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

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ABSTRACT

We examine four life course processes as they relate to adolescent SES, adult SES, and cardiovascular risk – sensitive periods, pathways model, accumulation model, and social mobility. We consider differences in these life course processes across race/ethnicity and by gender. We analyzed Waves I and IV of the National Longitudinal Study of Adolescent Health, restricting our sample to whites, blacks, and Latinos. Analyses were stratified by race/ethnicity. Cardiovascular risk score at Wave IV combined four risk factors: waist circumference, blood pressure, hemoglobin A1c, and C-reactive protein. All four life course processes were present for white women, but only the sensitive period was indicated for white men, Latino men, and Latina women. The sensitive period and the accumulation model were supported for black women. No life course processes were significant for black men. Our findings demonstrate the importance of nuanced examinations of structural factors for CVD risk over the life course.

The socio-economic gradient in health is well established; individuals with higher socio-economic status (SES) report better health and greater longevity than individuals with lower SES (Adler and Stewart 2010, Eide and Showalter 2011, Kawachi, Adler and Dow 2010). One of the most consistent SES gradients is found for cardiovascular risk and disease (CVD) (Glymour, Clark and Patton 2014, Kanjilal et al. 2006, Kaplan and Keil 1993, Sharma et al. 2004, Thurston et al. 2014). Life course sociologists and epidemiologists have for decades posited the importance of early life and childhood exposures for later adult health (Ferraro and Shippee 2009, Kuh et al. 2003, Luo and Waite 2005, Umberson et al. 2014, Walsemann, Geronimus and Gee 2008), 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 and Kaufman 2005). Such conclusions, however, may be premature, as most of these studies were either based on racially homogenous samples outside of the United States or on community samples that may not be generalizable to the United States adult population (Pollitt, Rose and Kaufman 2005). Indeed, few studies consider how race/ethnicity or gender may alter the relationship between SES and CVD across the life course. A life course perspective recognizes the important role that race/ethnicity and gender play in structuring individuals' lived experiences from conception to death (Gee, Walsemann and Brondolo 2012, Geronimus 2013, Umberson et al. 2014). We approach our study from this perspective to suggest that the relationship between life course SES and CVD risk may manifest differently within race/ethnicity and by gender.

The meaning of SES and its importance for preventing or forestalling health problems is not equivalent for blacks and Latinos as it is for whites, nor is it likely to be the same for men and women (Pearson 2008, Walsemann, Gee and Ro 2013, Williams et al. 2010). Prior studies have found, for example, that the adult SES gradient in health varies in strength across race/ethnicity or gender (Ailshire and House 2012, Farmer and Ferraro 2005, Montez, Hummer and Hayward 2012, Ross, Masters and Hummer 2012); however, most studies fail to consider CVD risk at the intersection of race/ethnicity *and* gender. Applying a life course perspective, we use data from the National Longitudinal Study of Adolescent Health to examine how SES measured in adolescence (ages 12-18) influences CVD risk in early adulthood (ages 26-34). 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 United States – 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 of exposure to different levels of SES. The life course perspective provides a beneficial framework for understanding the importance of timing for health (Elder, Kirkpatrick Johnson and Crosnoe 2003, George 2014, Taylor 2008). In this study, we examine the life course tenet of time by examining the extent to which the timing and duration of exposure to socioeconomic disadvantage can impact cardiovascular disease risk. 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 the modifiability of prior exposures are captured in four conceptual frames common in the life course and health literature: the sensitive period; the pathways model; the accumulation model; and social mobility. A number of scholars have

cogently articulated the interdependent nature of these four conceptual frames (Hamil-Luker and O'Rand 2007, Murray et al. 2011, Pudrovska and Anikputa 2014, Rosvall et al. 2006). Rather than viewing them as competing theories that should be examined independently, these authors argue that these four frames 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 four life course frames and recognizing the interrelatedness of these life course processes for CVD risk.

Sensitive periods

A sensitive period refers to a developmental window of time when exposure to a given event, circumstance, or experience exerts a stronger effect than it would if it occurred during a different developmental window (Kuh et al. 2003). In this regard, a sensitive period is similar to the concept of critical period with two exceptions. First, in the critical period model, an exposure outside of a given developmental window produces no additional disease risk unlike a sensitive period model where the effect is merely stronger in a given developmental window than outside of it. Second, the long-term health consequences of a given exposure during a sensitive period can be modified or reversed, whereas if the exposure follows a critical period model, there is a very limited window of opportunity to intervene and modify the consequences of the behavior (Ben-Shlomo and Kuh 2002). This is an important distinction both conceptually and empirically, although these concepts are often used interchangeably. In considering which model might be operating on a given health outcome, both the exposure and the timing of the exposure are essential factors. As it relates to our study, SES in adolescence is likely to follow a sensitive period model – it may exert a stronger effect on CVD risk than adult SES, but subsequent increases or declines in SES may modify adult CVD risk.

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. For example, low child SES was associated with higher risk of 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 a "critical period" model seemed to best describe 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.

Pathways model

The pathways model, also known as a "chains of risk" model, acknowledges the importance of early life SES for adult chronic disease, but posits that the effect of early life SES is mostly indirect, influencing adult chronic disease 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 and O'Rand 2007, Kuh et al. 2003, Pollitt, Rose and Kaufman 2005). That is, individuals who experience social or economic disadvantage in youth are at greater risk of experiencing additional disadvantages, such as restricted educational opportunities that reduce their chances of attending and completing college. They are also more likely to smoke (Fagan et al. 2005, Soteriades and DiFranza 2003), be obese (Clarke et al. 2010, Walsemann et al. 2012, Wardle et al. 2006), and be physically inactive (Ferreira et al. 2007), all behavioral risk indicators associated with CVD. Thus, in this model, adult SES and health behaviors are conceptualized as mediators that connect early life

SES to adult CVD risk (Pudrovska and Anikputa 2014). Empirically, this model would be supported if early life SES was associated with CVD risk when examined without adjustment for adult SES and adult health behaviors, but would be attenuated after adjustment for adult SES and health behaviors.

A number of studies find evidence to support the pathways model, although it is difficult to fully ascertain the strength of the evidence because most studies are only able to consider SES at two points in the life course and often do not consider the potential mediating role of health behaviors (Pollitt, Rose and Kaufman 2005). For example, in a national sample of middle-aged US adults, there was a stronger association between adult SES and allostatic load compared to child SES, although child SES remained a significant predictor of allostatic load once adult SES was adjusted (Gruenewald et al. 2012). A study of employed Scottish men found that although child SES and adult SES were independently associated with a number of CVD risk factors, the strength of the association was stronger for adult SES than for child SES (Blane et al. 1996). Using a community sample of US adults 45 to 64 years old, Roberts and colleagues (2010) found that although child SES was associated with incident heart failure, after adjustment for SES in early and mid-to-older adulthood, the association was attenuated. Taken together, findings from these studies suggest that much of the association between early life SES and CVD risk may work indirectly through adult SES.

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 model 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 cumulative damage to biological systems brought on by long-term exposure to low SES across the life course may play a significant role in CVD risk (Kuh et al. 2003, Murray et al. 2011).

Within the CVD literature, the most common way to assess the accumulation model is to sum or average SES across different time points, usually childhood and adulthood (Murray et al. 2011). This reflects an additive approach to testing this model. That is, each period of low SES has similar, independent effects on CVD risk (Mishra et al. 2013, Pudrovska and Anikputa 2014). This, however, may not be the most accurate way to conceptualize cumulative impact, especially if the effects of earlier disadvantage on CVD risk are amplified with additional exposure to disadvantage. To empirically examine this scenario, interactions between SES at separate time points would be needed.

The accumulation model appears to find the most consistent support in the CVD literature (Pollitt, Rose and Kaufman 2005). Individuals who experience low SES at multiple time points – usually at least during childhood and adulthood – have the highest likelihood of CVD risk, including inflammation (Loucks et al. 2006, Pollitt et al. 2008), allostatic load (Gruenewald et al. 2012), hypertension (James et al. 2006), coronary heart disease (Loucks et al. 2009), and intima-media thickness (Carson et al. 2007).

Social mobility

The social mobility model is most interested in examining how life course patterns of SES influence health, and in our case, CVD risk. Central to the social mobility model is the question – do our bodies remember or can they be made to forget? This model recognizes that 1) early life SES is an important factor in CVD risk; 2) the health effects of early life SES may be modified or reversed by later socioeconomic circumstances; and 3) adult CVD risk is a result of a life-long process that is influenced by social structure, human agency, and underlying physiological processes (Ferraro, Shippee and Schafer 2009, Ferraro and Shippee 2009). Thus, individuals who are upwardly mobile – experienced socioeconomic disadvantage in childhood, but socioeconomic advantage in adulthood – may experience lower CVD risk than individuals who experience chronic socioeconomic disadvantage. Conversely, individuals who are downwardly mobile – experienced socioeconomic advantage in childhood, but socioeconomic disadvantage in adulthood – may see the health advantages that they experienced earlier in life erode as a consequence of later disadvantage. As a result, the health of socially mobile individuals likely falls somewhere in-between the health of those who are consistently advantaged or consistently disadvantaged (Bartley and Plewis 1997, Högberg et al. 2011).

One of the most common approaches to studying social mobility is to examine life course patterns of SES and compare patterns of mobility to patterns of cumulative advantage or disadvantage (Gruenewald et al. 2012, Högberg et al. 2011, James et al. 2006). In the CVD literature, the findings have been mixed (Pollitt, Rose and Kaufman 2005). For example, both Gruenewald et al. (2012) and James (2006) found a gradient in the relationship between life course SES and CVD risk such that those who were low SES in childhood and adulthood reported the worst health outcomes and those who were high SES in childhood and adulthood 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).

Social mobility could also be empirically assessed by creating an interaction between SES at two or more time points, as would be the case when examining the accumulation model. This reflects the model's emphasis on how changes in SES across the life course may be important for CVD risk (Pudrovska and Anikputa 2014). Thus, the focus in the social mobility model would be on those individuals who change their SES over the life course. In comparison, the focus of the accumulation model is on the stability of SES – those who are persistently advantaged or disadvantaged.

Differences across Race/Ethnicity and by Gender

Inequality occurs across multiple systems of stratification (Ferraro and Shippee 2009, Weber 2010), yet most studies interested in the relationship between life course SES and CVD risk ignore other forms of stratification, such as gender and race/ethnicity (Berkman 2005), or assume that they contribute additively to CVD risk. Indeed, what we know about how life course SES influences CVD risk comes mainly from racially homogenous studies based in Europe, the UK, and New Zealand (Högberg et al. 2011, Melchior et al. 2007, Murray et al. 2011, Pollitt, Rose and Kaufman 2005), many of which are not generalizable to women or persons outside of the labor force (Blane et al. 1996, Stringhini et al. 2013). Further, U.S. based studies tend to 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. Thus, current understanding of how life course SES shapes CVD risk may be incorrect, especially as it applies to US blacks, Latinos, and women.

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 or gender; the meaning of education, for example, and its associated outcomes are quite different for white males than they are for any other group. Women, on average, earn less money at an equivalent level of education than men; whereas Black and Latino men, on average, earn less money at an equivalent level of education than White men (Walsemann, Gee and Ro 2013, Williams et al. 2010). Likewise, the quality of education that one receives differs across race/ethnicity; predominantly-minority schools tend to be underfunded, which results in fewer educational opportunities for their students (Darling-Hammond 2004). Second, efforts to become socially mobile in the United States have varying levels of psychosocial and health costs depending on race/ethnicity and gender (Pearson 2008). According to Pearson (2008), individuals from marginalized groups who engage in high-effort psychological coping in an effort to secure economic and cultural capital ultimately pay a health penalty. For instance, studies have found that sustained, high-effort coping in the face of adversity is associated with increased risk of hypertension among Black men (James 1994). Finally, society structures the lives of men and women in distinct ways, which results in different opportunities, social roles, and norms (Moen and Chermack 2005). Thus, there is reason to suspect that the consequences of life course SES for CVD risk will depend upon gender.

Although few studies in the life course SES and CVD literature have considered how life course processes may vary within race/ethnicity and by gender, those that have report inconsistent results. For example, using a community sample of young adults, Karlamangla and colleagues (2005) found that the association between childhood SES and CVD risk was strongest in white women and weakest in black men. A study using a community sample of blacks and whites in the US found that indicators of cumulative SES and adult SES were strongly and inversely associated with inflammation among whites, whereas adult SES, but not cumulative SES, was inversely associated with inflammation among blacks (Pollitt et al 2008). Conversely, others have found no racial/ethnic and gender differences in the association between life course SES and CVD risk (c.f., (Roberts et al. 2010)).

Research Questions and Hypotheses

We have two main research questions. First, what life course processes best describe the relationship between SES and CVD risk in white, black, and Latino young adults? Based on prior research, we hypothesize that there will be stronger evidence in support of the four life course processes previously described among whites, and weaker evidence for blacks and Latinos. Second, within race/ethnicity, does gender modify the relationship between life course SES and CVD risk? Given the different locations held by women and men within social institutions (e.g., family, work, government) and the systematic inequities women experience within those social institutions (Hamil-Luker and O'Rand 2007, Moen and Chermack 2005), we hypothesize that life course processes will vary by gender.

METHODS

Sample

Restricted data from Wave I (1994/5) and IV (2008/8) of the National Longitudinal Study

of Adolescent Health (Add Health; Harris et al. 2009) were used. The Add Health sample is nationally representative of adolescents in grades 7-12 in Wave I from US schools 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 biomarker data collected in Wave IV. Biomarker data collection used non-invasive procedures, and included the collection of anthropometric (i.e., waist circumference), cardiovascular (i.e., blood pressure, pulse rate), metabolic (i.e., hemoglobin A1c) and inflammatory measures (i.e., C-reactive protein). Metabolic and inflammatory measures were based on the analysis of dried blood spots.

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 1,736 respondents were excluded due to item non-response on biomarkers and 137 were excluded due to item non-response on covariates. After exclusions, our final analytic sample consisted of 11,298 respondents.

Measures

Cardiovascular Risk: Four established risk factors for cardiovascular disease were used to construct the dependent variable – cardiovascular risk (CVR) score: waist circumference, blood pressure, hemoglobin A1c (HbA1c), and C-reactive protein (CRP). Each indicator was categorized as 0 = "low risk"; 1 = "medium risk"; and 2 = "high risk" based on established cut-points (Lean, Han and Morrison 1995). 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.

Adolescent SES was constructed using a composite measure calculated using indicators from Wave I. Standardized (z-score) measures 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) were used to calculate the mean composite score. Scores were calculated for all respondents with information on at least one of the indicators. Respondents residing with just one parent used information from that parent in constructing the measure while 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, when respondents were 26-34 years old. This measure was calculated as the mean of standardized (z-score) measures of family poverty (i.e., respondent-reported household income to federal poverty level in 2007), respondents' educational level (i.e., respondent-reported 9-level ordinal variable ranging from ''completed 8th grade or less'' to ''completed professional training beyond a 4-year degree''), and respondents' occupation (i.e., respondent-reported 7-level ordinal variable).

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). Items reported the number of times respondents participated in a variety of activities (e.g., rollerblading, bicycling, jogging, swimming, walking, dancing, etc.) in the past week with response options ranging from "not at all" to "5 or more times". The items were summed such that higher values represent greater physical activity. An indicator of physical activity at Wave IV was constructed to measure change in physical activity from Wave I to Wave IV; however, the Wave IV indicator used 6 items assessing engagement in moderate to vigorous activities. The 6 indicators were summed, then divided by 6 (representing the average score of individual physical activities) and multiplied by 3 to make the Wave IV indicator comparable to Wave I (Gordon-Larsen, Nelson, and Popkin 2004). *Change in physical activity* was calculated by subtracting Wave I from Wave IV physical activity. We include Wave I physical activity and change in physical activity from Wave I to IV in our analysis.

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

Depressive symptoms were measured at Wave I using the 9-item Center for Epidemiological Studies Depression Scale (CES-D) available in Add Health. Respondents were asked how often in the past week they had experienced 9 symptoms. Per convention, positively worded items were reverse coded and the 9 items were summed (Cronbach's α=0.XX), such that higher values represent greater depressive symptoms. C*hange in depressive symptoms* from Wave I to Wave IV were calculated by subtracting the 9-item CES-D scale measured at Wave I from the 9-item CES-D scale measured at Wave IV. Wave I depressive symptoms and change in depressive symptoms from Wave I to IV were used in our analysis.

Economic hardship at Wave IV is a count of the number 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.

Control Variables: Respondents were categorized as *immigrants* if they reported being born outside of the US to non-US citizens. *Family structure* in Wave I was categorized as nuclear (two biological parents), step-family (one biological and one step-parent), female-headed, extended/intergenerational family, and other. *Age* in Wave IV ranged from 24 to 34 years old. *Marital status* in Wave IV was categorized as unmarried, married, or other. *Baseline health* was assessed using the following question measured in Wave I: "In general, how is your health? Would you say excellent, very good, good, fair, or poor?" Higher values reflect better health. Finally, because CRP results may be biased upward if individuals experienced an infection recently, the number of inflammatory or common *infections* reported in the prior two weeks at Wave IV was controlled for, which was coded as 0, 1, 2, and 3 or more.

Analytic Approach

All analyses are stratified by race/ethnicity. We begin with descriptive statistics to understand the data distribution, which we further stratify by gender. Next, we present estimates of accumulation and social mobility within race/ethnicity for women and men. Multiple linear

regression assessed the association between life course SES and cardiovascular risk. All analyses were weighted to account for the complex sampling design and respondent attrition from Wave I to Wave IV using the *svy* commands in Stata v13.

RESULTS

Sample Characteristics

Table 1 presents sample characteristics across race/ethnicity for women and men. Among whites, men have a significantly lower CVD risk score (*M*=3.3) than women (*M*=3.5). No gender differences were found in adolescent SES; however, in adulthood white men reported lower SES (*M*=-0.1) than white women *(M*=0.2). Other gender differences of note include higher rates of physical activity in Wave 1 among men than women, but a steeper reduction in their physical activity between W1 and W4. White men also reported fewer depressive symptoms than white women (*M*=4.9 vs *M*=6.1), but no gender differences in the rate of change in depressive symptoms between W1 and W4. Men also endorsed fewer economic hardships than women.

Among blacks, men have a significantly lower CVD risk score (*M*=3.7) than women (*M*=4.3). Both black men and women experienced levels of adolescent SES that was below the mean of the sample (-0.2 and -0.3, respectively), indicating greater disadvantage in adolescence. This continued in adulthood; but black men reported significantly lower adult SES than black women. Similar patterns in gender differences in health behaviors were found among blacks as found among whites. Black men were more physically active at W1 than black women and also experienced greater declines in physical activity between W1 and W4 compared to black women. Black men reported fewer depressive symptoms at W1 than black women; whereas black women experienced a decline in depressive symptoms between W1 and W4 (*M*=-0.7), black men did not (*M*=0.2). Black men were also much more likely to be daily smokers than black women.

Among Latinos, CVD risk was statistically similar between men (M=3.7) and women (M=3.9). On average, Latino men and women experienced levels of adolescent SES below the mean (M=-0.5 and M=-0.4, respectively), indicating a more disadvantaged adolescence than their peers. This continued into adulthood; although Latinas were closer to the mean (M=-0.0) in adulthood. Men reported lower SES in adulthood than women (M=-0.3). Like whites and blacks, there were gender differences in physical activity among Latinos; men were more physically active at W1 than women, but experienced a greater decline in physical activity by W4. Non-daily smoking was also more common among men than women. Latino men also endorsed fewer economic hardships than women (M=0.3 vs M=0.6, respectively).

Social Mobility and Accumulation

Table 2 reports the percentage of respondents who experienced social mobility (upward and downward) as well as cumulative advantage (i.e., high adolescent SES & high adult SES) or cumulative disadvantage (i.e., low adolescent SES & low adult SES). Among whites, over 20% of women experienced cumulative advantage, whereas only 2.7% reported upward mobility from low adolescent SES to high adult SES, although almost 16% moved from average adolescent SES to high adult SES. Gender differences were found across most of the adolescent SES – adult SES categories. More men reported cumulative disadvantage than women, and fewer men than women reported cumulative advantage.

Among blacks, few gender differences were found. However, more blacks experienced cumulative disadvantage (17.4% women, 22.1% men) than cumulative advantage (7.0% women,

7.5% men). Upward mobility from low adolescent SES to high adult SES occurred among 5.6% of women and 2.8% of men, whereas downward mobility from high adolescent SES to low adult SES occurred among 5.5% of women and 8.3% of men.

Among Latinos, significantly more men (24.7%) experienced cumulative disadvantage than women (14.9%); however, this was the only gender difference found. Around 6% of women and men experienced cumulative advantage. Fewer reported downward social mobility from high adolescent SES to low adult SES (2.3% of women, 4.9% of men), whereas slightly more Latinos experienced upward mobility from low adolescent SES to high adult SES (8.6% of women, 7% of men).

Multivariate Analyses

Table 3 presents estimates from weighted multiple regression models stratified by race/ethnicity. All models adjust for gender, nativity, family structure (Wave I), self-rated health (Wave I), age (Wave IV), marital status (Wave IV), and number of inflammatory or common infections in the two weeks prior to the interview (Wave IV). Model 1 estimates the direct effect of adolescent SES on CVD risk. Model 2 estimates the direct effect of adult SES on CVD risk. Model 3 tests the sensitive period and pathways model by simultaneously estimating the effects of adolescent SES and adult SES. Model 4 tests the social mobility and accumulation models by including an interaction between adolescent SES and adult SES. In each model, we consider if the effects differ by gender by including interaction terms between gender and the respective SES indicators.

Whites

When modeled separately, adolescent SES (b=-0.38; Model 1) and adult SES (b=-0.15; Model 2) are significantly associated with lower CVD risk among white men and women. Model 3 suggests that after adjustment for adolescent SES, the relationship between adult SES and CVD risk is null for white men (b=-0.02), but is inversely associated with CVD risk among white women (b=-0.19). Further, adolescent SES remains significantly associated with CVD risk among white men (b=-0.38) after adjustment for adult SES, but the strength of the association is weaker for white women (b=-0.19 [-0.38+0.19]). Model 3, therefore, provides support for the sensitive period model for men and women, but not the pathways model for men.

In Model 4, the interaction between adolescent SES and adult SES is negative and significant, and this association does not differ by gender. We plot the main effects and interactions in Figure 1. Doing so reveals support for social mobility and the accumulation model among white women. For example, the CVD risk score is 3.8 for women disadvantage at both life course periods, 3.67 for upwardly mobile women, 3.71 for downwardly mobile women and 3.0 for women advantage at both developmental periods. In some respects, the opposite patterns are found among white men. Upwardly mobile white men have the highest CVD risk score (y=4.1), and downwardly mobile white men have the lowest (y=3.2). This likely reflects the strong sensitive period effect of adolescent SES for white men. These patterns do not change with inclusion of the mediators (Model 5).

Blacks

Neither adolescent SES nor adult SES is associated with CVD risk, when modeled individually (Model 1 and Model 2) or jointly (Model 3) for black men and women. The inclusion of the two-way (adolescent SES x adult SES) and three-way interactions (adolescent

SES x adult SES x female) in Model 4 indicates that, among black women, the accumulation model is tentatively supported, but the social mobility model is not (see Figure 2). We qualify this support because the association between adult SES and CVD risk as well as the interaction between adolescent SES and adult SES among black women were only marginally statistically significant. According to Figure 2, black women who were advantaged at both developmental periods experienced the lowest CVD risk (y=3.7), whereas black women who were disadvantaged at both life course periods experienced high CVD risk (y=4.5). This level of risk, however, was similar across women with low adolescent SES regardless of adult SES. None of the patterns for black men were statistically significant, suggesting that SES is not a strong indicator of CVD risk among this population. Inclusion of mediators (Model 5) did little to alter these patterns.

Latinos

Adolescent SES is inversely associated with CVD risk among Latino men and women (b=-0.20; Model 1), but adult SES is not (Model 2). This association held with adjustment for adult SES (Model 3), providing support for the sensitive period model, but not the pathways model. Estimates from Model 4 suggest no support for the social mobility model or accumulation model among Latino men or women. Inclusion of mediators did not alter these findings (Model 5).

DISCUSSION – (To be added)

Table 1: Sample characteristics by race/ethnicity and gender, National Longitudinal Study of Adolescent Health, W1 & W4, Mean (SE) or % presented. [a, b]

	White N=6,906		**Black** N=2,487		**Latino** N=1,905	
	Women	Men	Women	Men	Women	Men
Cardiovascular Risk Score	3.5 (0.05)	3.3 (0.05)*	4.3 (0.08)	3.7 (0.09)*	3.9 (0.09)	3.7 (0.09)
Childhood SES	0.1 (0.03)	0.1 (0.04)	-0.3 (0.06)	-0.2 (0.08)	-0.4 (0.07)	-0.5 (0.06)
Adult SES	0.2 (0.03)	-0.1 (0.04)*	-0.2 (0.06)	-0.4 (0.08)*	-0.0 (0.04)	-0.3 (0.05)*
Mediators						
Physical Activity W1	3.4 (0.07)	4.1 (0.08)*	2.8 (0.11)	4.1 (0.12)*	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	26.7	23.9	59.0	40.2*	45.4	33.6*
Former Smoker	31.7	31.3	16.6	18.0	29.8	28.0
Non-Daily Smoker	12.9	14.0	12.4	18.4	13.8	22.4*
Daily Smoker	28.7	30.9	12.0	23.4*	11.0	16.0
CES-D W1 [c]	6.1 (0.12)	4.9 (0.10)*	7.2 (0.22)	5.3 (0.17)*	7.4 (0.31)	5.7 (0.21)*
W4 – W1 CES-D [c]	-0.7 (0.13)	-0.4 (0.10)	-0.7 (0.27)	0.2 (0.30)*	-1.0 (0.33)	-0.6 (0.30)
Economic Hardship W4	0.4 (0.03)	0.4 (0.02)*	0.7 (0.06)	0.6 (0.05)	0.6 (0.05)	0.3 (0.04)*
Control Variables						
Nativity						
Born US Citizen	99.6	99.6	99.1	99.2	81.4	79.3
Born Non-US Citizen	0.4	0.4	0.9	0.8	18.6	20.7
Family Structure W1						
Nuclear	52.8	55.8	22.4	20.9	37.5	45.3
Step-Family	10.4	11.2	5.9	6.7	8.4	5.3
Female Headed	13.9	12.6	25.2	27.1	11.5	11.4
Extended	17.5	14.0*	40.8	39.1	38.2	31.8
Other	5.4	6.4	5.7	6.2	4.4	6.3
Self-Rated Health W1	3.8 (0.03)	4.0 (0.02)*	3.8 (0.04)	4.0 (0.05)*	3.7 (0.06)	3.9 (0.05)*
Age (years) W4	28.7 (0.13)	28.9 (0.14)*	28.9 (0.21)	29.3 (0.23)*	28.8 (0.24)	29.0 (0.23)
Marital Status W4						
Married	51.1	42.7*	25.0	26.5	46.2	39.4
Cohabitating	19.9	19.7	24.2	23.5	18.7	15.6
Never Married	24.7	33.9*	46.5	46.8	30.5	41.2*
Other	4.3	3.7	4.4	3.2	4.7	3.9
# of Infections W4	0.5 (0.02)	0.4 (0.02)*	0.4 (0.03)	0.4 (0.02)	0.5 (0.04)	0.4 (0.03)*

Notes: [a] Percentages may not add to 100 due to rounding. [b] Higher values = greater physical activity, more depressive symptoms, higher perceived social status, greater economic hardship, and better self-rated health. [c] CES-D = depressive symptoms scale, *$p<0.05$, adjusted for multiple comparisons using Bonferroni method.

Table 2: Percentage of respondents in each adolescent SES – adult SES category by gender, stratified by race, weighted estimates, National Longitudinal Study of Adolescent Health, W1 & W4.[a]

		White N=6,906		**Black** N=2,487		**Latino** N=1,905	
		Women	Men	Women	Men	Women	Men
Adolescent SES	Adult SES						
Low	Low	8.2	12.7*	17.4	22.1	14.9	24.7*
Low	Avg	9.6	5.2*	17.0	9.1*	23.0	18.2
Low	High	2.7	2.0	5.6	2.8	8.6	7.0
Avg	Low	7.6	16.7*	8.9	19.2*	8.3	10.2
Avg	Avg	21.6	17.3*	18.7	13.1	19.0	16.1
Avg	High	15.9	12.7*	10.1	8.8	10.8	6.5
High	Low	2.8	5.4*	5.5	8.3	2.3	4.9
High	Avg	11.3	11.8	9.8	9.1	6.9	6.1
High	High	20.3	16.2*	7.0	7.5	6.2	6.4

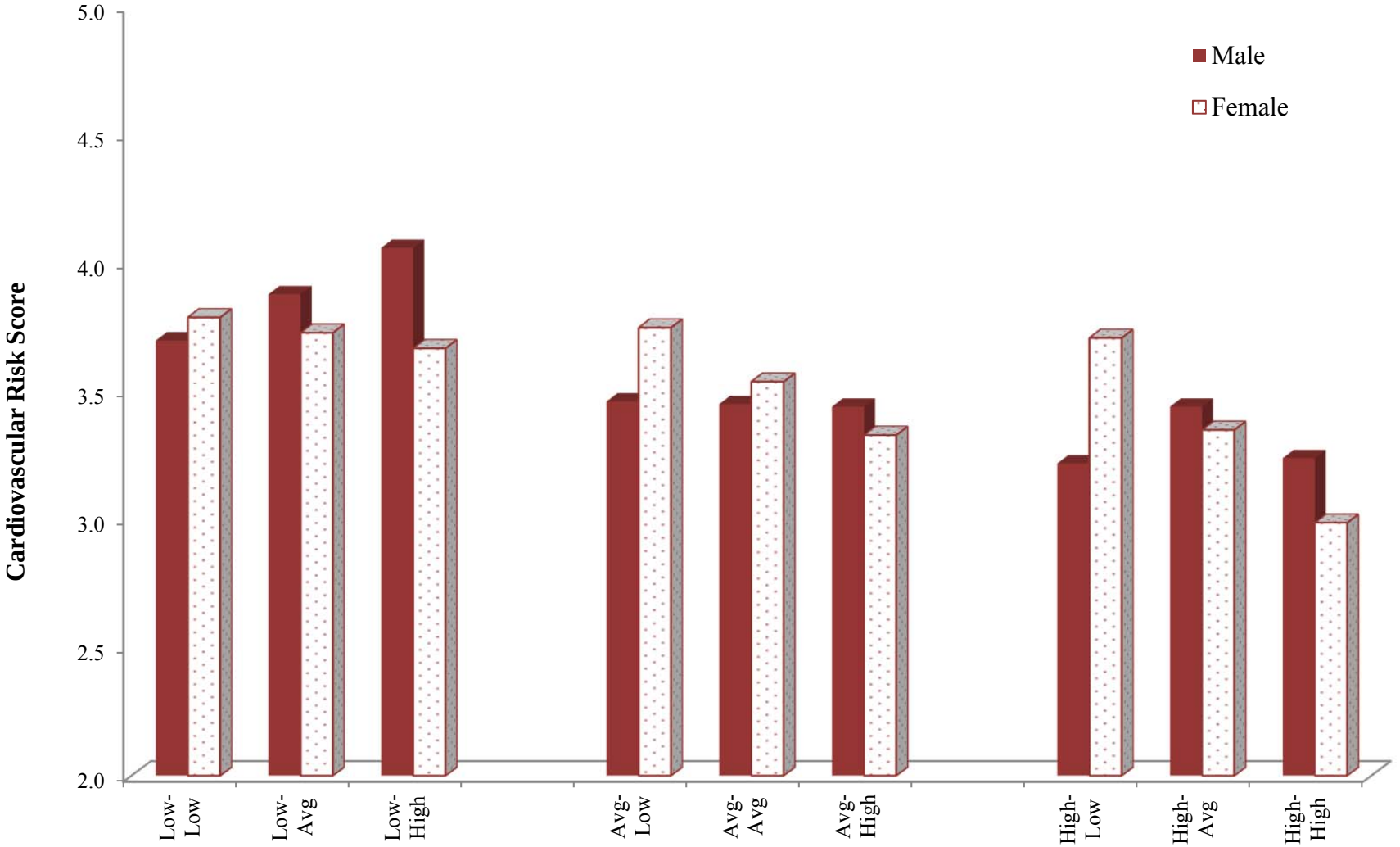
Notes: [a] Percentages may not add to 100 due to rounding. [b] SES measures categorized as low (<-0.5 SD), average (-0.5 SD – 0.5 SD), and high (>0.5 SD).
*$p<0.05$, adjusted for multiple comparisons using Bonferroni method

Table 3: Regression coefficients from multivariate linear regression, stratified by race/ethnicity, weighted analysis, National Longitudinal Study of Adolescent Health, W1 & W4

	Model 1 b (SE)	**Model 2** b (SE)	**Model 3** b (SE)	**Model 4** b (SE)	**Model 5** b (SE)
Whites (n=6,906)					
Adolescent SES	-0.38 (0.06)***		-0.38 (0.08)***	-0.43 (0.08)***	-0.41 (0.08)***
Female x Adolescent SES	0.11 (0.07)		0.19 (0.08)*	0.24 (0.08)**	0.22 (0.08)*
Adult SES		-0.15 (0.05)**	-0.02 (0.05)	-0.01 (0.05)	-0.01 (0.06)
Female x Adult SES		-0.13 (0.07)	-0.19 (0.08)*	-0.20 (0.08)*	-0.22 (0.08)*
Adolescent SES x Adult SES				-0.19 (0.07)**	-0.19 (0.07)*
Female x Adolescent SES x Adult SES				0.04 (0.09)	0.06 (0.09)
Female	0.08 (0.05)	0.14 (0.05)**	0.10 (0.05)	0.09 (0.06)	0.10 (0.06)
Intercept	3.40 (0.04)***	3.35 (0.05)***	3.40 (0.04)***	3.45 (0.05)***	3.19 (0.06)***
Blacks (n=2,484)					
Adolescent SES	0.05 (0.12)		0.03 (0.12)	0.15 (0.11)	0.17 (0.11)
Female x Adolescent SES	-0.20 (0.15)		-0.14 (0.15)	-0.32 (0.14)*	-0.32 (0.13)*
Adult SES		0.06 (0.12)	0.05 (0.12)	0.09 (0.12)	0.05 (0.12)
Female x Adult SES		-0.25 (0.15)	-0.20 (0.15)	-0.29 (0.15)‡	-0.28 (0.14)‡
Adolescent SES x Adult SES				0.23 (0.16)	0.21 (0.14)
Female x Adolescent SES x Adult SES				-0.43 (0.22)‡	-0.38 (0.19)*
Female	0.53 (0.11)***	0.52 (0.11)***	0.50 (0.11)***	0.55 (0.12)***	0.39 (0.12)**
Intercept	3.75 (0.08)***	3.77 (0.09)***	3.77 (0.09)***	3.74 (0.10)***	3.71 (0.10)***
Latinos (n=1,905)					
Adolescent SES	-0.20 (0.10)*		-0.20 (0.10)*	-0.21 (0.10)*	-0.22 (0.09)*
Female x Adolescent SES	-0.05 (0.13)		0.02 (0.14)	0.03 (0.14)	0.07 (0.14)
Adult SES		-0.07 (0.12)	-0.02 (0.12)	-0.05 (0.15)	-0.09 (0.14)
Female x Adult SES		-0.32 (0.21)	-0.31 (0.22)	-0.23 (0.23)	-0.19 (0.24)
Adolescent SES x Adult SES				-0.06 (0.11)	-0.09 (0.11)
Female x Adolescent SES x Adult SES				0.17 (0.16)	0.23 (0.16)
Female	0.12 (0.15)	0.15 (0.14)	0.15 (0.15)	0.13 (0.16)	0.06 (0.15)
Intercept	3.60 (0.10)***	3.67 (0.10)***	3.59 (0.10)***	3.30 (0.10)***	3.34 (0.11)***

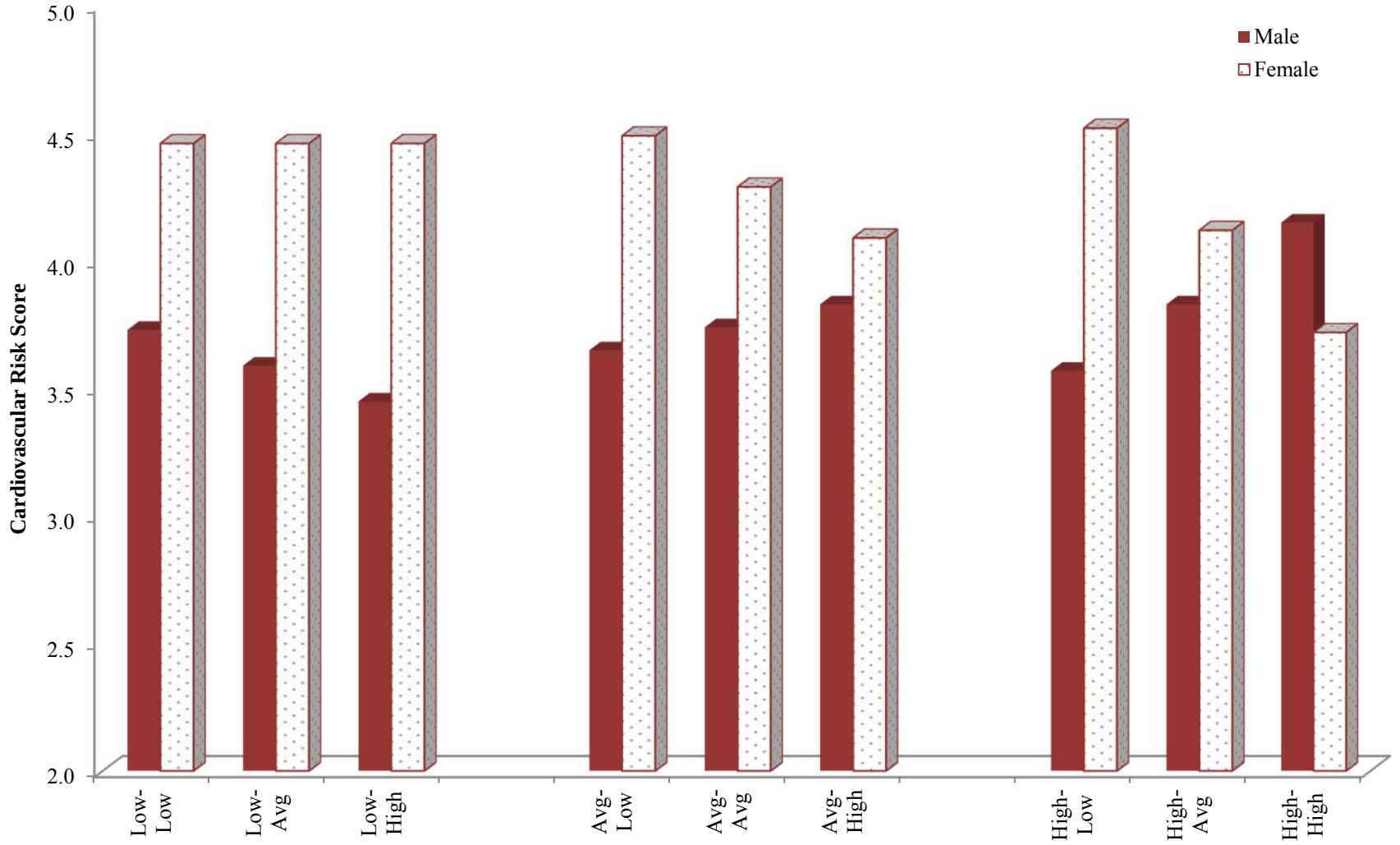
Notes: All models adjust for nativity, family structure W1, self-rated health W1, age W4, marital status W4, and # of infections W4. Model 5 further adjusts for physical activity W1, change in physical activity between W4 and W1, smoking status W4, depressive symptoms W1, change in depressive symptoms between W4 and W1, perceived social status W4, and economic hardship W4.
‡$p<.10$; *$p<0.05$; **$p<0.01$; ***$p<0.001$

Figure 1: Whites



Notes: Estimates based on Model 4 from Table 3. "Low" refers to low SES in adolescence or adulthood (1 SD below mean). "Avg" refers to mean SES in adolescence or adulthood (0 SD). "High" refers to high SES in adolescence or adulthood (1.0 SD above mean).

Figure 2: Blacks



Notes: Estimates based on Model 4 from Table 3. "Low" refers to low SES in adolescence or adulthood (1 SD below mean). "Avg" refers to mean SES in adolescence or adulthood (0 SD). "High" refers to high SES in adolescence or adulthood (1.0 SD above mean).

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