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Life course SES and cardiovascular risk: Heterogeneity across race/ethnicity and gender

Katrina M. Walsemann, Ph.D., MPH^a, Bridget J. Goosby, Ph.D.^b, and Deeonna Farr, MPH^a

^aDepartment of Health Promotion, Education, and Behavior, Arnold School of Public Health, University of South Carolina, Discovery I, 915 Greene Street, Room 529, Columbia, SC 29208, USA

^bDepartment of Sociology, University of Nebraska-Lincoln, 741 Oldfather Hall, Lincoln, NE 68588, USA

Abstract

We examine four life course models as they relate to adolescent SES, adult SES, and cardiovascular risk – the sensitive period, pathways, accumulation, and social mobility models. Accounting for race/ethnic and gender differences in life course processes, we analyzed Waves I and IV of the National Longitudinal Study of Adolescent to Adult Health, a nationally representative sample of individuals enrolled in grades 7–12 when they were first interviewed in 1994/5. We restricted our sample to whites, blacks, and Latinos who were interviewed in Waves I and IV and provided biomarker data (n=11,397). The cardiovascular risk score at Wave IV combined waist circumference, blood pressure, hemoglobin A1c, and C-reactive protein. We found evidence for each of the four life course models for white women, whereas the sensitive period was indicated for white men. Upward mobility was also associated with higher CVD risk among white men as compared to those who were socio-economically advantaged at both time points. The pathway model was significant for Latino women. No life course models were significant for black men or women or Latino men. Our findings demonstrate the importance of applying an intersectional lens to understanding CVD risk over the life course.

Keywords

CVD; race/ethnicity; gender; life course; SES

The socio-economic gradient in health is well established; individuals with higher socio21 economic status (SES) report better health and greater longevity than individuals with lower SES (Elo, 2009). One of the most consistent SES gradients is found for cardiovascular risk and disease (CVD) (Glymour, Clark, & Patton, 2014). Life course researchers have posited

*Correspondence should be sent to Katrina M. Walsemann, Department of Health Promotion, Education, and Behavior, Arnold School of Public Health, University of South Carolina, Discovery I, 915 Greene Street, Room 529, Columbia, SC 29208, Phone: 1-803-777-1904, Fax: 1-803-777-6290, kwalsema@sc.edu.

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the importance of early life exposures for later adult health (Ferraro & Shippee, 2009, Kuh, Ben-Shlomo, Lynch, Hallqvist, & Power., 2003), and a large body of evidence now exists to support the link between child SES and adult CVD risk (Karlamangla et al. 2005, Murray et al. 2011, Pollitt, Rose, & Kaufman 2005).

The protective nature of higher SES for preventing or forestalling health problems is not equivalent for blacks and Latinos compared to whites, nor is it likely to be the same for men and women (Pearson, 2008; Walsemann, Gee, & Ro, 2013; Williams, Mohammed, Leavell, & Collins, 2010). Prior studies have found that the adult SES gradient in health is most consistently found for white men, whereas it is often weaker or non-existent for racial/ethnic minorities and women (Kimbro, Bzostek, Goldman, & Rodriguez, 2008; Walsemann et al., 2013). Most CVD studies, however, fail to consider CVD risk at the intersection of race/ethnicity *and* gender. This is an important omission as social class, race/ethnic, and gendered experiences may shape life course health.

Applying a life course perspective, we use data from the National Longitudinal Study of Adolescent to Adult Health to examine how SES measured in adolescence (ages 12–18) influences CVD risk in early adulthood (ages 24–34). Furthermore, using SES measured in early adulthood, we assess the extent to which additional life course processes are evident. Central to our research question is the explicit examination of these life course processes within the three largest racial/ethnic groups in the U.S. – whites, blacks, and Latinos. We also consider if, within race/ethnicity, gender differences in these life course processes emerge.

BACKGROUND

Socioeconomic status is not static and is likely to change over the life course with varying health consequences based on timing and duration of exposure to different levels of SES. Though the timing and duration of exposure to socioeconomic disadvantage can have long-term health consequences, they need not be inexorable. The concepts of timing, duration, and modifiability are captured in four frameworks common in life course and health research: the *sensitive period model*, the *pathways model*, the *accumulation model*, and the *social mobility model*. A number of scholars have cogently articulated the interdependent nature of these four frameworks (Hamil-Luker & O'Rand, 2007; Murray et al., 2011; Pudrovska & Anikputa, 2014). Rather than viewing them as competing theories that should be examined independently, these authors argue that they should be considered within the same analytic model in order to provide a more comprehensive and nuanced understanding of how life course SES influences health. We follow these recommendations by exploring each of these life course frameworks for CVD risk.

Sensitive period

A sensitive period refers to a developmental window of time when exposure to a given event or experience exerts a stronger effect than it would if it occurred during a different developmental window (Kuh et al., 2003). Importantly, the health consequences of a sensitive period exposure can be modified or reversed by subsequent changes in the exposure or through intervention (Ben-Shlomo & Kuh, 2002). Consistent with a sensitive

period model, a number of studies have found significant associations for early life SES on CVD risk independent of adult SES (Laitinen et al., 2013; Melchior, Moffitt, Milne, Poulton, & Caspi, 2007; Murray et al., 2011). For example, low child SES was associated with higher CVD risk among a cohort of 32 year-olds in New Zealand. This association persisted after adjustment for adult SES (Melchior et al., 2007). Another study of UK adults found that this model best described the association between child SES and adult CVD risk among men (Murray et al., 2011). Finally, in a systematic review of life course SES and CVD risk, Pollitt and colleagues (2005) concluded that a moderate level of support exists for the sensitive period model. As it relates to our study, SES in adolescence may reflect a sensitive period given the confluence of changing social contexts and pubertal development – it may exert a stronger effect on CVD risk than adult SES. Empirically, this indicates that adolescent SES would be inversely and significantly associated with CVD risk after adjustment for adult SES.

Pathways model

The pathways model acknowledges the importance of adolescent SES for adult health, but posits that the effect of adolescent SES is mostly indirect, influencing adult health by placing individuals on a given life trajectory that reflects a combination of social, economic, and health behaviors that are protective of and/or detrimental to health (Hamil-Luker & O'Rand, 2007; Kuh et al., 2003; Pollitt et al., 2005). That is, individuals who experience social or economic disadvantage in youth are at greater risk of experiencing additional disadvantages that increase their odds of CVD risk in adulthood. For example, disadvantaged youth are more likely to experience restricted educational opportunities that reduce their chances of attending and completing college (Walsemann et al., 2013). Lower educational attainment is associated with lower occupational status and earnings (Elo, 2009) that in turn increase the risk of experiencing financial strain, which is independently related to poorer mental and physical health (Kahn & Pearlin, 2006).

In addition to adult SES and financial strain, health behaviors may also partially explain the association between adolescent SES and adult CVD risk, although this aspect is understudied. According to Cockerham (2013), decisions to engage in certain health behaviors over others reflect a health lifestyle that is based on a set of options available to individuals according to their life chances, or social class. The social class of origin seems to be important for establishing *health* lifestyles, particularly because health behaviors are often established in adolescence (Chassin et al., 1996; Telama et al., 2005). Thus, in the pathways model, adult SES, financial strain, and health behaviors are conceptualized as mediators that connect adolescent SES to adult CVD risk (Pudrovska & Anikputa, 2014). Though studies support the pathways model linking child SES to adult CVD risk via adult SES (Blane et al., 1996; Gruenewald et al., 2012), few studies consider the role of financial strain or health behaviors – potentially important pathways linking child SES to adult CVD risk (Pollitt et al., 2005) – as we do in this study. Empirically, this model would be supported if adolescent SES was inversely and significantly associated with CVD risk when examined without adjustment for adult SES, financial strain, and health behaviors, and if the relationship was attenuated after adjustment for these factors.

Accumulation model

The accumulation model is a model of cumulative impact, meaning that exposure to socioeconomic disadvantage (or advantage) over multiple periods has a stronger impact on later health than an individual episode of socioeconomic disadvantage (or advantage). This concept is reflected in the weathering hypothesis, which contends that prolonged exposure to negative social, economic, and/or psychosocial conditions accelerates the aging of biological systems resulting in increased risk of disease at earlier ages in the life course (Geronimus, 2013).

The accumulation model finds the most consistent support in the CVD literature (Pollitt et al., 2005). Individuals who experience low SES at multiple time points have the highest CVD risk of any SES group, including inflammation (Pollitt et al., 2008), allostatic load (Gruenewald et al., 2012), type II diabetes (Stringhini et al., 2013), hypertension (James et al., 2006), coronary heart disease (Loucks et al., 2009), and intima-media thickness (Carson et al., 2007). Empirically, this model has been examined as the additive or interactive effect of adolescent and adult SES. Thus, in our study, the accumulation model would be supported if adolescent SES *and* adult SES were inversely and significantly associated with CVD risk or if, in an interactive model, the main effects and the interaction were all inversely and significantly associated with CVD risk.

Social mobility

The social mobility model primarily emphasizes how life course patterns of SES influence health. Central to the social mobility model is the question – do our bodies remember or can they be made to forget? This model recognizes the importance of adolescent SES for CVD risk, but also allows for the possibility that the health effects of adolescent SES may be modified or reversed by later socioeconomic circumstances (Ferraro & Shippee, 2009). Thus, individuals who are upwardly mobile – experienced adolescent socioeconomic disadvantage, but adulthood socioeconomic advantage – may experience lower CVD risk than chronically disadvantaged individuals. Conversely, downwardly mobile individuals – experienced adolescent socioeconomic advantage, but adulthood socioeconomic disadvantage – may see the erosion of early life health advantages as a consequence of later disadvantage. Thus, the health of socially mobile individuals is expected to fall in-between the health of those who are consistently advantaged or consistently disadvantaged (Högberg, Cnattingius, Lundholm, Sparén, & Iliadou, 2012).

In the CVD literature, support for the social mobility model has been mixed (Pollitt et al., 2005). Gruenewald et al. (2012) and James et al. (2006) found a gradient in the relationship between life course SES and CVD risk such that those who were consistently disadvantaged reported the worst health outcomes and those who were consistently advantaged reported the best health outcomes. Individuals who were downwardly mobile and upwardly mobile fell in-between. Conversely, social mobility was unrelated to hypertension among a national sample of black Americans (Broman, 1989). Health behaviors may partially explain this relationship. Upwardly mobile individuals may have spent a considerable part of their life engaging in health behaviors that place them at higher CVD risk, whereas the converse may be true for downwardly mobile individuals. Further, if adolescent health behaviors are

established and difficult to modify, upwardly mobile individuals may continue engaging in more unhealthy behaviors as compared to consistently advantaged individuals, placing the upwardly mobile at higher CVD risk. Thus, changes in health behaviors that accompany (or do not accompany) changes in SES may mediate the life course SES and CVD risk relationship.

Similar to the accumulation model, the social mobility model is empirically examined by including the main and interactive effects of adolescent and adult SES. To see the expected relationship described above, the main effect of both SES measures (adolescent *and* adult) and the interaction term must be inversely and significantly associated with CVD risk *or* the main effect of adult SES and the interaction term must be inversely and significantly associated with CVD risk.

Differences across Race/Ethnicity and by Gender

Inequality occurs across multiple systems of stratification, yet most studies interested in the relationship between life course SES and CVD risk consider only the independent effects of SES, race/ethnicity, and gender (Berkman, 2005), if they are considered at all. Indeed, what we know about life course SES and CVD risk comes mainly from racially homogenous samples based in Europe, the UK, and New Zealand (Högberg et al., 2012; Melchior et al., 2007; Murray et al., 2011; Pollitt et al., 2005), many of which are not generalizable to women or persons outside of the labor force (Blane et al., 1996; Stringhini et al. 2013). Moreover, U.S. studies heavily rely on community samples that are not representative of the adult population and often only include white and/or black respondents (Carson et al., 2007; Gruenewald et al., 2012; James et al., 2006; Lemelin et al., 2009; Pollitt et al., 2008; Roberts et al., 2010). Latinos have been virtually ignored within this body of research. An intersectional approach recognizes that neither the meaning of SES, race/ethnicity, and gender, nor their associated health outcomes can be understood apart from the other (Weber & Fore, 2007). Empirically, this requires the joint, simultaneous assessment of SES, race/ethnicity, and gender on CVD risk across the life course.

There are strong theoretical reasons to expect that life course processes in CVD risk may manifest differently within racial/ethnic groups and by gender. First, SES is not equivalent across race/ethnicity or gender; the meaning of education, for example, and its associated economic and health benefits are often greater for white men than for racial/ethnic minorities or women (Walsemann et al., 2013; Williams et al., 2010). This may translate into intergenerational effects such that the average economic benefits of growing up in a household with a white male wage earner will be greater than growing up in a household where the wage earner is a black woman, for example. A child's gender may also interact with her parents' economic circumstances. Hamil-Luker & O'Rand (2007) found that women growing up in less advantaged households did not accumulate as much human, social, and health capital as their male counterparts. Others document less social mobility for women than men (Warren, Sheridan, & Hauser, 2002). Together, these findings suggest that adolescent SES may have a more profound effect on women, and that white women may benefit the most from adolescent socioeconomic advantage, although this is not consistently found as it relates to CVD risk. For example, using a community sample of young adults,

Karlamangla and colleagues (2005) found that the association between child SES and CVD risk was strongest in white women and weakest in black men. Conversely, child SES was more strongly associated with CVD risk among white men than women in the UK (Murray et al., 2011).

Second, upward mobility in the United States has varying levels of psychosocial and health costs depending on race/ethnicity and gender. Building upon the theory of “John Henryism” (James, 1994), Pearson (2008) argues that individuals from racially marginalized groups who actively resist the institutionalized discrimination that they encounter when attempting to secure economic and cultural capital ultimately pay a health penalty. Thus, the relationship between life course SES and CVD risk may not follow the expected pattern found for white men, since racial/ethnic minorities and women often must contend with structural racism and/or sexism that may diminish the positive health benefits of securing a higher SES.

Research Questions and Hypotheses

We have two main research questions. First, what life course processes best describe the SES and CVD risk relationship among white, black, and Latino young adults? We hypothesize that there will be stronger evidence supporting the four life course processes previously described among whites, and weaker evidence for blacks and Latinos given prior research that finds a weaker SES-health gradient among blacks and Latinos (Kimbrow et al., 2008; Williams et al., 2010). Second, within race/ethnicity, does gender modify the life course SES and CVD risk relationship? We hypothesize that evidence for the four life course processes will vary by gender; although given inconsistent findings in the literature we do not hypothesize a direction of effect.

DATA AND METHODS

Data

Restricted data from Waves I (1994/5) and IV (2008/8) of the National Longitudinal Study of Adolescent to Adult Health (Add Health; Harris, 2013) were used. The Add Health sample is nationally representative of U.S. adolescents who were enrolled in grades 7–12 in 1994/5 with respect to region of country, urbanicity, school size, school type (private/public), and race/ethnicity. Our analysis used data from in-home interviews of respondents in Waves I and IV, along with Wave IV biomarker data. Biomarker data collection used non-invasive procedures and included the collection of anthropometric, cardiovascular, metabolic, and inflammatory measures. Metabolic and inflammatory measures were based on validated analysis of dried blood spots (McDade, Williams, & Snodgrass, 2007).

Our sample was restricted to those assigned probability weights in Wave IV, who were not pregnant at the time of the interview, and who self-reported as non-Hispanic white, non-Hispanic black, or Latino (n=13,171). Approximately 1,774 respondents were excluded due to item non-response on biomarkers, resulting in a final analytic sample of 11,397 respondents (6,943 non-Hispanic whites, 2,527 non-Hispanic blacks, and 1,927 Latinos).

Measures

Dependent Variable—Four established risk factors for cardiovascular disease were used to construct the dependent variable – cardiovascular risk score (hereafter, CVD risk): waist circumference, blood pressure, hemoglobin A1c (HbA1c), and C-reactive protein (CRP) (Emerging Risk Factors Collaboration, 2010; May, Kuklina, & Yoon, 2012). Each indicator was categorized as 0 = “low risk”, 1 = “medium risk”, and 2 = “high risk” based on established cut-points that represent clinically significant demarcations for CVD risk (Chobanian et al., 2003; Lean, Han, & Morrison, 1995; Lorenzo et al., 2010; Salazar et al., 2014). Measured waist circumference was categorized as 0 = <94 cm (men) or <80 cm (women), 1 = 94 cm – <102 cm (men) or 80 – <88 cm (women), and 2 = ≥102 cm (men) or ≥88 cm (women). Measured diastolic (DBP) and systolic (SBP) blood pressure were used to categorize respondents as 0=normotensive (<120 SBP & <80 DBP), 1=prehypertensive (120–139 SBP or 80–89 DBP), and 3=hypertensive (≥140 SBP or ≥90 DBP). Measured HbA1c was used to categorize respondents as 0=non-diabetic (<5.7%), 1=pre-diabetic (5.7–6.4%), and 2=diabetic (≥6.5%). CRP was categorized as 0 <1.0 mg/L, 1 = 1.0 – 3.0 mg/L, and 2 = >3.0 mg/L. The four indicators were summed, resulting in a normally distributed risk score ranging from 0 to 8. Additional details about the distribution of each of the four CVD risk factors are included in Appendix Table 1.

Independent Variables

Adolescent SES was constructed using a composite measure that was calculated using

standardized (z-score) measures ($\frac{z_i = x_i - \bar{X}}{SD}$) of family poverty (i.e., parent-reported household income to federal poverty level in 1995), parental educational level (i.e., parent-reported 10-level ordinal variable ranging from “did not go to school” to “professional training beyond a 4-year degree”), and parental occupation (i.e., respondent-reported 7-level ordinal variable ordered from low to high by adapting Erikson and Goldthorpe’s class schema (2002), all measured at Wave I. Scores were calculated for all respondents with information on at least one of the indicators. Respondents residing with one parent used information from that parent in constructing the measure, whereas respondents residing with two parents used the average of both parents’ information. Positive values represent higher levels of adolescent SES.

Similar to adolescent SES, a composite measure of *adult SES* at Wave IV was created. This measure was calculated as the mean of standardized (z-score) measures of family poverty, respondents’ educational level, and respondents’ occupation. Composite scores were calculated for all respondents who had information on at least one of the indicators used in the composite measure. Positive values represented higher levels of adult SES.

Mediators

Physical activity at Wave I was assessed using 3 items that met the average metabolic equivalent (MET) value of 5.0 or higher, representing moderate to vigorous activity (Ainsworth et al., 2011). The items were summed such that higher values represent greater physical activity. A similar measure was constructed for physical activity at Wave IV

following the approach described by Gordon-Larsen, Nelson, and Popkin (2004). *Change in physical activity* subtracted Wave I from Wave IV physical activity.

Smoking status at Wave IV was categorized as non-smoker, former smoker, non-daily smoker, and daily smoker.

Economic hardship at Wave IV is a count of 5 economic strains reported in the past 12 months: could not pay rent/mortgage, was evicted, could not pay electrical/gas/oil bills, electrical/gas/oil service was shut-off, or worried that food would run out.

Covariates

We include *nativity* (foreign-born to non-US citizens versus US born), *family structure* in Wave I (two biological parents, one biological and one step-parent, female-headed, extended family, and other), *age* in Wave IV, and *marital status* in Wave IV (married, cohabitating, never married, or other). *Self-rated health* at Wave I was a five-level ordinal variable ranging from 1=poor to 5=excellent. Finally, because CRP results may be biased upward if individuals experienced a recent infection (McDade, Rutherford, Adair, & Kuzawa, 2010), the number of inflammatory or common *infections* reported in the prior two weeks at Wave IV was controlled in all models and was coded as 0, 1, 2, and 3 or more infections.

Analyses

To address issues of item non-response, we imputed data for all covariates (but not the dependent variable) using the *mi impute* command with chained equations specification in Stata v13. This produced 50 data sets. Analyses were replicated across the 50 datasets and combined using *mi estimate*. These analyses were also weighted to account for the complex sampling design and respondent attrition from Wave I to Wave IV using the *svy* commands and the sampling weights provided by Add Health. All analyses were stratified by race/ethnicity. Descriptive statistics were further stratified by gender.

RESULTS

Sample Characteristics

Among whites, men had significantly lower CVD risk ($M=3.3$, $SE=0.05$) than women ($M=3.5$, $SE=0.04$; Table 1). No gender differences were found in adolescent SES; however, in adulthood white men reported lower SES ($M=-0.1$, $SE=0.04$) than white women ($M=0.2$, $SE=0.03$). Similarly, among blacks, men had significantly lower CVD risk ($M=3.7$, $SE=0.08$) than women ($M=4.3$, $SE=0.08$). Both black men and women experienced levels of adolescent SES below the mean of the sample ($M=-0.3$, $SE=0.08$ and $M=-0.4$, $SE=0.06$, respectively), indicating greater disadvantage in adolescence. This continued in adulthood; but black men reported significantly lower adult SES ($M=-0.4$, $SE=0.07$) than black women ($M=-0.2$, $SE=0.06$). Among Latinos, CVD risk was statistically similar between men ($M=3.7$, $SE=0.09$) and women ($M=3.9$, $SE=0.11$). Latino men and women experienced levels of adolescent SES below the mean ($M=-0.6$, $SE=0.06$ and $M=-0.5$, $SE=0.07$, respectively), indicating a more disadvantaged adolescence than their peers. Latino men

reported lower SES in adulthood ($M=-0.3$, $SE=0.04$) than Latino women ($M=-0.0$, $SE=0.05$). Additional characteristics are presented in Table 1.

Multivariable Analyses

Table 2 presents estimates from weighted multivariable regression models stratified by race/ethnicity. All models adjust for nativity, family structure, self-rated health, age, marital status, and number of infections in the prior two weeks. Model 1 estimates the direct effect of adolescent SES on CVD risk. Model 2 tests the sensitive period and pathways model by simultaneously estimating the effects of adolescent SES and adult SES. Model 3 tests the social mobility and accumulation models by including an interaction between adolescent SES and adult SES. Model 4 tests whether health behaviors and financial strain mediate the relationship between life course SES and CVD risk. In each model, we consider if the effects differ by gender by including interaction terms between gender and the respective SES indicators.

Whites

In Model 1, adolescent SES ($b=-0.38$) was significantly associated with lower CVD risk among white men and women. Adolescent SES remained significantly associated with CVD risk among white men ($b=-0.38$, Model 2) after adjustment for adult SES, but this association was reduced for white women ($b=-0.23$ [$-0.38+0.15$]) with the inclusion of adult SES. Adult SES was associated with lower CVD risk only among white women ($b=-0.20$ [$-0.02+-0.18$]). Post-hoc tests indicated that the reduction in the adolescent SES coefficient was statistically significant for white women ($p<0.01$). Model 2, therefore, provides support for the sensitive period model for white men and women and the pathways model for white women.

In Model 3, the interaction between adolescent SES and adult SES was negative and significant ($b=-0.18$), and this association did not differ by gender. We plot the main effects and interactions in Figures 1 (women) and 2 (men). For illustrative purposes, we present estimates at low (-1 SD), average (0 SD), and high ($+1$ SD) SES. We find support for social mobility and accumulation models among white women. The CVD risk score was 3.56 for white women disadvantaged at both life course periods, 3.38 for upwardly and 3.36 for downwardly mobile women, and 2.74 for women advantaged at both developmental periods. In some respects, the opposite pattern was found for white men. Upwardly mobile white men had the highest CVD risk score ($y=3.75$), whereas downwardly mobile white men had a lower CVD risk score ($y=2.97$) than the consistently disadvantaged ($y=3.41$), but a higher score than the consistently advantaged ($y=2.63$). This pattern emerged because of the significant main effect of adolescent SES and the non-significant main effect of adult SES among white men. These patterns did not change with inclusion of the mediators (Model 4).

Blacks

Among black men, adolescent SES was not associated with CVD risk when modeled without adjustment for adult SES (Model 1), but adolescent SES was inversely associated with CVD risk among black women ($b=-0.30$ [$0.07+-0.37$]). With adjustment for adult SES (Model 2), adolescent SES was no longer associated with CVD risk for black women, but

neither was adult SES associated with CVD risk among black men or women. The inclusion of the two-way (adolescent SES×adult SES) and three-way interactions (adolescent SES × adult SES × female) in Model 3 did not alter these findings, nor did the inclusion of mediators (Model 4). Thus, none of the life course models were supported among black young adults.

Latinos

Adolescent SES was not associated with CVD risk among Latino men when modeled without adjustment for adult SES (Model 1) or after adjustment for adult SES (Model 2). Adult SES was also unrelated to CVD risk among Latino men. No gender differences were found in these estimates; however, a Wald test indicated that the main effect of adolescent SES was significantly different from zero among Latino women ($b=-0.31$, $SE=0.11$; $p<0.05$) in Model 1 as was the main effect of adult SES in Model 2 ($b=-0.41$, $SE=0.16$; $p<0.05$). With adjustment for adult SES, the main effect of adolescent SES among Latino women was no longer significant ($b=-0.18$, $SE=0.14$; $p>0.05$; Model 2). This provides support for the pathways model among Latino women. Estimates from Model 3 suggest no evidence for the social mobility or accumulation models among Latino men or women. Inclusion of mediators did not alter these findings (Model 4).

Sensitivity Analyses

We re-estimated Model 4 by including a variable that captured transitions in obesity between Waves I and IV. This variable was not a significant mediator (as determined by a Sobel test) and was thus excluded given its high correlation with waist circumference at Wave IV, a component of the CVD risk score. We also considered if self-reports of height or weight discrimination mediated the relationship between life course SES and CVD risk since obesity-related discrimination may be a factor in limiting upward mobility, particularly for white women. We found no evidence for mediation. Given that some respondents were enrolled in school at Wave IV, we estimated mean adult SES by enrollment status. Respondents enrolled in school at Wave IV reported higher SES in adulthood regardless of race/ethnicity or gender.

DISCUSSION

Increasing attention has been paid to the relationship between life course SES and cardiovascular risk. Yet, few studies have considered how the relationship between life course SES and cardiovascular risk intersects with race/ethnicity and gender. Using data from Add Health, we systematically investigated four life course frameworks linking life course SES to early adult cardiovascular risk – the sensitive period, pathways, accumulation, and social mobility models. We found evidence for all four life course frameworks, but the extent to which these frameworks were supported varied across race/ethnicity and gender.

We hypothesized that the strongest evidence for the four life course frameworks would be found among whites. Although this hypothesis was corroborated in part, we also discovered gender differences in the extent to which these life course processes manifested for whites. Among white men, adolescent SES was strongly associated with adult CVD risk, but the

association was null for adult SES. Further, upwardly mobile white men did not receive a health benefit from their upward mobility and actually reported higher CVD risk than consistently disadvantaged white men. Conversely, downwardly mobile white men continued to receive a health benefit from living in high SES families during adolescence. The significance of child SES for CVD risk among white men has been documented in the UK and Sweden (Högberg et al., 2012; Murray et al., 2011), but no studies to our knowledge have found a reverse social mobility effect for white men.

Conversely, all four life course models were supported for white women. Our results are consistent with prior studies that have found support for the sensitive period model (Högberg et al., 2012; Karlamangla et al., 2005), the accumulation model (Carson et al., 2007; Murray et al., 2011), and the social mobility model (Högberg et al., 2012; Karlamangla et al., 2005) in white women, with relationships between life course SES and CVD risk or hypertension occurring in the anticipated direction. Further, in our study, adult SES was more strongly associated with CVD risk for white women than men, which mirrors findings from research on CVD and hypertension (Colhoun, Hemingway, & Poulter 1998; Karlamangla et al., 2005). In part, the stronger life course SES patterns for white women and the reverse mobility effects for white men in our sample may be due to the measurement of SES. Indeed, many CVD studies use only one indicator to measure SES (Melchoir et al., 2007; Murray et al., 2011) and some use different indicators of adult SES relative to child SES (Lemelin et al., 2009; Stringhini et al., 2013). In contrast, our SES measure combines the three most commonly used SES indicators into an index and assesses SES similarly in adolescence and adulthood. Thus, our SES indicators may more fully capture economic, social, and human capital influences on CVD risk as compared to prior studies. It is also possible that white women in the Add Health cohort were more successful than earlier cohorts in translating their education into economic and health benefits. Indeed, in our sample, white women reported higher SES in early adulthood than white men.

We found no support for any of the four life course frameworks among black men or women. Our results substantiate empirical evidence that SES is either unrelated (Karlamangla et al., 2005) or positively related (Lemelin et al., 2009) to CVD risk among black men. Our results contradict those of Karlamangla and colleagues (2005), who found that upward social mobility was inversely associated with change in CVD risk among black women and results by Carson and colleagues (2007) who report an inverse association between cumulative SES and intermedial thickness and peripheral artery disease for black women. Both studies used community samples, however, and may have more limited generalizability than our sample.

Our findings are consistent with theoretical arguments that the benefits of securing economic and cultural capital may not outweigh the health costs black individuals pay for engaging with institutions that often racially discriminate against them (Pearson, 2008). Evidence suggests, for example, that discrimination exposure increases with increasing SES, with higher SES blacks experiencing markedly more *acute* racially discriminatory events during adulthood than lower SES blacks as a consequence of greater immersion into predominantly white environments (Hudson, Neighbors, Geronimus, & Jackson, 2015). The fact that there were no resultant health benefits of upward mobility for black men or women

in terms of lowered CVD risk points to the very real possibility that chronic life course exposure to racism is not easily overcome through hard work and places black men and women at significant early adult CVD risk (c.f., Gee, Walsemann, & Brondolo, 2012; Goosby & Heidbrink, 2013). Thus, we suggest that race related stressors not captured in our SES measures may explain the null findings and may be an important avenue for future investigation.

We found that the pathways model best explained the relationship between life course SES and CVD risk for Latino women, but that life course SES was unrelated to CVD risk among Latino men. Our findings suggest that for Latino women, SES in adolescence and adulthood play an important role in shaping early adult CVD risk. Other studies, however, find a weaker SES gradient in health among Latinos depending on nativity and ethnicity (Kimbrot et al., 2008; Pearson, 2008). In post-hoc analysis, we identified different life course processes by nativity; the pathways model best explained the life course SES and CVD risk relationship among foreign-born women, whereas evidence of a reverse social mobility effect was found among US-born Latinos, regardless of gender. That is, upwardly mobile US-born Latinos experienced greater CVD risk than those who were consistently advantaged. These findings raise the intriguing and important idea that SES may have different health effects depending on developmental period, nativity, and gender.

Several limitations warrant mention. First, we were only able to assess SES at two points in the life course. The relationship between race/ethnicity, gender, and life course SES may change as individuals enter mid-life and the benefits of adult SES accumulate or, conversely, as individuals transition from receiving economic assistance from their parents in early adulthood (particularly among whites) to providing economic assistance to their parents in mid-life. Still, our study investigates an important, but often neglected, life course period – early adulthood – in the establishment of CVD risk. This is especially true given that issues of selective mortality can bias our understanding of life course processes if we only focus our study of CVD risk on mid441 life and older adults. Second, our sample represents individuals born between 1974 and 1983; thus, our results can only be generalized to this population. Third, we were unable to disaggregate Latinos by ethnicity or nativity due to small sample sizes. Because life course SES patterns may be masked by such heterogeneity, it is imperative that future studies investigate this possibility.

This study provides a vital addition to the larger body of work examining how life course SES influences CVD risk. Specifically, our study uncovers marked variations in the degree to which life course SES defines CVD risk among U.S. young adults, not only by race/ethnicity, but also by gender. By applying an intersectional lens, our study reiterates the need for incorporating nuance into the contributions of socioeconomic standing for the development of CVD across diverse U.S. populations.

Supplementary Material

Refer to Web version on PubMed Central for supplementary material.

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Research Highlights

- We examined the relationship between life course SES and early adult CVD risk
- We considered gender differences within white, black, and Latino sub-groups
- Data come from the National Longitudinal Study of Adolescent to Adult Health
- Life course processes for CVD risk differed by race/ethnicity and gender
- Applying an intersectional lens is important for understanding life course CVD risk

Figure 1.
White Women

Figure 2.
White Men

Notes: Estimates based on Model 4 from Table 2. “Low” refers to 1 SD below the mean of SES. “Average” refers to mean SES (0 SD). “High” refers to 1 SD above the mean of SES.

Table 1

Sample characteristics by race/ethnicity and gender, National Longitudinal Study of Adolescent to Adult Health, W1 & W4, Mean (SE) or % presented, weighted estimates.^a

	Whites N=6,943 ^b		Blacks N=2,527		Latinos N=1,927	
	Women	Men	Women	Men	Women	Men
Cardiovascular Risk Score	3.5 (0.04)	3.3 (0.05)*	4.3 (0.08)	3.7 (0.08)*	3.9 (0.11)	3.7 (0.09)
Adolescent SES	0.1 (0.03)	0.1 (0.04)	-0.4 (0.06)	-0.3 (0.08)*	-0.5 (0.07)	-0.6 (0.06)
Adult SES	0.2 (0.03)	-0.1 (0.04)*	-0.2 (0.06)	-0.4 (0.07)*	-0.0 (0.05)	-0.3 (0.04)*
Mediators						
Physical Activity W1	3.4 (0.07)	4.1 (0.08)*	2.8 (0.11)	4.1 (0.11)*	3.0 (0.09)	4.1 (0.13)*
W4 – W1 Physical Activity	-1.7 (0.07)	-2.3 (0.08)*	-1.5 (0.13)	-2.1 (0.11)*	-1.5 (0.10)	-2.2 (0.14)*
Smoking Status W4 (%)						
Never Smoker	27.0	23.9	58.8	39.7*	46.5	34.4*
Former Smoker	31.6	31.2	16.8	18.3	29.1	27.6
Non-Daily Smoker	12.9	13.9	12.4	18.5	13.6	22.2*
Daily Smoker	28.5	31.0	12.0	23.5*	10.8	15.8
Economic Hardship W4	0.4 (0.03)	0.4 (0.02)*	0.7 (0.06)	0.6 (0.05)	0.6 (0.06)	0.3 (0.04)*
Control Variables						
Nativity (%)						
Born US Citizen	99.6	99.6	99.1	99.2	81.5	79.6
Born Non-US Citizen	0.4	0.4	0.9	0.8	18.5	20.4
Family Structure W1 (%)						
Nuclear	52.6	55.6	22.3	21.1	36.8	45.1
Step-Family	10.5	11.2	6.0	6.8	10.1	5.3
Female Headed	13.8	12.5	25.3	27.0	11.1	11.5
Extended	17.8	14.2*	40.8	39.1	37.7	31.6
Other	5.3	6.5	5.6	6.0	4.3	6.5
Self-Rated Health W1	3.8 (0.02)	4.0 (0.02)*	3.8 (0.04)	4.0 (0.05)*	3.7 (0.07)	3.9 (0.05)

	Whites N=6,943 ^b		Blacks N=2,527		Latinos N=1,927	
	Women	Men	Women	Men	Women	Men
Age (years) W4	28.7 (0.13)	28.9 (0.14)*	29.0 (0.22)	29.3 (0.23)*	28.9 (0.25)	29.0 (0.23)
Marital Status W4 (%)						
Married	51.0	42.6*	24.6	26.9	45.4	38.8
Cohabiting	19.8	20.0	24.1	23.3	18.3	15.5
Never Married	24.9	33.7*	46.8	46.7	31.9	41.9*
Other	4.3	3.7	4.5	3.1	4.4	3.8
# of Infections W4	0.5 (0.02)	0.4 (0.01)*	0.4 (0.03)	0.4 (0.02)	0.5 (0.04)	0.4 (0.03)*

Notes:

^a Higher values = greater physical activity, greater economic hardship, and better self-rated health.

^b Unweighted sample size.

* $p < 0.05$, adjusted for multiple comparisons using Bonferroni method.

Table 2

Regression coefficients from multivariable linear regression, stratified by race/ethnicity, weighted analysis, National Longitudinal Study of Adolescent to Adult Health, W1 & W4

	Model 1 b (SE)	Model 2 b (SE)	Model 3 b (SE)	Model 4 b (SE)
Whites (n=6,943)				
Adolescent SES	−0.38 (0.06) **	−0.38 (0.08) *	−0.42 (0.08) **	−0.39 (0.08) **
Female × Adolescent SES	0.06 (0.08)	0.15 (0.09)	0.20 (0.09) *	0.18 (0.09)
Adult SES		−0.02 (0.06)	−0.01 (0.06)	−0.00 (0.06)
Female × Adult SES		−0.18 (0.09) *	−0.19 (0.08) *	−0.20 (0.09) *
Adolescent SES × Adult SES			−0.18 (0.07) *	−0.17 (0.07) *
Female × Adolescent SES × Adult SES			0.06 (0.10)	0.06 (0.10)
Female	0.08 (0.05)	0.11 (0.06) *	0.09 (0.06)	0.07 (0.06)
Intercept	3.39 (0.04) **	3.39 (0.04) **	3.44 (0.05) **	3.19 (0.06) **
Blacks (n=2,527)				
Adolescent SES	0.07 (0.12)	0.04 (0.14)	0.13 (0.12)	0.14 (0.12)
Female × Adolescent SES	−0.37 (0.15) *	−0.31 (0.17)	−0.40 (0.16) *	−0.39 (0.15) *
Adult SES		0.09 (0.13)	0.17 (0.14)	0.12 (0.13)
Female × Adult SES		−0.12 (0.15)	−0.21 (0.17)	−0.23 (0.16)
Adolescent SES × Adult SES			0.24 (0.18)	0.23 (0.17)
Female × Adolescent SES × Adult SES			−0.27 (0.22)	−0.26 (0.21)
Female	0.49 (0.10) **	0.46 (0.11) **	0.51 (0.13) **	0.39 (0.12) **
Intercept	3.73 (0.08) **	3.75 (0.09) **	3.72 (0.10) **	3.65 (0.10) **
Latinos (n=1,927)				
Adolescent SES	−0.20 (0.13)	−0.17 (0.13)	−0.20 (0.12)	−0.20 (0.12)
Female × Adolescent SES	−0.10 (0.16)	−0.00 (0.19)	0.01 (0.19)	0.05 (0.19)
Adult SES		−0.07 (0.14)	−0.19 (0.16)	−0.22 (0.16)
Female × Adult SES		−0.30 (0.24)	−0.15 (0.31)	−0.12 (0.31)
Adolescent SES × Adult SES			−0.20 (0.14)	−0.21 (0.13)
Female × Adolescent SES × Adult SES			0.26 (0.24)	0.29 (0.23)
Female	0.10 (0.19)	0.15 (0.19)	0.12 (0.22)	0.06 (0.22)
Intercept	3.60 (0.11) **	3.59 (0.11) **	3.62 (0.11) **	3.35 (0.12) **

Notes: All models adjust for nativity, family structure W1, self-rated health W1, age W4, marital status W4, and # of infections W4. Model 4 further adjusts for physical activity W1, change in physical activity between W4 and W1, smoking status W4, and economic hardship W4. All covariates centered at their grand mean, except for gender.

*
 $p < 0.05$;

**
 $p < 0.01$