Published in final edited form as:
Soc Sci Med. 2009 March ; 68(5): 807–813. doi:10.1016/j.socscimed.2008.12.029.

Racial and socioeconomic disparities in arterial stiffness and intima media thickness among adolescents

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Abstract

Racial and socioeconomic status (SES) disparities in cardiovascular disease (CVD) risk are well established among adults. However, little is known about disparities in CVD risk among adolescents, particularly considering indices of subclinical CVD. Our aim was to examine socioeconomic and racial disparities in subclinical CVD indices among adolescents. We hypothesized that African American and lower SES adolescents would show greater arterial stiffness and intima media thickness compared to Caucasian and higher SES adolescents, respectively. Participants were 81 African American and 78 Caucasian adolescents (mean age = 17.8) from two schools in Pittsburgh, PA, USA. Measures of subclinical CVD were pulse wave velocity and intima media thickness, as assessed by Doppler and B-mode ultrasound, respectively. SES indices included parental education, family income, family assets, subjective social status, and census-derived neighborhood SES. Hypotheses were evaluated in multiple linear regression models with the covariates age, gender, body mass index, and systolic blood pressure. Results indicated that African American adolescents were more often in low SES positions than Caucasians. When considered individually, racial and SES disparities in pulse wave velocity, and to a lesser extent, intima media thickness, were evident. When race and SES were considered together, high school education, low or medium income, and low neighborhood SES were associated with higher pulse wave velocity. Fewer assets were associated with higher intima media thickness. In conclusion, racial and SES disparities in indices of subclinical CVD were observed, with findings most pronounced for SES disparities in pulse wave velocity. This study extends previous findings in adults to adolescents, indicating that disparities in arterial stiffness and intima media thickness occur as early as adolescence. Efforts to reduce socioeconomic and racial disparities in CVD should target disparities early in life.

Keywords

USA; Socioeconomic status (SES); Socioeconomic position; Race; Cardiovascular disease; Pulse wave velocity; Intima media thickening; Adolescents

Introduction

The pronounced racial and socioeconomic disparities in cardiovascular disease (CVD) risk among adults are well established. Among adults, low socioeconomic status (SES) is associated with increased risk for incident CVD, CVD mortality, and CVD risk factors (Kaplan & Keil, 1993). These associations are observed for individual, family, and neighborhood-level SES. Racial differences in cardiovascular risk are also well-documented, with African American

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adults showing higher prevalence of multiple CVD risk factors and total and premature CVD mortality than Caucasians (Ferdinand, 2006; Rosamond et al., 2007). Racial differences in CVD may be attributed in part to the overrepresentation of African Americans in low SES positions (LaVeist, 2005; Williams, 1999; Williams & Collins, 1995). However, the relative contributions of SES and race to CVD disparities are not fully understood.

It has been proposed that CV risk associated with low SES begins early in life, the result of a lifetime of exposure to adverse conditions associated with low SES (Lynch, 2000). Notably, childhood SES predicts cardiovascular outcomes in adults that are not fully explained by adult SES (Galobardes, Smith, & Lynch, 2006). However, this research has largely been conducted in adults. Less is known about disparities in CVD risk among children or adolescents. Existing studies of disparities among youth have been largely restricted to cardiovascular risk factors. A previous report from the present sample indicated that low SES and African American race were associated with higher ambulatory blood pressure among adolescents (McGrath, Matthews, & Brady, 2006). Other studies suggest that disparities in cardiovascular risk factors among children and adolescents may vary by the risk factor under investigation (Chen, Matthews, & Boyce, 2002; Starfield, Riley, Witt, & Robertson, 2002).

Subclinical CVD measures have potential utility in investigating CVD risk among adolescents (Slyper, 2004). These noninvasive measures include carotid artery intima media thickening (IMT), a marker of early atherosclerosis (O'Leary & Polak, 2002), and pulse wave velocity (PWV), a measure of arterial stiffness (Arnett, Evans, & Riley, 1994), both of which are considered early markers of disease and both of which have been prospectively linked to future CVD events (Chambless et al., 1997; Mattace-Raso et al., 2006; O'Leary et al., 1999; Sutton-Tyrrell et al., 2005; Willum-Hansen et al., 2006). Evidence of CVD is apparent early in life, with autopsy studies revealing fatty streaks and early atherosclerotic lesions in the vascular beds of children and adolescents that correlate well with CVD risk factors (Berenson et al., 1998; McGill et al., 2000). Similarly, increased IMT and arterial stiffness observed among children and adolescents with elevated CVD risk factors (Im, Lee, Shim, Lee, & Lee, 2007; Riley et al., 1986; Slyper, 2004), including hypercholesterolemia (Pauciullo et al., 1994), insulin resistance (Gungor, Thompson, Sutton-Tyrrell, Janosky, & Arslanian, 2005; Lee et al., 2007), and obesity (Gungor et al., 2005; Meyer, Kundt, Steiner, Schuff-Werner, & Kienast, 2006). Finally, CVD risk factors in childhood and adolescence predict carotid IMT, coronary calcification, and arterial stiffness in adulthood (Li, Chen, Srinivasan, & Berenson, 2004; Li et al., 2003; Mahoney et al., 1996; Raitakari et al., 2003).

In contrast to research among adults (Diez-Roux, Nieto, Tyroler, Crum, & Szklo, 1995; Din-Dzietham, Couper, Evans, Arnett, & Jones, 2004; Lynch, Kaplan, Salonen, & Salonen, 1997; Manolio et al., 1995; Troxel, Matthews, Bromberger, & Sutton-Tyrrell, 2003), there has been little examination of racial and SES disparities in IMT or PWV among children and adolescents. There is some evidence that African American adolescents may have more extensive fatty streaks in vascular beds (McGill et al., 2000; Wissler & Strong, 1998), and that African American young adults have greater carotid IMT (Urbina et al., 2002) and stiffening (Urbina et al., 2004) than Caucasians. However, findings are not consistent, as African American adolescents may also have fewer raised lesions than Caucasians (McGill et al., 2000; Wissler & Strong, 1998). Even less is known about the joint associations of race and SES with subclinical CVD indices among youth, which is particularly important given the overrepresentation of African American families in low SES positions (Williams, 1999; Williams & Collins, 1995).

The study aim is to examine the associations between race and SES with IMT and PWV among African American and Caucasian adolescents. It is hypothesized that African American and lower SES adolescents have increased IMT and PWV relative to their Caucasian and higher

SES counterparts. Given the multidimensional nature of SES (LaVeist, 2005; Williams, 1999; Williams & Collins, 1995), objective and subjective SES, and SES measured on individual and neighborhood levels are examined. Independent vs. interactive effects of race and SES are also explored.

Method

Study sample

Participants were derived from African American and Caucasian adolescents participating in a study of cardiovascular functioning. At baseline (1999–2002), 214 adolescents aged 14–16 were recruited from two racially and socioeconomically diverse high schools in Pittsburgh, PA. Adolescents self-reported their race/ethnicity, which was subsequently verified with parental interview. Exclusion criteria included congenital heart disorders or a history of cardiovascular or renal disease; use of prescription medications affecting the cardiovascular system; use of recreational drugs; inability to read questionnaires; severe learning disability or mental retardation; documented psychiatric disorder; unwillingness to not smoke for 12 h before testing, and parent reported participant weight >80% of ideal for age and gender norms. The University of Pittsburgh Institutional Review Board approved study procedures, and parents/guardians and adolescents provided informed consent and assent, respectively.

Participants continuing to meet initial eligibility criteria (except the weight criterion) were invited to return for a follow-up assessment an average of 3.3 (SD = 0.82, range 1.5–5.9) years after baseline (2003–2005), during which IMT and PWV were measured. Fifty-three participants were not available for follow-up because they could not be located ($n = 29$), they refused ($n = 4$), they relocated ($n = 6$), or they had scheduling conflicts ($n = 14$). Two additional individuals did not undergo measurement of subclinical CVD indices. Those who were lost to follow-up had a higher BMI ($p < 0.05$) and were somewhat more likely to have parents with low education ($p = 0.08$) than those who attended the follow-up. The present investigation is based upon the 81 African American and 78 Caucasian participants undergoing IMT or PWV measures at this follow-up. Of these 159 adolescents, 16 were excluded from PWV models due to missing PWV data and one was excluded from PWV and IMT models due to missing systolic blood pressure (SBP). In addition,18 participants missing data on one or more SES measures (income $n = 16$, education $n = 2$, subjective social status $n = 1$; one person had >one missing SES variable) were excluded from models using that SES variable. Those missing these data were somewhat more likely to live in lower SES neighborhoods than those with these data ($p = 0.06$).

Socioeconomic status

SES was measured at baseline via parental interview. This interview was not repeated at the follow-up visit. Parental education was highest level of reported educational attainment, categorized as ≤high school, associates degree/some college, ≥college degree. The higher attainment of two parents was used for two parent households. Family income was reported as gross annual family pre-tax income, categorized according to the sample distribution (low: < $30,000, medium: $30,000–$50,000, high: >$50,000). Assets were a summed index of number of cars, bedrooms (house or apartment), insurance policies (medical, auto, home/renters, life), loans (auto, student), and homeownership (yes/no). Loans were included in this index given their significant positive correlation with other asset measures ($p < 0.05$), contingency upon adequate credit, and inverse relation to CVD indices in preliminary analyses. The specific asset items were based on indicators of family SES commonly used in the epidemiologic literature. As reported elsewhere (Brady & Matthews, 2002), these asset measures form a single factor in factor analysis and are correlated with relevant constructs such as stressful life events exposure and other SES indices, independent of race/ethnicity, among adolescents.

Neighborhood SES was calculated from two 2000 census tract indices corresponding to family address (the mean of % of households with incomes above the poverty threshold and % of households with ≥high school education). Higher scores indicate higher neighborhood SES.

Subjective social status was assessed at the follow-up assessment with the MacArthur Scale of Subjective Social Standing for adolescents (Adler, Epel, Castellazzo, & Ickovics, 2000; Goodman et al., 2001). This scale assesses perceptions of placement in a social hierarchy, and in previous investigations, has predicted health outcomes among adolescents above objective SES (Adler et al., 2000; Goodman et al., 2001; Lemeshow et al., 2008). Participants locate his/her family in the US society and him/herself in the school community on one of 10 rungs on a ladder numbered 1–10. Higher scores indicate higher status. Individual school community rankings are presented here as they tap an individual status comparison construct quite distinct from objective SES (although findings for the US ladder were comparable with results for the school ladder). Given skew of this ordinal measure, scores were categorized into approximate tertiles based upon the sample distribution.

Pulse wave velocity

Aortic pulse wave velocity is a measure of arterial stiffness, based upon the principle that velocity of pressure waves traveling down the aorta increases with stiffer vessels. Using nondirectional transcutaneous Doppler flow probes (model 810-a, 9 and 8.5 MHz; Parks Medical Electronics, Aloha, OR), simultaneous Doppler flow signals were obtained from right carotid and femoral arteries. The distance between carotid and femoral sites was measured using a metal tape measure and calculated (carotid −femoral distance = (suprasternal −umbilicus) + (umbilicus − femoral) −(carotid − suprasternal)). Three runs per participants were performed, with the mean of waveforms from all usable runs used. Data were scored using software developed by the Laboratory of Cardiovascular Science, Gerontology Research Center, National Institute on Aging, which averages the waveforms and determines the time from the R-wave to foot of each waveform. PWV was calculated as the distance between carotid and femoral arteries divided by the time interval between carotid and femoral waveforms. Higher PWV indicates stiffer vessels. Reproducibility from previous PWV measures from this laboratory is high (intraclass correlation (ICC)= 0.72–0.86) (Sutton-Tyrrell et al., 2001). Due to skew, PWV values were log transformed for analyses.

Intima media thickening

B-mode ultrasound images from the carotid artery were obtained using a 5.0 MHz linear array transducer and Toshiba SSA-270A scanner (Toshiba American Medical Systems, Tustin, CA). Images were obtained from four locations on each of left and right common carotid arteries: near and fall walls of distal common carotid artery, far walls of carotid bulb, and internal carotid artery. Participants were examined in a supine position. Trained readers first scanned the entire circumference of each segment, and the optimal longitudinal view for each segment was captured electronically and stored on magnetic optical disc. The lumenintima interface and the mediaadventitia interface were traced electronically across a 1-cm segment, and the computer generated a measurement for each pixel over this area using a computerized reading program developed for the Cardiovascular Health Study (O'Leary et al., 1992) modified in Pittsburgh. Mean IMT was the average readings across the eight locations. Readings were conducted at the University of Pittsburgh Ultrasound Research Laboratory with careful quality control measures (Thompson, Sutton-Tyrrell, & Wildman, 2001). Reproducibility of this protocol and between technicians is high (ICC =0.91–0.99) (Thompson et al., 2001).

Covariates

The covariates body mass index (BMI) and blood pressure were derived from measures taken during the follow-up assessment. BMI was calculated as weight (kg)/height $(m)^2$. Resting blood

pressure was calculated as the mean of three seated measurements taken after a 5-min rest. Lack of cigarette smoking within 8 h of assessment of IMT and PWV was also verified.

Statistical analyses

Differences in subject characteristics by race were calculated via *t*-tests, chi-square tests, and logistic regression. Correlations among SES and outcome measures were calculated via Spearman's rho and Pearson correlation coefficients.

Race and each SES indicator were first estimated separately in relation to PWV or IMT via multiple linear regression. Covariates were chosen based upon previously documented relations with PWV and IMT and relation to the outcome at $p < 0.10$. Covariates were age, BMI, gender, and SBP. Next, race was included with each SES measure in multiple linear regression models. Finally, all SES variables significantly related to the outcome were included simultaneously in models with race and covariates. Neighborhood SES was not included in these models due to collinearity between individual and neighborhood SES measures. While we considered evaluating associations with neighborhood and individual SES indices using multilevel modeling, this approach was not used due to lack of adequate clustering of participants within neighborhoods (of the 78 census tracts, 35% contained one participant, 14% two participants, 13% three participants, and 16% ≥four participants). All models were evaluated for interactions with gender and race, and stratified models presented in the presence of a significant interaction term. Analyses were performed with SAS (v.8.02, SAS Institute, Cary, NC) and were two-sided, alpha =0.05.

Results

Sample characteristics are presented in Table 1. Relative to Caucasians, a higher proportion of African American participants lived in families in the low family income and low parental education categories. They also scored lower on the household assets index, lower on the neighborhood SES measure, and higher on PWV and IMT indices than Caucasians.

PWV was uncorrelated with IMT for the total sample (r=−0.04, p= 0.60), and by race (Caucasians: r=−0.10, p = 0.40, African Americans: r=−0.05, p= 0.52). Significant intercorrelations between SES indices, with the exception of subjective social status, were observed (Table 2). Parental education was positively associated with neighborhood SES exclusively among Caucasians.

Pulse wave velocity

African American adolescents had significantly higher PWV than Caucasians, controlling for age, gender, BMI, and SBP in linear regression models (b (SE)= 0.04 (0.01), p = 0.01). Moreover, considered separately, ≤high school parental education (vs. ≥college, b (SE) = 0.04 (0.02), p = 0.008), low (vs. high, b (SE) = 0.05 (0.02), p= 0.005) or medium family income (vs. high, b (SE) =0.04 (0.02), p = 0.02), and lower neighborhood SES (b (SE)=−0.002 (0.0005), p= 0.0006) were associated with higher PWV, controlling for age, gender, BMI, and SBP. Neither household assets nor subjective social status was associated with PWV.

Each SES measure was next considered in the same regression model with race in relation to PWV. In general, the objective SES indices showed more consistent relations with PWV than race (Table 3). A significant interaction between race and income was observed (p = 0.02), with low family income associated with higher PWV among African Americans (low income vs. high, b (SE) = 0.08 (0.03), p = 0.003; medium income vs. high, b (SE)= 0.06 (0.03), 0.02 p =0.06), but not Caucasians (low income vs. high, b (0.03), p =0.57; medium income vs. high,

b (SE) = 0.03 (0.02), *p* =0.24). Notably, Caucasians were underrepresented in the low income category. No interactions with gender were observed.

When all objective individual-level SES variables related to PWV (education, income) were included with race in relation to PWV, again only family income remained associated with PWV (low income vs. high, *b* (SE)= 0.03 (0.02), *p* = 0.12; medium income vs. high, *b* (SE) = 0.04 (0.02), *p* =0.04) for the sample as a whole. Stratified by race, low income was associated with PWV among African Americans (low income vs. high, *b* (SE) =0.08 (0.03), *p* =0.008; medium income vs. high, *b* (SE) = 0.05 (0.03), *p*= 0.10), whereas low education was associated with higher PWV among Caucasians (≤high school vs. ≥college, *b* (SE)= 0.06 (0.03), *p* =0.02; associates/some college vs. ≥college, *b* (SE)= 0.03 (0.03), *p* =(0.40).

Intima media thickening

African Americans had marginally higher IMT than Caucasians (*b* (SE) =0.009 (0.005), *p* = 0.07), controlling for age, BMI, SBP, and gender. Of the SES indicators, only fewer household assets (*b* (SE)=−0.002 (0.001), *p* = 0.008) were significantly associated with higher IMT, controlling for age, BMI, SBP, and gender.

We next considered race with SES in relation to IMT. Assets remained significantly associated with lower IMT, and race was no longer significant (Table 3). A significant interaction between race and neighborhood SES was observed (*p*= 0.03), with lower neighborhood SES associated with higher IMT among African Americans (*b* (SE)=−0.0008 (0.0004), *p*= 0.03), but not Caucasians (*b* (SE) = 0.0002 (0.0003), *p*= 0.51). No interactions with gender were observed.

Discussion

It has been hypothesized that SES disparities in adult CVD are the result of a lifetime of cumulative exposure to adverse conditions. A corollary is that disparities should begin to appear early in life. However, there has been little examination of disparities in subclinical CVD indices among children and adolescents. In the present study, racial and SES disparities were observed, with findings more pronounced for PWV than for IMT.

Limited prior work has examined racial disparities in subclinical CVD indices among youth. In the PDAY autopsy study, African American adolescents had more extensive fatty streaks in vascular beds than Caucasians, although greater raised lesions were evident among Caucasians (McGill et al., 2000; Wissler & Strong, 1998). In the Bogalusa Heart Study, African American young adults had greater carotid IMT (Urbina et al., 2002) and carotid artery stiffening (Urbina et al., 2004) than Caucasians. Among adults, greater arterial stiffness (Din-Dzietham et al., 2004) and IMT (Manolio et al., 1995) are generally observed among African Americans vs. Caucasians. The present study importantly extends this work to youth, finding that racial differences in arterial stiffness are observed as early as adolescence.

Given the overrepresentation of African Americans in low SES positions, whether racial disparities in health are accounted for by SES has been debated (LaVeist, 2005; Williams, 1999; Williams & Collins, 1995). Among adults, there is a modest residual association of race above SES, although this finding varies with SES indicator (LaVeist, 2005; Williams, 1999; Williams & Collins, 1995). It is important to note that, rather than a confounder, SES can be considered a pathway by which disparities manifest (Williams, 1999). Although relatively understudied, it is also possible that disadvantage associated with minority race and low SES would be synergistic, placing low SES African Americans at particular risk (Anderson & Armstead, 1995). We know of no study examining SES disparities in IMT or PWV among children and adolescents, either alone or with race. This omission stands in contrast to the

burgeoning literature emphasizing the importance of childhood SES in the development of adult CVD (Galobardes et al., 2006; Lynch, 2000).

African American adolescents were in lower socioeconomic positions than Caucasians, as assessed by family income, parental education, household assets, and neighborhood SES. In turn, several indices of low SES were associated with higher PWV, and fewer assets with greater IMT. These differences largely persisted controlling for race. Racial differences remained significant only in models that included an SES indicator lacking a univariate association with the outcome under study. Thus, SES disparities in vascular structure or function may be evident early in life and may play a key, but not exclusive, role in racial disparities in CVD.

There was also suggestion of interactions between race and SES. Low income and low neighborhood SES were associated with higher PWV and IMT, respectively, chiefly among African American adolescents. These interactions must be interpreted with caution given the under-representation of Caucasian adolescents in low income households and limitations in our neighborhood measure (minimal clustering of individuals within neighborhoods). However, prior research, although limited and contradictory (Anderson & Armstead, 1995; Farmer & Ferraro, 2005; Williams & Collins, 1995), suggests that low SES African Americans may experience particularly poor health. For example, African Americans in poverty may be considered among the “truly disadvantaged” (Wilson, 1987), subject to the “double jeopardy” of poverty and racism (Landrine, Klonoff, Alcaraz, Scott, & Wilkins, 1995). Moreover, any one index of low SES may be associated with greater concurrent SES disadvantage among African Americans. For example, at a given income, African Americans have lower wealth, and a given educational level confers lower economic return among African Americans relative to Caucasians (Braveman et al., 2005). Thus, low SES as measured by any one index may not be equivalent among African Americans and Caucasians due to this concurrent disadvantage, with associated health implications for African Americans. Finally, neighborhoods predominated by African Americans vs. Caucasians often differ, varying on economic and non-economic dimensions that may potentiate the impact of low SES on health (Williams & Collins, 2001). Notably, residential segregation has been cited as a key driver of racial disparities in health (Williams & Collins, 2001).

SES is a multidimensional construct, reflecting access to material goods as well as status and prestige. This study assessed multiple SES dimensions, including objective and subjective indices on individual, household, and neighborhood levels. Although tapping different dimensions, objective SES indices were moderately to strongly correlated, and for traditional SES indicators, consistently related to PWV. However, subjective social status was largely uncorrelated with objective SES, evidenced no racial differences, and showed weak if any relations with IMT and PWV. These findings suggest that among adolescents, subjective social status may have assessed a construct distinct from SES. Notably, the status question asked for ranking of oneself relative to peers on grades, school activities, and popularity, a likely appropriate measure of social status among adolescents (Goodman et al., 2001). However, this construct is distinct from SES and although related to indices such as BMI changes among female adolescents in previous work (Lemeshow et al., 2008), appears less related to adverse vascular changes.

Observed associations were more robust for PWV than for IMT. Reasons for these differences are not clear. However, IMT measures intimal thickening of the vessel wall, and PWV measures arterial stiffness. Therefore, PWV is more of a functional measure potentially more responsive to acute stressors (Vlachopoulos et al., 2006). Moreover, it has been proposed that the arterial stiffening may begin earlier in life than increases in IMT (Riley et al., 1986). Accordingly, IMT in this sample of adolescents was low.

There are multiple and complex pathways by which racial and SES disparities in cardiovascular risk develop. SES and/or race-based disadvantage among adolescents has been associated with greater rates of smoking and sedentary behavior (Starfield et al., 2002), greater perceived stress (Goodman, McEwen, Dolan, Schafer-Kalkhoff, & Adler, 2005), more mental health problems (Costello, Swendsen, Rose, & Dierker, 2008), poorer self-rated health (Starfield et al., 2002), and higher BMI, waist circumference, and insulin resistance (Goodman, McEwen, Huang, Dolan, & Adler, 2005) than their more advantaged counterparts. SES and/or race-based disadvantage has also been associated with adverse stress-related neuroendocrine and inflammatory processes among adults (Cohen, Doyle, & Baum, 2006; Janicki-Deverts et al., 2007; Nazmi & Victora, 2007). Among children, social and economic disadvantage is associated with elevated salivary cortisol concentrations (Lupien, King, Meaney, & McEwen, 2000), overnight urinary catecholamines, and overall physiologic wear and tear (Evans, 2003), particularly in the context of low maternal responsiveness (Evans, Kim, Ting, Tesher, & Shannis, 2007). Adolescents living in low SES neighborhoods and households are at greater risk of exposure to violence, pollutants, noise, crowding, family turmoil, few safe places for physical activity, more atherogenic food choices as well as more limited educational/occupational opportunities and community supports (Diez Roux, 2003; Evans, 2004; Williams & Collins, 2001). Finally, African American youth from low SES families may additionally experience institutional and individual racism with associated health ramifications (Williams, 1999). Thus, the mechanisms by which SES and race-based disadvantage may translate into adverse cardiovascular outcomes are likely multiple and interacting over time.

These findings should be interpreted in light of several limitations. First, consistent with study exclusions, the sample was fairly healthy, with restricted range for IMT, likely impacting the ability to detect associations. Moreover, given lack of clustering of households within zipcodes, it was not possible to use multilevel modeling to disentangle relative contributions of household vs. neighborhood SES. Future work should consider these associations with more clustered data allowing more precise delineation of these respective individual and contextual associations. The study was observational, and the causal nature of relations cannot be assumed. Finally, objective SES measures were assessed several years prior to IMT and PWV measures, resulting in potential misclassification of SES. However, this misclassification should only impact measures more variable over time, such as income, and would have increased error, making detection of significant results less likely. Assessment of multiple SES indices, including relatively stable indicators such as parental education, helps avoid erroneous conclusions due to misclassification on one indicator.

This study is unique in is assessment of multiple indices of SES as well as early markers of vascular thickening or stiffening among African American and Caucasian adolescents, allowing examination of the relative contributions of SES and race to these vascular changes. Moreover, adolescents comprised the sample, a relatively understudied group (Starfield et al., 2002), allowing particular insight into the oft-stated theory that disparities in CVD begin early in life. Further, multiple dimensions of SES were included, allowing an extensive examination of SES important in understanding racial disparities (LaVeist, 2005). Finally, measurement of both IMT and PWV allowed comparison across measures.

In conclusion, both SES and racial disparities in subclinical CVD indices were observed among adolescents. Findings were most pronounced for PWV, a measure of arterial stiffness that may mark early atherosclerosis and arteriosclerotic remodeling. This study also suggested that low SES may be one important pathway by which racial disparities manifest, with additional suggestion that low SES African American adolescents may be at particular risk. These findings support the hypothesis that racial and socioeconomic disparities in disease begin early in life, underscoring the importance of prevention efforts to reduce disparities in CVD that begin early in life. Policy efforts to reduce disparities should include a strong infrastructure to monitor

disparities and a systematic evaluation of the impact of policies across sectors on these disparities (Kawachi, Daniels, & Robinson, 2005). Our findings suggest that best practices in population health and human development policy would include reducing race-based and socioeconomic adversity in health among youth. Future work should evaluate the psychological, social, and environmental pathways by which disparities in adolescent health manifest.

Acknowledgments

This research was supported in part by the National Institutes of Health grants (HL07560, HL25767, HL076852, HL076858, AG029216) and by the John D. and Catherine T. MacArthur Foundation Research Network on Socioeconomic Status and Health.

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Table 1

Sample characteristics by race.

	African American (*N* = 81)	Caucasian (*N* = 78)	p
Age, years, *M* (SD)	17.8 (1.1)	17.8 (0.9)	0.71
Gender, *n* (%)			
Male	41 (50.6)	42 (53.9)	
Female	40 (49.4)	36 (46.2)	
BMI, *M* (SD)	24.4 (4.4)	24.1 (3.8)	0.51
SBP, mmHg, *M* (SD)	109.3 (9.1)	107.1 (8.6)	0.12
DBP, mmHg, *M* (SD)	63.4 (7.9)	63.1 (9.1)	0.81
Education,[a] *n* (%)			
≤High school	43 (54.4)	19 (24.4)	<0.0001
Associates degree/some college	16 (20.3)	13 (16.7)	
≥College	20 (25.3)	46 (59.0)	
Total annual family income, *n* (%)			
Low (<$30,000)	32 (47.1)	11 (14.7)	<0.0001
Medium ($30,000–$50,000)	21 (30.9)	32 (42.7)	
High (>$50,000)	15 (22.1)	32 (42.7)	
Assets,[b] *M* (SD)	9.5 (3.2)	11.3 (2.0)	<0.0001
Subjective social status, *M* (SD)			
Low (≤6)	24 (30.0)	30 (38.4)	0.41
Medium (7)	24 (30.0)	24 (30.8)	
High (≥8)	32 (40.0)	24 (30.8)	
Neighborhood SES,[c] *M* (SD)	58.9 (10.5)	72.9 (12.1)	<0.0001
PWV (cm/s), *M* (SD)	571.4 (122.2)	525.3 (116.4)	0.01
IMT (mm), *M* (SD)	0.54 (0.03)	0.53 (0.04)	0.05

[a] Higher of two parents for two parent households.

[b] Sum of number of cars, homeowner status, number of bedrooms, number of insurance policies, number of loans.

[c] Mean of census-derived % nonpoor and % ≥high school education.

Table 2

Rank-order correlations among SES variables, by race.

	Education[a]	Family income	Assets[b]	Subjective social status	Neighborhood SES[c]
Education[a]	-	0.34**	0.48****	0.05	0.18
Family income	0.46****	-	0.80****	−0.21†	0.41***
Assets[b]	0.31**	0.42***	-	−0.10	0.39***
Subjective social status	0.12	−0.10	0.14	-	−0.04
Neighborhood SES[c]	0.67****	0.47****	0.22†	0.04	-

Note: correlations for African Americans above the diagonal, Caucasians below the diagonal.

† $p < 0.10$

** $p < 0.01$

*** $p < 0.001$

**** $p < 0.0001$

[a] Higher of two parents for two parent households.

[b] Sum of number of cars, homeowner status, number of bedrooms, number of insurance policies, number of loans.

[c] Mean of census-derived % nonpoor and % ≥high school education.

Soc Sci Med. Author manuscript; available in PMC 2010 June 18.

Table 3

Associations between race, SES, and measures of subclinical CVD.

	PWV *b* (SE)	IMT *b* (SE)
Education[a]		
≤High school	0.03 (0.02)†	−0.003 (0.007)
Associates/Some college	0.01 (0.02)	−0.005 (0.008)
≥College	ref	ref
Race	0.02 (0.02)	0.01 (0.006)‡
Family income		
Low (<$30,000)	0.04 (0.02)*	0.01 (0.008)
Medium ($30,000−$50,000)	0.04 (0.02)*	−0.002 (0.007)
High (>$50,000)	ref	ref
Race	0.03 (0.02)‡	0.005 (0.007)
Number of assets[b]	−0.002 (0.003)	−0.002 (0.001)*
Race	0.03 (0.02)*	0.007 (0.006)
Subjective social status		
Low (≤6)	0.03 (0.02)‡	−0.01 (0.007)
Medium (7)	0.00 (0.02)	0.003 (0.007)
High (≥8)	ref	ref
Race	0.04 (0.01)**	0.009 (0.005)‡
Neighborhood SES[c]	−0.002 (0.0006)*	−0.0003 (0.0002)
Race	0.01 (0.02)	0.007 (0.006)

Note: each SES variable is considered in separate model; sample sizes for IMT models $n = 158$, except in models with education $n = 156$, with income $n = 142$, and with assets $n = 156$; sample sizes for PWV models $n = 142$, except with education $n = 141$, and with income $n = 131$. Covariates included in linear regression models were age, gender, race, BMI, SBP.

Covariates included in linear regression models were age, gender, race, BMI, SBP.

** $p < 0.01$

* $p < 0.05$,

† $p = 0.05$

‡ $p < 0.10$.

[a] Higher of two parents for two parent households.

[b] Sum of number of cars, homeowner status, number of bedrooms, number of insurance policies, number of loans.

[c] Mean of census-derived % nonpoor and % ≥high school education.