES and health has mainly focused on adult populations, and we know little about these processes in childhood and adolescence. However, findings from the mental health literature suggest that children and adolescents are affected by both timing and dynamic SES indicators (17–19). Specifically, early childhood experiences of poverty influence future mental health outcomes; regardless of subsequent changes in the child's SES environment (18). Furthermore, the experience of persistent poverty is associated with worse mental health outcomes than the experience of transient poverty or no poverty (19). Clearly, patterns of SES over time have implications

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for children's mental health; and it is also important to test whether these findings extend to markers of children's physical health.

Recent evidence indicates that poor cardiovascular (CV) health begins in the early decades of life. Many young individuals have developed atherosclerotic plaque by the time they reach adolescence (20,21), and risk factors for cardiovascular disease (CVD), including obesity, high blood pressure, and elevated lipids track across childhood and adolescence and into adulthood (22,23). The life-course approach has been used successfully among adult populations to examine relationships between childhood and adulthood socioenvironmental circumstances and CV mortality (10,24,25).

The current article examines patterns of SES through childhood and their association with systolic blood pressure (SBP), diastolic blood pressure (DBP), heart rate (HR), and body mass index (BMI) in adolescence. To capture changes in family SES over time, we focused on wealth, or the resources or spending power a family has accumulated. Because we did not think participants could provide accurate data on assets and debts (the two components that define wealth) for each year, we instead used an indirect but easily recalled proxy, the number of bedrooms in the family home. We also should note that we recognize SES to be a multidimensional construct (26), with wealth representing only one approach to conceptualizing it. SES could also be conceptualized in terms of social prestige (e.g., education, occupation), and in terms of poverty (e.g., living below poverty level, house crowding, housing instability). In the present study, we chose to focus on wealth because it is more dynamic than prestige measures such as education, and therefore more appropriate for looking at trajectories of SES over time. In addition, wealth measures capture the entire SES spectrum, as opposed to poverty measures, which capture only one end of the spectrum.

Our study objective was to determine which life-course model (or combination of models) best explains the impact of SES throughout childhood on adolescent health. We took two different approaches to answering this question. First, we utilized a new statistical approach to identify common trajectories of family SES across an adolescent's life span, and then used these trajectories to predict adolescent CV risk markers. We expected to identify distinct trajectories of family SES that would represent variations in the timing and dynamics of SES experiences. To the extent that distinct trajectories of SES could be identified, we expected that they would be differentially related to adolescents' CV outcomes. Given that we could not know in advance which types of trajectories would emerge in our sample (or whether they would correspond to the life-course models of interest), our second approach used specific indices of early-life SES, cumulative SES, current SES, and social mobility to hone in on each of the life-course models and test their relations to adolescents' SBP, DBP, HR, and BMI. Although we expected the results from the two analytic approaches to converge, we thought that the former would be informative about the kinds of life-course patterns in our sample, whereas the latter would serve as a direct test of the early-life, cumulative, current, and mobility models of SES.

TABLE 1. Demographic and Health Characteristics of the Sample (N = 102 Unless Otherwise Indicated)

Characteristic	Mean ± SD or Frequency
Family	
Current family income (1–6 scale)	4.0 ± 1.5
Parent years of education	15.9 ± 2.4[a]
Adolescents	
Age	15.6 ± 1.1
Female	53%
White	75%
African American	24%
SBP (mm Hg)	108.9 ± 9.2
DBP (mm Hg)	60.2 ± 6.2
HR (bpm)	73.1 ± 10.8 (N = 101)
BMI	24.2 ± 4.9 (N = 100)

For family income, category 3 corresponds to $50,000–74,999, and category 4 corresponds to $75,000–99,999. Parent years of education represent the average of both parents.

[a] Families in our sample were predominantly middle to upper class.

METHOD

Participants

Public high school students in the St. Louis area were recruited via school flyers, announcements, and classroom presentations. Adolescents were eligible for the study if they were a) between the ages of 14 and 18, b) fluent in the English language, c) free of chronic medical conditions and mental health problems, and d) not using medication that affected the CV system. The final sample consisted of 102 adolescents whose characteristics are described in Table 1. The Institutional Review Board at Washington University approved this study. The data were collected between 2002 and 2003.

Socioeconomic Status

To capture SES trajectories over time, parents were asked to indicate the number of bedrooms in the family home during each year of the child's life. Number of bedrooms is accurate to the year, meaning that it captures the year a move was made but not when during that year the move happened. Number of bedrooms was used as a marker of SES for a number of reasons. First, retrospective recall of housing is more accurate than other indicators of wealth such as family income (27). Second, housing is a more dynamic measure than other SES indicators, such as parental education. In our sample, most families (68.6%) had lived in two or more homes through the child's life, allowing us to capture changes in family housing status over time. We also considered whether it would be more appropriate to use a measure of number of bedrooms per family size. However, number of bedrooms per family size would capture crowding, which we argue is distinct from wealth, and is best used to distinguish individuals at the low end of the SES spectrum. To empirically test our reasoning, we correlated both current number of bedrooms in the home and current number of bedrooms per family size with two well-established indicators of wealth—current family income and savings. Whereas current number of bedrooms was significantly associated with both savings and income (r = 0.25, p = .02 and r = 0.31, p = .004, respectively), current number of bedrooms per family size was not significantly correlated to either savings or income (p values >.10). This suggests that the number of bedrooms measure is more similar to other indicators of wealth than the number of bedrooms per family size measure. That said, family size itself could be a potential confound linking bedrooms to biological outcomes; so, we evaluated it as a covariate in secondary analyses.

Physiological Measures

Blood Pressure

SBP and DBP were monitored using a Dinamap Pro 100 automated BP monitor (Critikon, Tampa, FL) with a standard occluding cuff on the participant's right arm. SBP and DBP measures were taken three times during the last 5 minutes of a 10-minute baseline rest period. The averages of the three measures of SBP and DBP were used in statistical analyses. The coefficient alpha was 0.96 for SBP and 0.94 for DBP.

Heart Rate

Heart rate (HR) was measured through electrocardiogram (EKG) monitoring. An EKG signal was transduced using two active Meditrace SF450 disposable silver or silver chloride electrodes (Kendall-LTP, Chicopee, MA) placed on each side of the abdomen and a ground electrode beside the navel. The EKG signal was filtered and amplified by the Biopac MP100 system (Biopac Systems, Santa Barbara, CA). HR was monitored continuously during the last 5 minutes of the 10-minute rest period. HR scores represent the average number of RR intervals (i.e., heart beats) per minute from the EKG recordings.

Body Mass Index

Height and weight were taken on a standard medical-grade balance beam scale and body mass (BMI) was computed from these two variables [BMI = (weight in kilograms/(height in meters) × (height in meters)]. For children and adolescents, the National Center for Health Statistics presents BMI by age and sex, using Z scores. These age- and sex-adjusted Z scores were used in this study.

Potential Confounders

We measured a number of processes that could provide alternative explanations for relations between childhood SES and biological outcomes. We collected demographic information, including participant age and ethnicity. Because the majority of the sample (99%) was of white or African American descent, we created a dichotomous ethnicity variable coded as 1 for white and 2 for Other. Finally, we asked parents to report on the number of people living in the family home (including immediate family, extended family, and nonfamily members) for each year of the child's life, to ensure that any associations were not simply an artifact of family size.

Procedures

On arriving at our laboratory, a research assistant described the study procedures in detail. Parents were interviewed about family SES and household size for each year of the child's life. Adolescents were seated in an individual testing room, and the three EKG electrodes were applied. The BP cuff was placed on the upper aspect of the participant's right arm with the microphone placed above an area where the brachial artery could be palpated. After a 15-minute adaptation period while instruments were calibrated, the 10-minute baseline period was begun, and BP and heart rate were monitored and averaged during the final 5 minutes.

Statistical Analyses

In preliminary analyses, we examined the distribution of study variables and screened for outliers. In each of the SBP and HR distributions, there was a score greater than 3 standard deviations from the sample mean. These scores were replaced with the next highest score in their respective distributions. We also conducted bivariate analyses to assess the relationship between study variables and potential confounds. In the first wave of analyses, we modeled trajectories of SES across childhood by using a SAS procedure called TRAJ (28) that separates individuals into trajectory groups. TRAJ is a semiparametric, group-based modeling strategy that identifies clusters of individual trajectories. Model estimation produces posterior probabilities of membership in each trajectory group for each participant. These probabilities are then used to assign individuals to the trajectory group to which they have the highest probability of belonging. In the first part of the analysis, we determined the number of trajectories that best represented patterns of SES in our sample. Next, we looked at the parameter estimates to determine the shape of each trajectory. We used these to determine whether each trajectory was linear, quadratic, or cubic. Finally, we examined the relationship between trajectory group membership and adolescents' biological outcomes.

The TRAJ analyses allowed us to look at overall patterns of SES across childhood and certain combinations of the life-course models specified in the introduction. This set of analyses could be thought of as an inductive empirical approach based on what the data reveal about the types of life-course patterns that are present in this sample. However, the trajectory analyses suffered from two limitations—they did not provide a direct comparison of specific life-course models, and they were not amenable to adjustment for potential confounders. Thus, the second wave of analyses used partial correlations to directly compare models of early-life SES, current SES, cumulative SES, and social mobility. For these analyses, we calculated four specific indices of SES: 1) Early-life SES was calculated by averaging the number of bedrooms in the family home across the first 3 years of the child's life (mean = 3.1, SD = 0.8). 2) Current SES was the number of bedrooms in the family's current home (mean = 3.8, SD = 0.8). 3) Cumulative SES was the average number of bedrooms in the family home through the child's life (mean = 3.5, SD = 0.7). 4) Slope of SES (an indicator of social mobility) was the slope of the number of bedrooms in the family home through the child's life, calculated by regressing number of bedrooms on years of life separately for each participant (mean = 0.05, SD = 0.06). We then computed parallel indices based on the number of people in the home for each year of the child's life (i.e., early-life number of people, current number of people, cumulative number of people, and slope of the number of people). Given that each number of bedrooms index was correlated with the parallel family size index (r values ranged from 0.35–0.64, $p < .001$), we decided to include these variables, along with gender, race, and age, as covariates in regression analyses. All p values reported are based on two-tailed tests.

RESULTS

Trajectories of SES and Biological Outcomes

Model Selection: Identifying the Number and Shape of Trajectories

To test the hypothesis that we would identify distinct trajectories of family SES, we first modeled the trajectory patterns of our sample. The Bayesian Information Criterion (BIC) continued to increase as the number of trajectories increased, indicating a better model fit with the addition of each trajectory: The BIC was −1564.86 for 3 groups, −1406.64 for 4 groups, and −1310.65 for 5 groups. To prevent the trajectory group sizes from getting too small, we did not exceed the 5-group model. Both the 4- and 5-group models yielded similar information. For parsimony, we retained the 4-trajectory model.

The shape of each trajectory was determined by initially including linear, quadratic, and cubic parameters for each trajectory, and then dropping the nonsignificant ones (Table 2). A parameter estimate divided by its standard error results in a t statistic, which is used to determine statistical significance. The shape of each trajectory is identified by the highest order term included in the model. In Model 1, linear, quadratic, and cubic parameters were included for each of the four trajectories. The cubic parameters were nonsignificant for each. Thus in Model 2, the cubic parameters were dropped from each trajectory. The quadratic parameter was nonsignificant for the first, second, and fourth trajectories. Thus in Model 3, the quadratic parameters were dropped from these trajectories. Because it had the highest BIC coefficient, we adopted Model 3 as our final model: the first, second, and

TABLE 2. Model Selection for the Shape of Each Trajectory

Model and Parameter	Trajectory 1		Trajectory 2		Trajectory 3		Trajectory 4		BIC
	Function Tested	Parameter Estimate	Function Tested	Parameter Estimate	Function Tested	Parameter Estimate	Function Tested	Parameter Estimate	
1	Cubic		Cubic		Cubic		Cubic		−1406.64
Intercept		1.84		2.85		3.25		4.31	
Linear		0.11		0.039		0.13**		0.082	
Quadratic		0.00		−0.002		−0.002		−0.002	
Cubic		0.00		0.00		0.00		0.00	
2	Quadratic		Quadratic		Quadratic		Quadratic		−1392.00
Intercept		1.83		2.85		3.23		4.32	
Linear		0.11***		0.036		0.15***		0.079**	
Quadratic		0.00		−0.001		−0.006***		−0.001	
3	Linear		Linear		Quadratic		Linear		−1382.06
Intercept		1.86		2.92		3.23		4.37	
Linear		0.11***		0.014**		0.15***		0.058***	
Quadratic						−0.006***			

BIC = Bayesian Information Criterion.
** $p < .01$, *** $p < .001$.

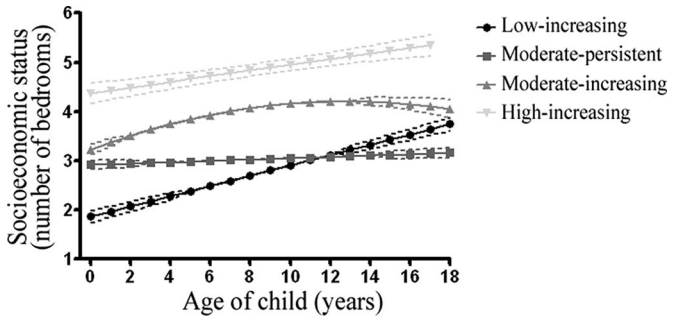

Figure 1. Estimates of SES trajectories and their accompanying 95% confidence interval from the child's birth to current age.

fourth trajectories were linear, and the third trajectory was quadratic.

As shown in Figure 1, the first trajectory group, which comprised 33% of the sample, showed the lowest SES in the child's early life. However, SES increased steadily through childhood and surpassed the next lowest group by early adolescence. We called this group "low-increasing." The second trajectory group, accounting for 21% of the sample, showed moderate SES at the time of the child's birth and did not change over the course of childhood. We called this group "moderate-persistent." A third trajectory group, accounting for 36% of the sample, showed moderate SES at the child's birth; however, SES improved through childhood and leveled off around early adolescence. We referred to this group as "moderate-increasing." Finally, a fourth trajectory group, comprised of 10% of the sample, showed the highest SES at the time of the child's birth and increased slightly with time. We called this group "high-increasing." It should be noted that trajectory group assignment is based on best fit; so, adolescents in each group do not follow exactly that trajectory.

Relating the Trajectories to Biological Outcomes

Next, we tested the hypothesis that SES trajectories would be differentially related to adolescent health outcomes. To test whether SBP differed by trajectory group, SBP was entered into the 4-group trajectory model specified above. This analysis yielded estimates of the average SBP and their corresponding standard errors for each trajectory group. The estimated average SBP was 114.2 mm Hg (SE = 1.9) for the low-increasing group, 106.8 mm Hg (SE = 1.6) for the moderate-persistent group, 108.3 mm Hg (SE = 1.5) for the moderate-increasing group, and 106.4 mm Hg (SE = 2.9) for the high-increasing group (Figure 2). Next, a Wald test was used to test the equality of the trajectory group SBP estimates. The Wald test is a χ^2-based test conducted on the variance estimates of the estimated trajectory group averages (29). First, an overall Wald test was used to test the null hypothesis that all groups were equal on SBP. Results indicated significant trajectory group differences in SBP ($\chi_3^2 = 10.17, p < .05$). Thus, pair-wise comparisons were tested (see Table 3 for a summary of trajectory group comparisons on SBP). The pattern of results indicated that the low-increasing group had higher SBP compared with the moderate-persistent, moderate-increasing, and high-increasing groups, all of which had similar SBP.

Next, we assessed trajectory group differences in DBP by entering DBP into the 4-group trajectory model. The estimated average DBP was 63.43 mm Hg (SE = 1.3) for the low-increasing group, 59.4 mm Hg (SE = 1.1) for the moderate-persistent group, 60.2 mm Hg (SE = 1.0) for the moderate-increasing group, and 56.4 mm Hg (SE = 1.9) for the high-increasing group (Figure 2). The overall Wald test was then used to test the equality of the trajectory group means on DBP. Results indicated significant group differences in DBP ($\chi_3^2 = 10.19, p < .05$). Thus, pair-wise comparisons were tested (Table 3). The pattern of results indicated that the low-increasing group had elevated DBP compared with the

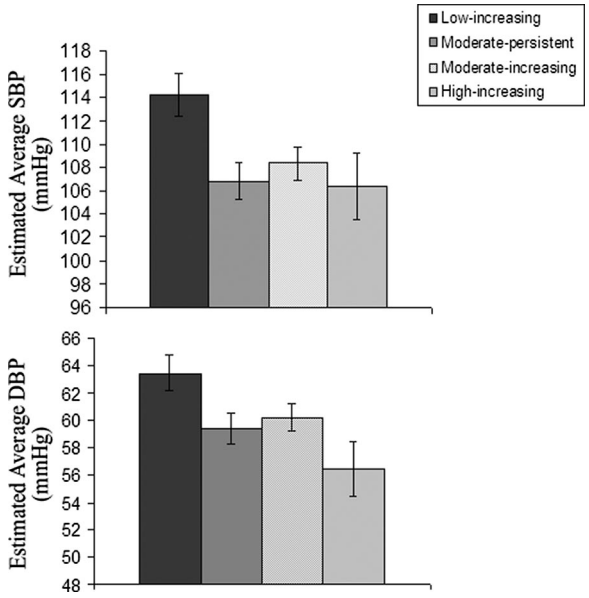

Figure 2. Estimates of average SBP and DBP by trajectory group. The error bars represent standard error of the mean. The estimated average SBP for the low-increasing group is significantly higher than the estimated averages for the moderate-persistent, moderate-increasing, and high-increasing groups. The estimated average DBP for the low-increasing group is significantly higher than the estimated averages for the moderate-persistent, moderate-increasing, and high-increasing groups.

TABLE 3. Trajectory Group Differences in Systolic Blood Pressure (SBP) and Diastolic Blood Pressure (DBP)

Trajectory Group Comparison	χ^2	
	SBP	DBP
Low-increasing vs. moderate-persistent	8.62**	5.24*
Low-increasing vs. moderate-increasing	5.94*	3.78*
Low-increasing vs. high-increasing	5.11*	9.03**
Moderate-persistent vs. moderate-increasing	0.51	0.30
Moderate-persistent vs. high-increasing	0.01	1.82
Moderate-increasing vs. high-increasing	0.34	2.99

* $p < .05$, ** $p < .01$.

moderate-persistent, moderate-increasing, and high-increasing groups.

Next, we tested trajectory group differences in HR and BMI. However, the overall Wald tests indicated no significant group differences in the estimated averages for these outcomes (p values $>.20$).

Life-Course Models of SES and Biological Outcomes

Partial correlation analyses were used to test specific life-course models of early SES, current SES, cumulative SES, and social mobility. All analyses controlled for age, race, gender, and number of people in the family home.

Correlations Among SES Indices

Early SES, current SES, and cumulative SES were significantly associated with one another (r values ranging from 0.51–0.84, p values $<.001$). Slope of SES was negatively related to early-life SES ($r = -0.44$, $p < .001$), positively related to current SES ($r = 0.49$, $p < .001$), and unrelated to cumulative SES ($p > .10$).

Partial Correlations Between SES Indices and Biological Outcomes

We first tested each type of SES measure separately for its ability to predict biological outcomes. We tested the critical period model by estimating partial correlations between early-life SES and biological outcomes, controlling for age, race, gender, and number of people in the family home. Lower early-life SES was associated with higher SBP and higher DBP ($r = -0.25$, $p < .01$ and $r = -0.26$, $p < .01$, respectively). Early-life SES was unrelated to HR and BMI ($r = 0.10$, $p > .20$ and $r = -0.09$, $p > .20$, respectively).

The second timing model proposes that current SES is what is most important to current adolescent health. Thus, we tested the associations between current SES and biological outcomes. Lower current SES was associated with higher DBP ($r = -0.20$, $p < .05$). Current SES was unrelated to SBP, HR, and BMI (r values ranging from -0.07 to 0.00, p values $>.10$).

To investigate relationships between dynamic SES and health, we first tested the model that cumulative SES would predict biological outcomes. Analyses indicated that lower cumulative SES was associated with higher SBP and DBP ($r = -0.20$, $p < .05$ and $r = -0.26$, $p < .01$, respectively). Cumulative SES was unrelated to HR and BMI ($r = 0.00$, $p > .20$ and $r = -0.08$, $p > .10$, respectively).

A second conceptualization of relationships between dynamic SES and health is that change in SES will affect adolescent health. To test this hypothesis, we correlated slope of SES with adolescent health outcomes. Analyses indicated that higher slope of SES was associated with higher SBP and BMI ($r = 0.23$, $p < .05$ and $r = 0.20$, $p < .05$, respectively). Slope of SES was unrelated to DBP and HR ($r = 0.05$, $p > .20$ and $r = -0.13$, $p > .20$, respectively).

Which Life-Course Model Best Explains Adolescents' BP?

Further partial correlation analyses were conducted to compare the effects of early-life SES, current SES, cumulative SES, and slope of SES on SBP and DBP. BMI was not included in these analyses because it correlated with only one SES index (slope of SES). HR was not included in these analyses because there were no significant associations. Analyses were done in pairs such that the correlation of one SES index with BP partialed out the effects of the other SES index, as well as age, race, gender, and number of people in the family home.

Given that early SES, cumulative SES, and slope of SES were all significantly related to SBP, we tested whether one SES index contributed to the prediction of SBP over and above the others. The results (Table 4) indicated that early-life SES predicts SBP independent of the effects of both cumulative SES and slope of SES.

TABLE 4. Partial Correlation Analyses Relating Socioeconomic Status (SES) Indices to Adolescent Systolic Blood Pressure (SBP) and Diastolic Blood Pressure (DBP)

Outcome	SES Indices	*r*
SBP	Early life SES Cumulative SES	−0.20* 0.06
	Early life SES Slope of SES	−0.19† 0.13
	Cumulative SES Slope of SES	−0.19† 0.25*
DBP	Early life SES Current SES	−0.20* −0.04
	Early life SES Cumulative SES	−0.12 −0.04
	Cumulative SES Current SES	−0.17 0.04

Partial correlations were done in pairs such that the test of one SES index partialed out the effects of the other.
Only those SES indices that were significantly associated with DBP and SBP were included in this set of analyses. SES indices are based on the number of bedrooms in the family home through childhood.
* $p < .05$, † $p = .06$.

Next, we compared the effects of early SES, current SES, and cumulative SES on DBP. These analyses indicated (Table 4) that early-life SES predicts DBP independent of current SES, but not cumulative SES.

DISCUSSION

The current study examined patterns of SES throughout childhood and their relationship to adolescents' SBP, DBP, HR, and BMI. The goal was to determine which life-course models best explained the relationship between SES experiences across an adolescent's life span and markers of current CV health.

Our first objective was to determine whether distinct trajectories of SES, as defined by number of bedrooms, would be evident in our sample. Analyses revealed four distinct trajectories of family SES: low-increasing, moderate-persistent, moderate-increasing, and high-increasing. Adolescents in the low-increasing group experienced low early-life SES and upward mobility through childhood. Adolescents in the moderate-persistent group experienced moderate early-life SES that remained stable over time. Adolescents in the moderate-increasing group experienced moderate early-life SES and upward mobility that leveled off in early adolescence. Finally, adolescents in the high-increasing group experienced high early-life, cumulative, and current SES.

Our next goal was to determine whether these trajectories differentially related to markers of CV risk. In respect to SBP, adolescents in the low-increasing group had the highest SBP of any of the trajectory groups. The difference between the low-increasing group and each of the other groups was quite large (around 7 mm Hg). In adult populations, SBP drops of this magnitude in middle-aged men are associated with a 16% reduction in first occurrences of major CVD over the following 10 years (30). We found a similar pattern of findings for DBP in that adolescents in the low-increasing group had the highest DBP of any of the groups. These findings suggest that upward mobility in the context of low early-life SES is particularly costly for children. However, from these analyses it remains unclear as to what is driving the effect—low early-life SES or subsequent upward mobility. Future work should settle this issue by directly comparing a low-increasing group to a low-persistent group. The results would have important theoretical implications—in terms of whether the difficulties associated with climbing up the social ladder outweigh the benefits of increased status—and potential ramifications for policies toward groups such as first and second generation immigrants.

Next, we performed direct tests of each of the life-course models. This approach allowed us to disentangle the effects of low early-life SES and upward mobility through childhood. Results indicated that both early-life SES and slope of SES (an indicator of mobility) were significantly and positively related to SBP. However, when we examined the impact of each SES index controlling for the effects of the other, only early-life SES was significantly related to SBP. Furthermore, early-life SES predicted SBP independent of cumulative SES. With respect to DBP, slope of SES was unrelated to DBP, and early-life SES predicted DBP independent of current SES. In sum, both the trajectory and partial correlation analyses provided support for the early-life model, and some hints that upward mobility may be important, too, for BP. However, the effects for upward mobility were less robust, as they emerged for SBP alone, and disappeared after controlling for early-life SES. Thus, of the life-course SES models we studied, the early critical period account best depicts BP in adolescence.

These findings suggest insights into how SES in early life comes to influence CV morbidity and mortality. They show that by the time children reach adolescence, the detrimental influences of low early-SES on CV health are already apparent. It may be that during critical periods of early-life low SES increases exposure to social and physical "pollutants"—such as crowding, violence, malnutrition, allergens—that have the capacity to program patterns of biological functioning over the long term (31). In rodents, this biological embedding of social experience results in differential adulthood regulation of stress-response systems such as the hypothalamic-pituitary-adrenocortical and sympathetic adrenal medullary axes (4,32,33). And recent evidence in humans suggests that early-life SES may get programmed in a way that favors the emergence of a pro-inflammatory phenotype in adolescence (34). This tendency could contribute to the disparities in BP we observed. Finally, early-life experiences may also influence health behaviors through the life course. For instance, early-life SES is associated with CV risk factors such as cigarette smoking, binge drinking, and obesity in adulthood (35,36).

In contrast, current SES was unrelated to adolescents' BP after controlling for the effects of early SES experiences. This finding is consistent with past research showing that SES is

unrelated to BP among adolescents. In general, studies in this area have shown that SES effects on BP are evident in childhood and adulthood, but not in adolescence (3). West (37) has suggested that SES differences in children's health diminish in adolescence due to a common youth culture in school that crosses socioeconomic lines. Thus current SES models may be more useful for explaining the relationship between SES and BP among populations of children and adults, rather than among adolescents.

We found little support for dynamic SES measures such as cumulative SES and social mobility as independent determinants of BP. However, it may be that cumulative SES is more important among lower SES families, who were underrepresented in our sample. For instance, the accumulation of SES experiences may lead to BP changes among children with low-persistent trajectories. Furthermore, our sample showed very little variability in social mobility; so, this dimension might be more salient in populations with varying degrees of upward and downward mobilities. In addition, it may be important to use measures of SES that are more sensitive to SES fluctuations (e.g., family income) and dynamic measures of health. For instance, changes in BP may occur closely in time with fluctuations in SES; so, without a repeated-measures design, increases and decreases in BP would get washed out over time. Finally, it is possible that the benefits of upward mobility or the costs of low cumulative SES in childhood are not apparent until adulthood. Future research should exam the relationship between trajectories of SES over the course of development and adult health.

Counter to our expectations, we found no association between trajectories of SES and adolescents' BMI and HR. These findings are inconsistent with past studies showing that lower SES is associated with higher BMI among adolescents (38,39). Recent evidence suggests that family and neighborhood resources have independent effects on adolescents' BMI (40). Thus, it may be important to take neighborhood characteristics into account when examining the relationship between family housing and BMI. Given that HR and BP are both controlled by the autonomic nervous system, we would expect to find a similar pattern of results for these outcomes. Thus, it is unclear why early-life SES would shape DBP and SBP, but not HR.

There are a number of limitations to this study that should be noted. First, assignment into trajectory groups is probabilistic in nature. In each trajectory group, some adolescents had a high probability of membership in that group, whereas others had a lower probability of membership in that group. Although this probabilistic assignment means that the trajectory groups should not be taken as real entities, any uncertainty in group membership is factored into the BP comparisons. Second, the study would have benefited from a more thorough examination of CV risk, including measures of cigarette smoking, physical activity, cholesterol, and triglycerides. Third, our outcome measures were limited by one-time laboratory assessment. Resting BP in the laboratory can be influenced by numerous factors, such as the white-coat effect. These problems are mitigated to a large extent by ambulatory BP measures, which should be used in the next wave of studies. Fourth, there may be problems associated with the measurement of SES via number of bedrooms. Specifically, this measure does not take into account whether families rent or own, which neighborhood they live in, or the costs of renting versus owning in their neighborhood. However, this imperfect indicator of SES should only diminish power to detect relations with BP. Future studies that collect a broad array of SES data prospectively, or the use of archival data such as social security contributions, would help resolve these difficulties. Fifth, trajectory group membership may be associated with birth order. Specifically, first-borns may be more likely to fall into the low-increasing group, as their parents may start out in a home that accommodates a small family and then move into larger homes as the family grows. Finally, given that our sample comprises predominantly upper middle class families, our findings should not be generalized across SES levels.

Despite these issues, findings from the current study indicate that, of the life-course models, timing of SES exposure plays the most important role in adolescents' BP. Specifically, SES experiences early in life were associated with adolescents' SBP and DBP, independent of current SES. These findings point toward early-life developmental processes as potential candidates for explaining the relationship between SES and risk factors related to CVD. In addition, our results suggest that interventions designed to reduce SES health disparities would be most effective if they had taken place within the first few years of a child's life.

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