CHILDHOOD SOCIOECONOMIC STATUS AND THE METABOLIC SYNDROME

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Variation in socioeconomic status (SES) is associated with the incidence of coronary heart disease (CHD) and its associated risk factors, with most studies focusing on individuals' current SES. Here, we examine whether childhood SES may be similarly associated with the metabolic syndrome and its component risk factors in a community sample of nonpatient volunteers. Subjects were 843 participants from the University of Pittsburgh Adult Health and Behavior project (age: 30-54 yrs., 51% female, 89% Caucasian/11% African-American). Childhood SES was defined by parental educational attainment and current SES was measured by subjects' years of education. The presence of the metabolic syndrome was identified according to both National Cholesterol Education Program (NCEP) and International Diabetes Federation (IDF) criteria. Logistic and linear regression analyses accounting for age, sex, and race showed parental education was a significant independent predictor of the metabolic syndrome in women (OR = 0.87; 95% CI = 0.79, 0.97; p = 0.008) but not in men (OR = 1.06; 95% CI = 0.89, 1.27; p = 0.527) after controlling for subjects' own education. Thus, a one year increase in parental education was found to predict a thirteen percent decreased likelihood of developing the metabolic syndrome in women after adjustment for both covariates and subjects' education level. Parental education was also a significant predictor of several metabolic syndrome risk factors in women (systolic blood pressure, diastolic blood pressure, triglycerides) and one risk factor in men (HDL cholesterol). Therefore, it appears that childhood SES, as indexed by parental

education, is an important independent predictor of increased cardiovascular risk in middle aged adults in this sample, particularly women.

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1.0 INTRODUCTION

Research on social inequalities has demonstrated a clear relationship between socioeconomic status (SES) and health. Individuals of lower SES experience higher rates of all-cause morbidity and mortality than do more advantaged individuals. This relationship is not attributable solely to poor health among the most deprived individuals, but reflects a gradient of health risk extending across the full distribution of socioeconomic position existing within populations (Adler et al., 1994).

Despite the recognition of this well-established association, the underlying mechanisms by which social inequalities impair health are not well understood. Measures of socioeconomic status indicate one's position within a broader society, and this position might affect health in various ways. Social position can determine both the availability of preventative and health promoting resources, as well as the amount of one's exposure to health-damaging environments (Lynch & Kaplan, 2000). SES may also influence health through its association with psychological and lifestyle-associated risk factors, such as smoking prevalence and subjective distress due to more frequent stressful life events (Anderson & Armstead, 1995). In the latter regard, chronic stressors associated with social position may conceivably alter neuroendocrine functioning, resulting in later health consequences (Brunner et al., 1997).

In the following sections, several aspects of SES and health will be reviewed. First, a conceptual basis for SES research will be presented, along with information concerning the

relationship of SES to cardiovascular health. The metabolic syndrome and its association with adult SES will then be discussed. Next, research on childhood SES as a predictor of adult CHD will be presented. Finally, a review of the current study will be provided, and implications of the findings will be discussed.

1.1 CONCEPTUALIZATION OF SOCIOECONOMIC STATUS

A conceptualization of socioeconomic status itself is necessary when attempting to explain the relationship between socioeconomic status and health. Socioeconomic status (SES) has been defined as one's relative "position" in society, as reflected in access to or the accumulation of material resources or prestige (Lynch & Kaplan, 2000). Measures of socioeconomic position indicate particular structural locations within society (Lynch & Kaplan, 2000) and attempt to quantify an individual's probability of success, i.e. "life chances". Current conceptualization of socioeconomic status relies heavily on the Marxist, Weberian and Functionalist sociological traditions, as summarized by Lynch & Kaplan (2000).

A discussion of socioeconomic status must first address the concept of social class. Social class refers to groups defined by interdependent economic and legal relationships, based on an individual's position within the economy (Krieger, Williams & Moss, 1997). Relationships between classes co-define each other, and are determined by a society's connections through production, consumption and distribution of goods (Krieger et al., 1997). Conceptualizing class as a social relationship emphasizes how members of different social classes advance their economic and social well-being, and how the well-being of one class is linked to the deprivation of another (Krieger et al., 1997). Measures of social class attempt to capture these economic

interactions among people, rather than identify the personal characteristics that determine an individual's position within a hierarchy.

Each sociological tradition approaches social class in a slightly different way. The Marxian definition of social class reflects stratification in relation to the means of production in society. A social class is a group within a society that is relatively similar in political, economic, educational, occupational, and prestige status (Lynch & Kaplan, 2000). According to the Weberian tradition, one's class position yields certain probabilities (or life-chances) of success. Society is stratified by class, status, and political power, and a lack of resources (i.e. goods, skills) places certain groups at a competitive disadvantage. The functionalist approach to stratification suggests that complex societies require stratification into sectors that are more or less valuable to social maintenance and progress. This position maintains that social inequality is necessitated by the need to preferentially reward, by money and power, individuals best qualified to occupy the positions of highest responsibility (Lynch & Kaplan, 2000). The sociological schools of thought described here maintain that macrosocial processes determine the socioeconomic prospects of individuals, with prevailing political and economic conditions generating hierarchies of social position.

Ignored here, however, are individual attributes, such as cognitive abilities and dimensions of personality, that covary with indices of socioeconomic status (Tomlinson-Keasey & Little, 1990). Although such individual characteristics are affected by a wide range of variables, including macrosocial factors, both personality and intelligence also have genetic bases and are influenced by idiosyncratic developmental experiences unrelated to social class (i.e. "nonshared" environmental effects). Individual differences in educational attainment, occupation, and earnings are themselves moderately heritable, and there is significant genetic

covariation of SES and intelligence (Lichtenstein & Pedersen, 1997; Rowe, Vesterdal & Rodgers, 1998). Therefore, it is likely that relative socioeconomic position results from a complex interplay of the political and economic structures described in sociological thought, along with individuals' intellectual and personality characteristics.

Although the concept of socioeconomic status has built upon these sociological traditions of social class, it is important to differentiate these two terms. Whereas social class refers strictly to social groups arising from interdependent economic relationships (i.e. "working class", "managerial class"), current measures of SES aim to quantify an individual's life chances of success in a social hierarchy by including both resource-based (material resources and assets) and prestige-based measures (rank or status in a hierarchy) (Krieger et al, 1997). For example, epidemiological research in England and many other countries draws upon social class data based on the Registrar-General's grouping of occupations, and categorizes individuals' structural location within the economy (Marmot, Kogevinas & Elston, 1987). Because social class in this sense is conceptualized as an ordinal variable, it cannot provide a meaningful measure of distance between adjacent occupational categories, and is therefore less precise. Measures of socioeconomic status, based on composites of resource-based and prestige-based measures at an individual, household, or childhood level, in contrast, provide a more continuous measure of one's standing in a social structure (Krieger, et al., 1997)

Most commonly, SES is assessed at the level of the individual, although household and neighborhood-level indicators are also used. The SES indicators described here are related, but not fully overlapping, and they may affect health through disparate pathways (Gallo & Matthews, 2000). Yet interestingly, disparities in health outcomes persist independently of the measure of socioeconomic status used.

The most widely reported measures of SES are educational attainment, occupational status, income, or some combination of these measures. In studying social inequalities, each measure may be seen to have both advantages and disadvantages. Individual or family income is commonly used to index SES, and can be quantified continuously or divided into categories. Income measures at any one point in time predict various health outcomes, and mortality is strongly and inversely associated with income (Kreiger et al., 1997). Limitations of using income alone as an indicator of SES include income's imperfect correlation with accumulated wealth and insensitivity to fluctuations in life circumstances over time. Level of education is an important marker of socioeconomic position in that it provides information about the likelihood of future success, and is also frequently an indicator of prestige. Potential limitations of using education as a sole measure of SES include variation in the "value" of differing educational experiences defining putatively similar levels of attainment, or in how particular educational accomplishments are rewarded in a given society or economic circumstance (Lynch & Kaplan, 2000). Occupational status is useful in reflecting the prestige, income level and educational requirements associated with various positions in the economic structure, as well as in providing information about job characteristics (such as environmental and working conditions), decision-making latitude, and psychological demands of the job (Lynch & Kaplan, 2000). Various measures of occupation categorize job types in order to reflect a particular occupational hierarchy, such as Rose and Marmot's (1981) Occupational Grade and the Registrar General's Classification (Szreter, 1984). One difficulty of using occupational status as a marker of social position, though, is that of quantifying change in occupational status over the life course (Krieger et al., 1997).

In sum, the intellectual traditions of Marx, Weber and the Functionalists provide a framework for research into social inequalities, and describe structural positions within society that can be measured in several ways. Interest in the associations between socioeconomic position and health has increased in recent years (Lynch & Kaplan, 2000). Most notably, the Whitehall study of mortality (Marmot et al., 1991) demonstrated a clear SES-health gradient among occupational grades of British Civil Servants. This gradient has been shown in U.S. studies as well, using both years of education (Kitagawa & Hauser, 1973) and income (Pappas, Queen, Hadden & Fisher, 1993). Advances in this body of research continue to spur efforts toward the understanding and measurement of socioeconomic variables in relation to health.

1.2 SOCIOECONOMIC STATUS AS A RISK FACTOR

As noted above, there is a well-established, inverse relationship of SES and health, with individuals lower in SES experiencing higher rates of all-cause morbidity and mortality than individuals of higher social position in industrialized nations. This association is observed irrespective of gender or race, even though at most levels of SES, morbidity and mortality rates are higher for blacks than for whites (Pappas et al., 1993; Anderson & Armstead, 1995); also, while some studies suggest that the association between SES and health is weaker among women than men (Dahl, 1993; Stronks, van de Mheen, van den Bos & Mackenbach, 1995; Matthews, Manor & Power, 1999), others suggest the opposite (Vogels, Lagro-Janssen, & van Weel, 1999; Thurston, Kubzansky, Kawachi, & Berkman, 2005). Lower SES predicts poorer health outcomes whether social standing is expressed as level of education, occupation, income, or a composite of these measures (Backlund, Sorlie & Johnson, 1996; Marmot et al., 1991; Adler et al., 1994;

Anderson & Armstead, 1995). Although the largest health deficits are linked to poverty, effects of SES on health are not restricted to the most deprived individuals. Thus, the relationship between socioeconomic status and health has been characterized as linear, wherein even persons of relatively high socioeconomic position prove less healthy than those comprising the most advantaged stratum in a social hierarchy (Adler et al., 1994).

Variation in socioeconomic status is inversely related to the incidence of coronary heart disease (Rose & Marmot, 1981; Liu et al., 1982; Diez-Roux, Nieto, Tyroler, Crum, & Szklo, 1995), as well as the incidence of cardiovascular mortality (Salonen, 1982; Seigel et al., 1987; Keil, Sutherland, Knapp, & Tyroler, 1992). Substantial evidence also documents a clear relationship between socioeconomic status and cardiovascular disease risk factors (Kaplan & Keil, 1993), including health-impairing attributes of behavior and lifestyle. For instance, the prevalence of cigarette smoking increases at successively lower levels of SES (Hay & Foster, 1981; Covey & Wynder, 1981; Dobson, Gibberd, Leeder & O'Connell, 1985; Pierce, Fiore, Novotny, Hatzianndreu & Davis, 1989; Zang & Wynder, 1998). In an extensive review, Sobal and Stunkard (1989) report a strong inverse relationship between socioeconomic status and generalized obesity among women in industrialized societies (albeit findings were less consistent for men), although Zhang and Wang (2004) suggest this association may be weakening for U.S. adults as the overall prevalence of obesity continues to increase. Direct associations have been found between physical activity levels and SES (Ford et al., 1991; Evenson et al., 2002). In terms of biologic risk factors, an inverse relationship between SES and blood pressure (Keil, Tyroler, Sandifer & Boyle, 1977; Sorel, Ragland, Syme & Davis, 1992; Vargas, Ingram, & Gillum, 2000) has been established. Findings are mixed regarding cholesterol, but generally show SES as positively associated with high-density lipoprotein (HDL) cholesterol concentrations (Heiss,

Johnson, Reiland, Davis, & Tyroler, 1980; Donahue, Orchard, Kuller, & Drash, 1985; Linn et al., 1989; Bobak, Hertzman, Skodova, & Marmot, 1999) and inversely related to non–HDL-cholesterol (Winkleby, Kraemer, Ahn, & Varady, 1998) and LDL concentrations (Brunner et al., 1997). Significant inverse relations between socioeconomic status and triglycerides (Brunner et al., 1997), plasma glucose concentrations (Brunner et al., 1997; Ko et al., 2001), plasma fibrinogen (Brunner et al., 1996; Wilson et al., 1993; Jousilahti, Salomaa, Rasi, Vahtera & Palosuo, 2003) and C-reactive protein (Jousilahti et al., 2003, Owen, Poulton, Hay, Mohamed-Ali, & Steptoe, 2003) have also been documented. Thus, these studies demonstrate an apparent association between socioeconomic circumstances and several well-known risk factors for cardiovascular disease.

1.3 METABOLIC SYNDROME

Many of the biological risk factors for cardiovascular disease that relate to socioeconomic status also covary across populations and tend to aggregate within individuals. This clustering, now recognized as a syndrome of pernicious influence on CAD risk, is commonly known as the metabolic syndrome. This syndrome is defined by a concurrence of disturbed glucose and insulin metabolism, abdominal fat distribution, dyslipidemia and hypertension. Alone, each component of this cluster conveys increased CVD risk, and their effects are additive in combination (WHO, 1999). The metabolic syndrome is associated with development of type 2 diabetes mellitus and coronary heart disease (Lakka et al., 2002) and increased mortality from cardiovascular disease and all causes (Ford, Giles, & Dietz, 2002).

Although the mechanisms underlying the metabolic syndrome are not fully known, insulin resistance is considered a critical feature (Reaven, 1999; Grundy, 1999) due to its effects on multiple organ systems and relation to dyslipidemia, hypertension, and glucose intolerance (Grundy, 1999). Obesity, and central adiposity in particular, is a well-recognized risk factor for CHD and is thought to contribute to each component of the metabolic syndrome. Excess visceral adipose tissue is associated with insulin resistance, hyperinsulemia, glucose intolerance, hypertriglyceridemia, increased plasma low-density lipoprotein (LDL) cholesterol concentrations, and decreased levels of high-density lipoprotein (HDL) cholesterol (McFarlane, Banerji, & Sowers, 2001). These relationships suggest that the several empirically covarying components of the metabolic syndrome, as commonly seen in epidemiologic investigation, may stem, in part, from a common cause.

Factor analytic strategies have been employed to investigate whether the metabolic syndrome truly represents a unitary construct of potential common etiology. Employing confirmatory factor analysis, Shen et al. (2003) tested a hierarchical four-factor model of the structure of the metabolic syndrome, with insulin resistance, obesity, lipids, and blood pressure as first order factors, and the metabolic syndrome as a second-order factor representing the syndrome itself. They found support for this model, demonstrating that the metabolic syndrome was represented primarily by the insulin resistance and obesity factors, followed by the lipid factor, and, to a lesser extent, blood pressure. McCaffrey et al. (2004) used this approach in a different sample, showing similar results. These studies confirm a common latent factor underlying the covariation of component variables of the metabolic syndrome (hyperinsulemia, hyperglycemia, dyslipidemia, central obesity and hypertension), while indicating that the several components also have, in varying degrees, independent determinants.

In an effort to facilitate consistent diagnosis of this syndrome among researchers and clinicians, both the World Health Organization (WHO, 1999) and the Third Report of the Expert Panel on Detection, Evaluation, and Treatment of High Blood Cholesterol in Adults (NCEP) provided formal criteria delineating the metabolic syndrome (NIH, 2001). Although the definitions are largely similar, some significant criterion differences exist. Both acknowledge the association between obesity and the metabolic syndrome, but because the presence of abdominal obesity has a higher correlation with the metabolic risk factors than does elevated body mass index (NIH, 2001), the NCEP recommends use of waist circumference as opposed to BMI as a morphometric indicator. Building on the WHO and NCEP criteria, the International Diabetes Federation (IDF) introduced a slightly different definition of metabolic syndrome which included the presence of central adiposity as a necessary component of the syndrome. Compared with NCEP criteria, the IDF also used lower fasting glucose levels, and in comparison with the WHO definition, the IDF used more stringent criteria for low HDL cholesterol, blood pressure, and fasting glucose. Most recently, the NCEP revised its original metabolic syndrome definition by reducing the threshold for elevated fasting plasma glucose, allowing drug treatment for high triglycerides, low HDL-C levels, and/or elevated blood pressure to count as risk factors, and lowering the waist circumference threshold for Asian-Americans.

Utilizing early NCEP criteria, the Third National Health and Nutrition Examination Survey (NHANESIII; data from 1988-1994) estimated the prevalence of the metabolic syndrome in U.S. adults to be approximately 22% (Ford et al., 2002). Because the prevalence of obesity continued to increase in the U.S. during the 1990s (Flegal, Carroll, Ogden, & Johnson, 2002), it was thought that the prevalence of the metabolic syndrome among adults would also rise. Using data from the National Health and Nutrition Examination Survey (NHANES) 1999–2000, and

again using the original NCEP definition, the prevalence of metabolic syndrome in U.S. adults was approximately 27% (Ford, Giles, & Mokdad, 2004). This increase in the prevalence of the metabolic syndrome across time indicates the likelihood of future increases in the metabolic syndrome, as well as increases in type 2 diabetes, coronary heart disease, and mortality from cardiovascular disease.

1.4 ADULT SES ASSOCIATION WITH THE METABOLIC SYNDROME

Insofar as low SES has been found to predict most or all components of the metabolic syndrome, it follows that SES also covaries inversely with the syndrome itself. As predicted, studies investigating socioeconomic relationships with the full metabolic syndrome have reported an inverse social gradient among adults (Brunner et al., 1997; Lawlor, Ebrahim & Davey Smith, 2002; Park et al., 2003; Dallongeville et al., 2005). This association was similar for both men and women of European, African American, and Mexican American ethnicities, and persisted whether using income or employment grade as an indicator of SES. Thus, socioeconomic status appears to share a negative association with the metabolic syndrome.

1.5 CHILDHOOD SES AND HEALTH

Socioeconomic status is not static. It may vary across the life span, and therefore potentially affect health in various ways over time. Yet, most studies of SES and health have focused primarily on adult socioeconomic status, and have not considered SES effects over stages of the

life course. As there is much evidence that the development of cardiovascular disease begins as early as childhood (Berenson et al., 1992), approaches that focus only on adulthood may fail to detect important health influences from early life.

Several conventional CHD risk factors measured in childhood and adolescence have been shown to predict CHD and stroke decades later (Hemmingsson & Lundberg, 2004). Blood pressure measured in childhood is a positive predictor of blood pressure in adulthood (Nelson, Ragland, & Syme, 1992, Wattigney, Webber, Srinivasan, & Berenson, 1995), and is positively associated with cardiovascular mortality later in life (McCarron, Davey Smith, Okasha, & McEwan, 2000). Serum lipid and lipoprotein concentrations have been shown to track from childhood into young adulthood (Wattigney et al., 1995; Nicklas, Duvillard, & Berenson, 2002), as has body mass index (Guo & Chumlea, 1999; Magarey, Daniels, Boulton, & Cockington, 2003; Fuentes, Notkola, Shemeikka, Tuomilehto, & Nissinen, 2003) and plasma insulin concentration (Bao, Srinivasan & Berenson, 1996). Low plasma HDL cholesterol, high plasma triglyceride levels and high body mass index in childhood are associated with insulin insensitivity in young adulthood (Clausen, Ibsen, Ibsen, & Borch-Johnsen, 1996). Considering the predictive value of childhood health for adult cardiovascular morbidity and mortality, understanding the SES effects on health in early life might contribute unique information germane to lifetime cardiovascular risk.

Children from lower SES families typically suffer worse health outcomes than do children from higher SES families (Chen, Matthews, & Boyce, 2002). More specifically, an inverse relation exists between cardiovascular risk factors and childhood variation in socioeconomic status. SES is inversely related to smoking prevalence (Winkleby, Robinson, Sundquist, & Kraemer, 1999; Chen et al., 2002) and overweight in childhood (Willms,

Tremblay, & Katzmarzyk, 2003; Langnase, Mast, & Muller, 2002), and parents' low socioeconomic position has been associated with higher blood pressure in children (Dekkers, Snieder, van den Oord, & Treiber, 2002; Chen et al, 2002). Therefore, if cardiovascular risk factors tend to cluster in children of lower SES families, and these risk factors tend to track over time, it would follow that lower childhood SES would increase the likelihood of developing disease in adulthood.

Although youths' socioeconomic status is strongly associated with adulthood SES, trajectories to adult social standing are not perfectly predicted by one's social standing in childhood. Studies of intergenerational persistence of certain economic characteristics have estimated the correlation between parental and child education to be between 0.14 and 0.45, and income to be between 0.11 and 0.58 (Behrman & Taubman, 1990; Solon, 1992; Mulligan, 1999). These socioeconomic indices are thus imperfectly correlated between parents and their offspring, suggesting that measures of childhood SES may be predictive of health status and risk independently of social position in adulthood. Indeed, using retrospective data from the Longitudinal Study of Socio-Economic Health Differences, van de Mheen, Stronks, Bos, and Mackenbach (1997) reported finding that about 9 percent of the relation between general adult health and adult socioeconomic status could be attributed to childhood socioeconomic conditions. Interactions of adult socioeconomic position on adult health problems (e.g., COPD, CHD, hypertension, stroke, diabetes, cancer) declined when childhood characteristics (mother's education level, father's occupation, and financial situation of the family) were entered first into the regression model, demonstrating that SES differences in health were partly explained by childhood environment. In sum, these studies support the assertion that childhood socioeconomic

status has a significant effect on adult health, independent of one's socioeconomic status as an adult.

1.6 CHILDHOOD SES AS A PREDICTOR OF CHD

Many retrospective studies indicate that lower childhood SES, independently of adult social standing, is associated with increased risk of coronary heart disease outcomes, including angina, ischemic heart disease, and non-fatal MI (Notkola, Punsar, Karvonen, & Haapakoski, 1985; Kaplan & Salonen, 1990; Gliksman et al., 1995; Wannamathee, Whincup, Shaper, & Walker, 1996; Wamala, Lynch & Kaplan, 2001; Davey Smith, Ben-Shlomo, & Lynch, 2002; Lawlor, Davey Smith, & Ebrahim, 2004), although some have not found significant associations (Hasle, 1990; Marmot, Shipley, Brunner, & Hemingway, 2001). In their review of childhood socioeconomic circumstances (such as water quality, number of siblings, father's occupation, and mother's marital status) and adult mortality, Galobardes, Lynch and Davey Smith (2004) found an inverse relationship between SES and CHD mortality in most (Gillum & Paffenbarger, 1978; Notkola, Punsar, Karvonen, & Haapakoski, 1985; Vagero & Leon, 1994; Davey Smith, Hart, Blane, & Hole, 1998; Dedman, Gunnell, Davey Smith, & Frankel, 2001; Modin, 2003: Hart & Davey Smith, 2003), but not all (Frankel, Smith, & Gunnell, 1999; Gliksman et al., 1995) studies reviewed. Socioeconomic status in childhood (often measured by father's social class at varying time points) predicts several components of the metabolic syndrome in adulthood, although adult SES is typically a stronger predictor (Blane et al., 1996; Davey Smith et al., 1998; Brunner, Shipley, Blane, Davey Smith & Marmot, 1999; Marmot et al., 2001; Pensola & Martikainen, 2003). This general association holds for both men and women, although the

strength of the relationship for some risk factors varies between sexes. For instance, a study of the Whitehall II cohort showed childhood social position associated with adult weight in both men and women, whereas an association between SES and current HDL cholesterol was found only in women (Brunner et al., 1999). Waist to hip ratio (Poulton et al., 2002), body mass index (Blane et al., 1996), blood pressure (Poulton et al., 2002; Blane et al., 1996), triglyceride concentrations (Blane et al., 1996), and fasting glucose concentrations (Ebrahim, Montaner, & Lawlor, 2004) have all been shown to vary inversely with childhood social class, whereas a positive relationship has been reported for HDL cholesterol (Lawlor et al., 2002; Ebrahim et al., 2004).

Given the association of childhood SES with components of the metabolic syndrome in adulthood, it follows that childhood SES may similarly predict the clustering of risk factors that defines the metabolic syndrome in adulthood as well. Ebrahim, Montaner, and Lawlor (2004) examined the clustering of CHD risk factors by childhood social class, and found that the co-occurrence of risk factors was more common in women whose father's occupation was classified as manual (vs. non-manual). Four other studies have investigated the association of childhood socioeconomic status with components of the metabolic syndrome in adulthood. Lawlor, Ebrahim, and Davey Smith (2002) assessed these associations in British women aged 60-79 years, and found that lower childhood social class predicted an increased risk of adulthood insulin resistance, dyslipidemia and obesity, after adjusting for adult social class. Parker et al. (2004) reported no relation of childhood social class at ages 5 and 10 with the metabolic syndrome in British adults aged 49-51, although social class at birth was related to BMI and WHR in men and with serum triglycerides in women. In their investigation of Finnish adults aged 24–39 years, Kivimaki et al. (2006) found that low SES in childhood was associated with

increased blood pressure and central obesity for both men and women, but no association was found for the metabolic syndrome. In contrast, Lehman et al. (2005) found that childhood SES (as measured by parental education) was related to NCEP-defined metabolic functioning in a biracial sample of male and female CARDIA subjects aged 33-45. Despite some evidence supporting a relationship, the few studies that have attempted to elucidate childhood environmental associations with the metabolic syndrome are not entirely consistent. Also, only two of the investigations used the 2001 NCEP recommendations for determining metabolic syndrome, and none have used the 2005 NCEP or IDF criteria, possibly introducing some limitations in terms of comparisons of these results. Although these studies provide a useful beginning, additional research is warranted to better understand the role of childhood SES in influencing the development of adulthood metabolic syndrome.

1.7 PURPOSE

A growing body of evidence supports the existence of a common construct, termed the metabolic syndrome, that captures shared variability in obesity, insulin resistance, dyslipidemia, and hypertension (Shen et al., 2003; McCaffrey, Shen, Niaura, Muldoon, & Manuck, 2004). The co-occurrence of risk factors that define the metabolic syndrome is known to confer increased risk of coronary heart disease (Lakka et al., 2002) and cardiovascular mortality (Ford, Giles, & Dietz, 2002). The components of the metabolic syndrome cluster in childhood and are known to track into adulthood (Bao et al., 1994). This clustering has been shown to relate to parental social class, indicating the potential importance of childhood influences. Childhood SES predicts coronary morbidity and mortality in adulthood (Gliksman et al., 1995; Wannamathee et al.,

1996; Pensola & Martikainen, 2003), as well as individual components of the metabolic syndrome. Lower socioeconomic status in childhood also predicts an increased prevalence of the metabolic syndrome in adults, albeit in only two previous investigations (Lawlor, Ebrahim & Davey Smith, 2002; Ebrahim et al., 2004).

The purpose of the study was to determine whether childhood socioeconomic status covaries with the presence of the metabolic syndrome in a biracial adult population in the United States, and to determine whether this association is also independent of current (adulthood) socioeconomic position. Three primary hypotheses were addressed:

1. *Does lower childhood socioeconomic status predict presence of the metabolic syndrome in adults?*
2. *Does lower childhood socioeconomic status predict the metabolic syndrome in adulthood when controlling for adult SES?*
3. *Does childhood socioeconomic status predict values of each individual component of the metabolic syndrome?*

2.0 METHODS

2.1 OVERVIEW

This investigation included a representative sample of 972 men and women (aged 30 - 54) who participated in the University of Pittsburgh Adult Health and Behavior (A.H.A.B.) project between 2001 and 2004. Established in 2001, A.H.A.B. is a data registry of behavioral and biological traits of individuals residing in Southwestern Pennsylvania. Subjects participating in A.H.A.B. were evaluated on various physical and biological measures, general and specific personality questionnaires and inventories, diagnostic interviews, and cognitive and neuropsychological tasks. Participants also provided demographic information germane to both current and childhood socioeconomic position. For the purposes of this investigation, information regarding participants' biological assessments and past and current demographic data were utilized.

2.2 PARTICIPANTS

Individuals residing in the greater Pittsburgh area, aged 30 to 54, were contacted through neighborhood mailings. Exclusion criteria included: insulin-dependent diabetes, kidney or liver disease, cancer or myocardial infarction within the past year, bypass surgery or balloon

angioplasty, multiple sclerosis or other serious neurological condition, or current use of medication used to treat depression or anxiety. Current use or past history of anti-psychotic medication was also an exclusion criterion. Women who were currently pregnant were not eligible for the study. Participants received $175 upon completion of 4 sessions of data collection.

Subjects initially included 972 A.H.A.B. participants, but this was reduced to 947 subjects due to incomplete parental data in 25 participants. In order to maintain consistent between-family comparisons, analyses were limited to intact families only (both female and male parental figure present at ages 5 and 10), reducing the number of subjects to 883. Of these, 34 subjects were excluded from metabolic syndrome analyses due to the use of cholesterol lowering medication, while 5 others were missing blood measures and 1 subject was missing waist circumference data. The final metabolic syndrome sample (n = 843), with complete childhood and adult SES and physiological data, consisted of 410 men (371 Caucasian/39 African-American) and 433 women (381 Caucasian/52 African American). The analyses of individual cardiovascular risk factors were based on this same sample, although subjects using antihypertensive medication (n=50), dyslipidemic medication (n=1), and diabetic medication (n=2) were further excluded, as the use of these medications would confound continuous variable analysis of blood pressure, triglycerides, HDL cholesterol and fasting plasma glucose. The final samples for analysis of individual cardiovascular risk factors were as follows: systolic and diastolic blood pressure (n=793), fasting plasma glucose (n=841), HDL cholesterol (n=842), triglycerides (n=842), and waist circumference (n=843).

2.3 MEASURES

2.3.1 Socioeconomic Indicators

Several studies investigating the relationship between SES measures and coronary risk indicate that the association is strongest and most consistent for education (Jacobson & Thelle, 1988; Winkelby et al., 1992; Luepker et al., 1993; Steenland, Henley, Calle, & Thun, 2004). Childhood SES assessment included parental years of education by the time the participant was age 18 and highest parental educational level completed when the participant was 5 and 10 years of age. Parental years of education years ranged from one to twenty-four years of education. Highest parental educational level completed was reported as follows: no high school diploma, GED or high school diploma, some college - no degree, Bachelors degree, Masters degree or MD/PhD/J.D./PharmD. Parental education variables were normally distributed. Several analyses were performed for each participant, examining mothers', fathers', and highest household education for both "years" and "level" indicators. Correlations between the parental education measures varied (highest parental education years vs. mother's education years = .797, highest parental education years vs. father's education years = 0.885, mothers' vs. fathers' education years = 0.606) indicating that each indicator may provide unique information regarding the contribution of parental SES to adult metabolic syndrome. In order to simplify reporting of results, only highest household parental education "years" analyses are discussed in detail below and are provided in Tables 6 - 29. Although not presented in this manuscript, parental education "level" analyses were performed and are available upon request. Because correlations between parental education "level" measures when participants were age 5 and age 10 were high (r's $\geq$ 0.99), only age 5 "level" analyses were performed.

Individual-level indicators of socioeconomic status were obtained for each participant. Information regarding current SES included: number of years of education, highest educational level completed (same as highest parental level completed), and total family income (reported as < $10,000/year, $10,000 - $14,999/year, $15,000 - 24,999/year, $25,000 - $34,999/year, $35,000 - $49,999/year, $50,000 - 64,999/year, $65,000 - 80,000/year, or > $80,000/year). Primary analyses employed educational level and years of education as indices of current SES, and were used as covariates in the analyses. When examining highest household parental education years as the indicator of childhood SES, participant's own number of education years was employed as the current SES measure, as described in detail below. However, more comprehensive results of the association of subjects' educational indicators with the metabolic syndrome and its components are provided in Tables 6 – 29. Supplementary analyses were performed utilizing family income as an additional index of current SES, but as this measure failed to contribute to prediction, results of these analyses were not reported in this manuscript.

2.3.2 Metabolic Syndrome

Two sets of clinical criteria were used to define metabolic syndrome in this study. In 2001, the National Cholesterol Education Program's Third Report of the Expert Panel on Detection, Evaluation, and Treatment of High Blood Cholesterol in Adults provided a working definition of the metabolic syndrome. NCEP criteria require at least three of the following risk determinants for diagnosis of metabolic syndrome: 1) abdominal waist circumference ≥ 102 cm for men, ≥ 88 cm for women; 2) triglycerides ≥ 150 mg/dL; 3) HDL cholesterol < 40 mg/dL for men, < 50 mg/dL for women; 4) blood pressure ≥ 130 mmHg systolic or ≥ 85 mmHg diastolic; and 5) fasting plasma glucose concentration ≥ 110mg/dL. These criteria were modified in 2005

(Grundy et al.), allowing drug treatment for high triglycerides, low HDL-C levels, and/or elevated blood pressure to count as risk factors, reducing the threshold for elevated fasting plasma glucose to ≥ 100, and lowering the waist circumference cut point for Asian-Americans to ≥ 90 cm for men, ≥ 80 cm for women. The most recent NCEP definition for the metabolic syndrome was used as the primary dependent variable for this study. The International Diabetes Federation (IDF) introduced a slightly different definition of the metabolic syndrome in 2005. According to IDF criteria, central adiposity is a necessary component of the syndrome, with a waist circumference of ≥ 94 cm in men and ≥ 80 cm in women serving as a pre-requisite. Ethnicity-specific values were also recommended by IDF for Asian groups, which did not apply to this study due to the absence of Asian participants. In addition to waist circumference, at least two of the following criteria must be present to be diagnosed with IDF-defined metabolic syndrome: 1) triglycerides ≥ 150 mg/dL, or specific treatment for this lipid abnormality; 2) HDL cholesterol < 40 mg/dL for men, < 50 mg/dL for women, or specific treatment for this lipid abnormality; 3) blood pressure ≥ 130 mmHg systolic or ≥ 85 mmHg diastolic, or treatment for hypertension; and 4) fasting plasma glucose concentration ≥ 100mg/dL, or previously diagnosed Type II diabetes (IDF, 2004). IDF-defined metabolic syndrome was used as an alternative dependent variable for this investigation.

As previously noted, participants taking any cholesterol lowering medication (n=34) were excluded from all metabolic syndrome analyses, while those taking antihypertensive (n=59) and hypoglycemic (n=3) medication were included. The presence or absence of the metabolic syndrome was identified according to both NCEP (2005) and IDF criteria and analyzed as a dichotomous variable.

2.3.3 Cardiovascular Risk Factors

Waist measurement: Waist circumference was assessed by measuring girth at the midpoint between the iliac crest and the ribs.

Blood pressure: Systolic and diastolic blood pressure readings were taken in the right arm by common auscultation following a 5 minute seated rest. Participants' blood pressures were determined as the average of two consecutive measurements.

Blood measures: All blood-derived measures were determined from a morning fasting blood sample by the Heinz Nutrition Laboratory, School of Public Health, University of Pittsburgh, which has met criteria for the Centers for Disease Control and Prevention – National Heart, Lung and Blood Institute Standardization Program since 1982. Triglycerides were determined using the procedure of Bucolo et al. (1973), and the interassay coefficient of variation was 1.7%. High-density lipoprotein cholesterol was determined after selective precipitation by heparin/manganese chloride and removal by centrifugation of very low density and low density lipoprotein, and the coefficient of variation for HDL cholesterol was 2.1%. Serum glucose was quantitatively assessed by an enzymic determination read at 340/380 nm with a procedure similar to that described by Bondar and Mead (1974), and the coefficient of variation was 1.8%. Because distributions of triglycerides and serum glucose values were positively skewed, reciprocal transformations were performed to normalize these data. The transformed variables were used in the statistical analyses.

3.0 RESULTS

3.1 DESCRIPTIVE CHARACTERISTICS

Demographic characteristics and cardiovascular risk factors for the sample as a whole are listed in Table 1. Subjects' average age was 44, 16% of the sample smoked at the time of study, and 64% of subjects were married. Approximately 57% of subjects were employed full-time and 22% part-time. Average annual family income during the years of participation (2001-2004) was between $35,000 - $49,999, although this varied widely, and average number of years of completed education was 16, with educational levels reported as follows: Less than High School Diploma (0.7%), GED or High School graduate (14.7%), 1-3 years of college (22.4%), college graduate (35.9%), and graduate degree (26.2%). Subjects reported the highest level of parental education in their household at age 5 as: Less than high school diploma (8.3%), GED or high school graduate (48.3%), 1-3 years of college (11.3%), college graduate (19.6%), and graduate degree (12.6%). The sample's average fasting plasma glucose level of 95.7 reflects a population bordering on the pre-diabetic range, and triglyerides in men were at the high end of normal. HDL cholesterol (52.6 mg/dL) and average resting systolic blood pressure (115.7 mmHg) and diastolic blood pressure (77.7 mmHg) for the sample were within normal limits. Men had significantly higher systolic blood pressure, diastolic blood pressure, fasting plasma glucose, and waist circumference than women, as well as a worse lipoprotein profile.

Table 2 shows the percentage of participants meeting criteria for individual metabolic syndrome risk factors and the full syndrome. Men were more likely in general to have the metabolic syndrome than women, with 30.9% and 35.4% of men meeting NCEP and IDF criteria respectively, as compared to 13.6% and 17.3% of women meeting NCEP and IDF criteria. After re-examining the data using the entire sample of 902 participants (as opposed to limiting the analyses to intact families), these percentages do not change significantly for women (NCEP = 14.4% versus 13.6%, IDF =18.2% versus 17.3%) or men (NCEP = 31.1% versus 30.9%, IDF = 35.2% versus 35.4%). Therefore, the use of intact families for our analyses does not appear to bias the data in terms of the percentage of subjects with the metabolic syndrome.

Table 3 presents the univariate correlations between individual SES indicators and parental SES indicators for the total sample, and Table 4 examines these relationships for men and women separately. Subjects' own educational achievement correlated significantly with all measures of parental education, although correlations tended to be higher between women and their parents as compared to men. Family income was inconsistently correlated with measures of parental education for the total sample, and this relationship disappeared completely for men upon sex-specific analyses. Point biserial correlations (Table 5) revealed that higher levels on all SES measures were related to being Caucasian American, with the strongest relationships being with years of individual education, individual education level, and family income ($r = -0.21, -0.22, -0.22$; $p's < 0.01$).

Univariate associations of cardiovascular risk factors with measures of both childhood and current individual SES are presented in Tables 6 and 7. For women, several risk factors were inversely associated with both subjects' own education and that of their parents, including systolic blood pressure, diastolic blood pressure, waist circumference, and triglycerides ($p's <$

0.01). These relationships were less consistent in men, although one's own education did tend to be associated inversely with systolic blood pressure ($p < 0.05$), diastolic blood pressure, glucose, and waist circumference ($p's < 0.01$). Point biserial correlations revealed significant inverse associations between individual and parental education and the presence of metabolic syndrome in women ($p's < 0.01$) and one's own education and the metabolic syndrome according to IDF criteria ($p's < 0.01$) in men.

Associations of cardiovascular risk factors with demographic characteristics are presented in Tables 8 and 9. All risk factors were significantly related to sex ($p's < 0.01$), while age was associated significantly with higher resting systolic and diastolic blood pressures, glucose concentrations, HDL ($p's < 0.01$), and greater waist circumference ($p < 0.05$). Point biserial correlations revealed that the presence of the metabolic syndrome was significantly associated with older age (NCEP $p < .05$, IDF $p < .01$) and being male ($p's < 0.01$). Notably, race was not associated with having the metabolic syndrome when examining the total sample, but was significantly related to the syndrome's presence in women (NCEP $p < 0.01$, IDF $p < 0.05$).

3.2 HYPOTHESIS 1 - DOES LOWER CHILDHOOD SOCIOECONOMIC STATUS PREDICT PRESENCE OF THE METABOLIC SYNDROME IN ADULTS?

A series of hierarchical regressions was conducted to determine whether lower childhood SES predicts the presence of the metabolic syndrome in adulthood, controlling for other sociodemographic predictors. Logistic hierarchical regressions were used to test associations with metabolic syndrome, and age, sex, and race were included as covariates in the regression analyses. In the first set of hierarchical regressions, covariates were entered on Step 1, childhood

SES on Step 2, and an interaction term of childhood SES by sex on Step 3. (The results of an additional set of regression analyses, whereby an interaction term of childhood SES by race was entered on Step 3, failed to demonstrate a significant interaction of parental education with race. These results are not reported here.) Parental education level was used as an index of childhood SES, and this was measured in several ways as noted in *"Socioeconomic Indicators"* above. Results of all analyses examining the relationship of parental education indices with both NCEP and IDF-defined metabolic syndrome are presented in Tables 10-33. In order to simplify reporting of these analyses, only the results of highest household parental education years will be discussed in detail.

In Step 1 of the logistic regression analysis for NCEP-defined metabolic syndrome (Table 10), covariates were associated significantly with the presence of the syndrome ($X^2 = 31.4$, $p = 0.000$): age was a significant independent predictor of the syndrome (OR = 1.04; 95% CI = 1.02, 1.07; p = 0.002), as was sex (OR = 0.31; 95% CI = 0.22, 0.45; p = 0.000), but race was not (OR = 1.27; 95% CI = 0.75, 2.17; p = 0.370). Highest household parental education was not a significant predictor of metabolic syndrome when entered on Step 2 (OR = 0.99; 95% CI = 0.92, 1.06; p = 0.798) but did interact with sex on Step 3 (OR = 0.88; 95% CI = 0.78, 0.99; p = 0.027). In sex-specific analyses (Table 11), parental education was a significant independent predictor of the syndrome in women (OR = 0.86; 95% CI = 0.78, 0.95; p = 0.003) but not in men (OR = 0.99; 95% CI = 0.93, 1.07; p = 0.874). In women, after statistically accounting for age and race, a one year increase in parental education was associated with a fourteen percent decreased likelihood of having the metabolic syndrome as an adult. In sum, after multivariate adjustment for standard risk factors, childhood SES was found to predict the presence of the metabolic syndrome in middle aged women according to NCEP criteria.

Similar results were found when utilizing the remaining indices of parental education (mothers' and fathers'), as the tables below demonstrate. For example, in the sex-specific analyses in Table 19, mother's education was a significant independent predictor of NCEP-defined metabolic syndrome in women (OR = 0.86; 95% CI = 0.77, 0.95; p = 0.004) but not in men (OR = 0.98; 95% CI = 0.90, 1.07; p = 0.616). Table 27 shows similar findings for father's education. It appears that, although fathers' education predicts the presence of NCEP-defined metabolic syndrome significantly in women, it is a slightly weaker predictor as compared to highest household parental education or mothers' education alone.

3.3 HYPOTHESIS 2 - DOES LOWER CHILDHOOD SOCIOECONOMIC STATUS PREDICT THE METABOLIC SYNDROME IN ADULTHOOD WHEN CONTROLLING FOR CURRENT SES?

Again, to simplify reporting of analyses, only the results of highest household parental education years as it relates to the development of the metabolic syndrome utilizing NCEP criteria will be discussed in detail. A series of logistic regressions was again employed, entering covariates on Step 1, individual-level SES (as indexed by one's own years of education) on Step 2, and childhood SES (as indexed by highest household parental education years) on Step 3. Tables 12 and 13 display the results of the analyses.

Results for Step 1 are the same as in the previous analyses. In Step 2, subjects' own education (years) was a significant predictor of NCEP-defined metabolic syndrome (OR = 0.93; 95% CI = 0.87, 0.99; p = 0.037) when included in the model (Table 12). Parental education was not a significant predictor of NCEP-defined metabolic syndrome when entered on Step 3 (OR =

1.01; 95% CI = 0.94, 1.08; p = 0.901), but did interact with sex on Step 4 (OR = 0.88; 95% CI = 0.78, 0.99; p = 0.031). Sex-specific analyses (Table 13) revealed that subjects' education was not a significant predictor of NCEP-defined metabolic syndrome in women (OR = 0.95; 95% CI = 0.86, 1.06; p = 0.348) when including parental education in the model, while subjects' education marginally predicted the syndrome in men (OR = 0.78; 95% CI = 0.60, 0.99; p = 0.050). Parental education was a significant independent predictor of NCEP-defined metabolic syndrome in women (OR = 0.87; 95% CI = 0.79, 0.97; p = 0.008) but not in men (OR = 1.06; 95% CI = 0.89, 1.27; p = 0.527) after controlling for age, sex, race, and subjects' own education. Therefore, a one year increase in parental education was found to predict a thirteen percent decreased likelihood of developing the metabolic syndrome in women after adjustment for both covariates and subjects' education level. In sum, parental education was associated with NCEP-defined metabolic syndrome in women after adjustment for covariates, and remained so after adjusting also for participants' own education levels.

Similar results were found when utilizing the remaining indices of parental education (mothers' and fathers'), as the tables below demonstrate. For example, in the sex-specific analyses in Table 21, mother's education was a significant independent predictor of NCEP-defined metabolic syndrome in women (OR = 0.87; 95% CI = 0.78, 0.97; p = 0.011), but not in men (OR = 0.99; 95% CI = 0.91, 1.09; p = 0.944), after controlling for covariates and subjects' education. Table 29 shows similar findings for father's education. After controlling for subjects' education, fathers' education significantly predicts the presence of NCEP-defined metabolic syndrome in women. However, it appears that fathers' education is a slightly less powerful predictor of the syndrome as compared to highest household parental education or mothers' education.

3.4 HYPOTHESIS 3 - DOES CHILDHOOD SOCIOECONOMIC STATUS PREDICT VALUES OF EACH INDIVIDUAL COMPONENT OF THE METABOLIC SYNDROME?

A series of hierarchical linear regression analyses was performed in order to predict the effect of childhood SES on each component risk factor of the metabolic syndrome. Separate analyses were performed for each risk factor, with the risk factor entered as the dependent variable (abdominal waist circumference, triglycerides, HDL cholesterol, blood pressure, and fasting plasma glucose concentration). Sex, current age, and race were entered as predictors in Step 1, the measure of childhood SES was entered in Step 2, with an interaction term for sex entered in Step 3. Again, only one index of childhood SES (highest parental education years) will be discussed in detail here. Tables 34-96 provide results for all analyses, including analyses using mothers' and fathers' education years as the childhood SES measure.

In Step 1, covariates (age, sex, and race) accounted significantly for proportions of variance of each risk factor as follows: 15% of systolic blood pressure (Table 34), 14% of diastolic blood pressure (Table 46), 8% of fasting glucose (Table 58), 22% of HDL cholesterol (Table 67), 21% of waist circumference (Table 73), and 14% of triglycerides (Table 85). Age and sex were individually associated with all risk factors (p's < 0.01), with race being associated with systolic and diastolic blood pressures, waist circumference, and triglycerides (p's < 0.01), but not with fasting glucose (p=0.564) or HDL cholesterol (p=0.497). On step 2, highest household parental education accounted for differing proportions of the variance for each risk factor as follows: 0.7% of the variance in systolic blood pressure ($F_{1, 788} = 6.4$, p= 0.012) (Table 34), 0.7% of the variance in diastolic blood pressure ($F_{1, 788} = 6.5$, p= 0.011) (Table 46), 0.2% of the variance in waist circumference ($F_{1, 837} = 3.5$, p= 0.131) (Table 73), 1.6% of the variance in

triglycerides ($F_{1, 836}$ = 13.9, p= 0.000) (Table 85), and none of the variance in fasting glucose (Table 58) or HDL cholesterol (Table 67). Step 3 showed significant interactions of parental education with sex for systolic blood pressure (p<0.01), diastolic blood pressure (p<0.04), glucose (p<.03), HDL cholesterol (p<0.03), waist circumference (p<0.05), and triglycerides (p<0.01). Sex-specific analyses were performed to determine the proportion of variance accounted for by parental education in women and men for each of these risk factors, and are reported in Tables 35, 47, 59, 68, 74, and 86. Among women, highest household parental education years accounted for significant proportions of the variance in systolic blood pressure (2.8% of total variance, $F_{1, 404}$ = 12.9, p = 0.000), diastolic blood pressure (2.6% of total variance, $F_{1, 404}$ = 13.2, p = 0.000), waist circumference (1.2% of total variance, $F_{1, 429}$ = 7.0, p = 0.019) and triglycerides (5.9% of total variance, $F_{1, 428}$ = 26.4, p = 0.000). No significant effects of parental education on systolic blood pressure, diastolic blood pressure, waist circumference, or triglycerides were seen in men. For glucose, no significant effects of parental education were found for either women (glucose = 0.3% of total variance, $F_{1, 429}$ = 2.3, p = 0.207) or men (glucose = 0.6% of total variance, $F_{1, 403}$ = 2.9, p = 0.113). For HDL cholesterol, no significant effects of parental education were found for women (HDL = 0.3% of total variance, $F_{1, 428}$ = 2.6, p = 0.224), although significant effects were seen in men (HDL = 1.0% of total variance, $F_{1, 405}$ = 3.2, p = 0.035).

To determine whether parental education predicts the occurrence of these risk factors in adulthood above and beyond one's own education, results of analyses controlling for subjects' own education are reported in Tables 36/37, 48/49, 60/61, 69/70, 75/76, 87/88. A series of hierarchical regression analyses was performed by entering covariates on Step 1, individual-level

SES (as indexed by one's own education) on Step 2, and childhood SES (as indexed by highest household parental education years) on Step 3.

Results for Step 1 are the same as in the previous analyses. In Step 2, subjects' own education (years) accounted for differing proportions of the variance for each risk factor as follows: 0.3% of the variance in systolic blood pressure ($F_{1, 788} = 6.4$, $p= 0.012$) (Table 36), 0.5% of the variance in diastolic blood pressure ($F_{1, 788} = 6.5$, $p= 0.019$) (Table 48), 1.2% of the variance in fasting glucose ($F_{1, 835} = 10.38$, $p= 0.001$) (Table 60), 0.4% of the variance in HDL cholesterol ($F_{1, 836} = 0.63$, $p= 0.048$) (Table 69), 1.3% of the variance in waist circumference ($F_{1, 837} = 3.5$, $p= 0.001$) (Table 75), and 1.6% of the variance in triglycerides ($F_{1, 836} = 13.9$, $p= 0.006$) (Table 87). Step 3 revealed that parental education was a significant predictor of systolic blood pressure, diastolic blood pressure, and triglycerides, but not glucose, HDL cholesterol, or waist circumference after controlling for subjects' own education. Step 4 showed significant interactions of parental education with sex for: systolic blood pressure ($p<0.01$), diastolic blood pressure ($p<0.03$), glucose ($p<.02$), and triglycerides ($p<0.01$), and a marginally significant interaction with HDL cholesterol ($p<.06$). Sex-specific analyses were performed to determine the proportion of variance accounted for by parental education in women and men for each of these risk factors, and are reported in Tables 36, 48, 60, 69, 75, and 87.

The results of these sex-specific analyses show that, in Step 2, subjects' own education was a significant predictor of waist circumference (2.5% of total variance, $F_{1, 429} = 9.8$, $p = 0.006$), and triglycerides (2.6% of total variance, $F_{1, 428} = 8.1$, $p = 0.011$) in women, and of glucose (1.5% of total variance, $F_{1, 403} = 5.2$, $p = 0.010$), waist circumference (1.2% of total variance, $F_{1, 405} = 8.7$, $p = 0.013$) and triglycerides (1.0% of total variance, $F_{1, 405} = 5.2$, $p = 0.040$) in men. In Step 3, parental education remained a significant predictor of systolic blood pressure

(2.6% of total variance, $F_{1, 403} = 9.4$, $p = 0.001$), diastolic blood pressure (2.4% of total variance, $F_{1, 403} = 10.3$, $p = 0.000$), and triglycerides (4.0% of total variance, $F_{1, 427} = 20.5$, $p = 0.000$) in women. Highest household parental education years significantly predicted HDL cholesterol in men (1.1% of total variance, $F_{1, 404} = 5.2$, $p = 0.029$), although opposite from the hypothesized direction, with higher parental education predicting higher HDL cholesterol (as opposed to lower). In sum, after controlling for covariates and subjects' education, parental education was a significant predictor of several metabolic syndrome risk factors in women (systolic blood pressure, diastolic blood pressure, triglycerides) and one risk factor in men (HDL cholesterol).

Similar results were found when utilizing the remaining indices of parental education (mothers' and fathers'), as the tables demonstrate. For example, after controlling for covariates and subjects' education, mother's education was a significant independent predictor of systolic blood pressure (Table 41), diastolic blood pressure (Table 53), and triglycerides (Table 92), with no significant effects for men. Analyses of fathers' education demonstrated results similar to that of mothers' education. The significant prediction of HDL cholesterol in men was not found using mothers' or fathers' education, a result that differs from the use of highest household parental education as a predictor. Again, it appears that fathers' education is a slightly less powerful predictor of components of the metabolic syndrome as compared to highest household parental education or mothers' education.

In conclusion, after multivariate adjustment for standard risk factors, parental education (measured in several ways) was found to predict the presence of the metabolic syndrome in middle aged women according to NCEP criteria. This association remained after adjusting for subjects' own education levels. Also, after controlling for covariates and subjects' education, parental education was a significant predictor of several metabolic syndrome risk factors in

women (systolic blood pressure, diastolic blood pressure, triglycerides) and one risk factor in men (HDL cholesterol). Therefore, it appears that childhood SES, as indexed by parental education, is an important independent predictor of increased risk of coronary heart disease in middle aged adults in this sample, particularly women.

4.0 DISCUSSION

In this study we tested three primary hypotheses related to potential socioeconomic influences on metabolic syndrome in a nonpatient sample of adults having no history of coronary heart disease. Based on evidence that both current and childhood SES are associated with several cardiovascular risk factors and with the prevalence of coronary heart disease, and that current SES also predicts coronary heart disease, we hypothesized that childhood SES, as indexed by measures of parental education, would similarly predict the presence of the metabolic syndrome in adulthood. Results of our analyses partially support this hypothesis, as parental education consistently predicted metabolic syndrome (using both NCEP and IDF criteria) in women, but not men. We hypothesized further that childhood SES would predict metabolic syndrome independently of participants' own SES. Our results partially support this hypothesis, as parental education again consistently predicted the presence of the metabolic syndrome in adult women, after controlling for subjects' current education. However, this relationship was not found in men. Thirdly, we hypothesized that childhood SES would predict individual components of the metabolic syndrome, above and beyond participants' own current SES. Again, our results partially supported this hypothesis, with parental education predicting several risk factors, principally in women. Thus, parental education, as indexed by highest parental, mothers', and fathers' educational level and years of education, was associated in women with systolic and diastolic blood pressure and triglyceride concentrations, and father's education alone predicted

waist circumference, after accounting for both covariates and individual SES. In men, childhood SES, as measured by the highest household parental years of education, predicted HDL cholesterol in adulthood after controlling for covariates and participants' current SES, albeit in the direction opposite to our hypothesis.

These findings should be interpreted in the light of several study limitations. The retrospective nature of reported childhood conditions may involve recall bias, although this is fairly unlikely using educational variables. Cross-sectional data collection might impede the ability to assess the directional nature of the relationship between current SES and the metabolic syndrome. The possibility that having the metabolic syndrome somehow influences one's own SES through occupation or income (reduced ability to work, lower wages) exists, but this is unlikely to have affected educational level. Another possible limitation concerns the comprehensiveness of the SES measures. Although education is a widely used index of SES, and is related to income and occupation, these indices do not fully overlap. The inclusion of an occupational indicator might have encompassed an additional dimension of SES. Perhaps a more relevant limitation of this study is its generalizability. This sample is predominantly middle-aged, Caucasian American, and relatively well-educated, limiting the ability to generalize our findings to a broader spectrum of national population.

In this discussion, the relationship between sex and the presence of the metabolic syndrome will be reviewed in light of past findings in this area. The relationships of childhood SES, individual SES, and race with the metabolic syndrome are discussed. Apparent sex differences in the association of childhood SES with metabolic syndrome will also be discussed.

4.1 PREVALENCE OF THE METABOLIC SYNDROME

In our study, men were more likely to have the metabolic syndrome than women, with 30.9% and 35.4% of men meeting NCEP and IDF guidelines, respectively, as compared to 13.6% and 17.3% of women meeting these criteria. According to Ford et al (2002), the prevalence of metabolic syndrome in the United States is approximately 22% as measured by NCEP criteria. However, this fails to take sex differences into account. As reported by the National Cholesterol Education Program Expert Panel (2001), middle-aged men in particular have a high prevalence of the major CHD risk factors and are predisposed to abdominal obesity and the metabolic syndrome, whereas the onset of CHD is delayed by 10-15 years in women. Therefore, perhaps our data are picking up the expected "lag" in development of the metabolic syndrome between women and men. Wilson et al. (2005) provide further support for this argument in their examination of men and women from the Framingham Offspring Study. At baseline, men with a mean age of 50 had a 21.4% prevalence of the metabolic syndrome (according to NCEP guidelines), whereas prevalence in women of this age group was 12.5%. Prevalence of the metabolic syndrome increased to 38.8% and 23.6% in men and women respectively at the eight-year follow up. Prevalence rates for the metabolic syndrome in A.H.A.B., then, are within expected ranges.

4.2 CHILDHOOD SES

While individual SES is associated inversely with most disease outcomes, recent research demonstrates that similar associations exist between childhood SES and health. Several studies

indicate that lower childhood SES, independent of adult social standing, is associated with increased risk of coronary heart disease. Although adult SES is typically a stronger predictor, socioeconomic status in childhood predicts several components of the metabolic syndrome in adulthood. An inverse association between individual SES and the metabolic syndrome has been documented, but few studies to date have examined the potential contribution of variation in childhood circumstances to the development of metabolic syndrome in adulthood. These findings suggest that individual SES alone does not fully account for the effects of social position on health, including the metabolic syndrome.

In order to study this potential association, we utilized two indices of childhood SES; parental educational level and parental years of education. Because we analyzed intact households only, we were able to examine each household in three ways using each index; highest in household, mothers', and fathers' score on both of the indices noted above. Therefore, each household was examined six ways.

Childhood SES, as indexed by parental education, contributed independently to the presence of the metabolic syndrome in women in this study. After adjusting for both covariates and the contribution of individual SES, childhood SES predicted the metabolic syndrome significantly in women (OR = 0.77; 95% CI = 0.60, 0.97; p = 0.029), but not in men (OR = 1.06; 95% CI = 0.89, 1.27; p = 0.527). A one year increase in the highest household parental education was found to predict a fourteen percent decreased likelihood of having the metabolic syndrome in adult women. Although research to date has shown that childhood SES, independently of individual SES, predicts components of the metabolic syndrome (Lawlor, Ebrahim, & Davey Smith, 2002; Ebrahim, Montaner & Lawlor 2004; Lehman et al., 2005), this

is the first study, to our knowledge, to document a relationship between childhood SES and the full metabolic syndrome as diagnosed by both NCEP (2005) and IDF criteria.

Our results are consistent with evidence showing childhood SES to predict several components of the metabolic syndrome in adulthood (Blane et al., 1996; Davey Smith et al., 1998; Brunner, Shipley, Blane, Davey Smith & Marmot, 1999; Marmot et al., 2001; Pensola & Martikainen, 2003; Lawlor et al., 2002; Parker et al., 2004), as well as the metabolic syndrome itself (Ebrahim et al., 2004; Lehman et al., 2005). This general association exists for both men and women, although the strength of the relationship for some risk factors has been shown to vary between sexes. The fact that an association between childhood SES and adult metabolic syndrome was found only in women in our sample is intriguing, and is similar to some prior investigations.

Previous studies examining childhood environmental associations with the metabolic syndrome in both men and women are not entirely consistent regarding sex differences. As in our study, Lehman et al. (2005) found that childhood SES was associated with adulthood metabolic functioning in women, a relationship that was not significant for men. Although Parker et al. (2004) reported no relation of childhood social class with the metabolic syndrome in middle-aged British adults, social class at birth was related to BMI and WHR in men and to serum triglycerides in women. In contrast, Kivimäki et al. (2006) found that lower childhood SES was associated with greater waist circumference in both men and women, but was not associated with the metabolic syndrome in either sex. Although the mixed nature of these findings precludes any firm conclusion regarding sex differences, the lack of a relationship between childhood SES and the metabolic syndrome in men in our sample warrants further exploration.

An examination of possible mechanisms linking childhood SES and adult metabolic syndrome may shed some light on the observed sex difference in our findings. There are numerous ways in which parental education might affect cardiovascular risk factors and the metabolic syndrome, and one such mechanism may be the sustained influence of early socioeconomic circumstances on health behaviors. According to Lehman et al. (2005), education may act as a marker for material conditions and often facilitates understanding of the importance of healthy lifestyles. Lower education may predict poor food choices and less physical activity, two factors that are related to the metabolic syndrome. In support of this link, physical activity was related to the metabolic syndrome in our sample, with self-reported exercise (kcal/wk) as measured by the Paffenbarger Physical Activity Questionnaire significantly predicting the NCEP-defined syndrome (OR = 0.65; 95% CI = 0.53, 0.79; p = 0.000).

It is possible that childhood SES is related to physical activity differently in men versus women. In fact, gender differences in physical activity are well-established, with boys generally being more active than girls (Sallis, Prochaska, & Taylor, 2000; Inchley et al., 2005). In their study of Scottish adolescents, Inchley et al. (2005) found that girls from the highest SES groups were less active than boys from the lowest SES groups, suggesting an additive effect of gender and SES that places girls from low SES backgrounds at particular risk of low physical activity. For women in our sample, physical activity patterns may have varied depending upon the educational level of their parents, whereas the men in the sample may have been physically active independent of their SES in childhood. In support of this hypothesis, additional analyses using the Paffenbarger Physical Activity Questionnaire show that higher parental education predicted significantly more weekly physical activity in women in our sample (0.02% of total variance, $F_{1, 429} = 7.9$, p = 0.003), but not in men. Although the addition of physical activity

scores to the original logistic regression model resulted in a reduction of the parental education by sex interaction, it did not fully account for the relationship between childhood SES and metabolic syndrome. Parental education remained a significant independent predictor of NCEP-defined metabolic syndrome in women (OR = 0.88; 95% CI = 0.80, 0.98; p = 0.017), but not in men, after controlling for age, sex, race, subjects' own education, and physical activity.

Another hypothesized link between childhood SES and adult health is that of psychosocial influences. Social, behavioral and biological stimuli perceived as "stressful" may play a role in the etiology of the metabolic syndrome (Raikkonen, Keltikangas-Jarvinen, Adlercreutz, & Hautanen, 1996). Psychosocial stress has been associated with several components of the metabolic syndrome, including abdominal fat (Lapidus et al., 1989; Wing et al., 1991; Raikkonen, Hautanen, & Keltikangas-Jarvinen, 1994; Bjorntorp & Rosmond, 1999), insulin resistance (Raikkonen, Keltikangas-Jarvinen, & Hautanen, 1994; Black, 2003), atherogenic lipid profiles (Niaura, Stoney, & Herbert; 1992; Brunner et al., 1993; Richards, Hof, & Alvarenga, 2000**)** and hypertension (Jonas, Franks, & Ingram. 1997; Lijing et al., 2003). Education may reflect material conditions associated with family and financial stress, with lower parental education indicating a more "risky" family environment that might lead to adverse alterations in biological systems related to metabolic dysregulation. Perhaps men in our sample were less influenced by risky childhood environmental circumstances than were women, decreasing the association of childhood environment and later metabolic syndrome development.

Childhood SES may contribute to adult health through pathways that do not involve behavioral or psychosocial factors. Several studies have suggested that cardiovascular and metabolic risk are related inversely to birth weight (Reynolds et al., 2005), and the association between size at birth and mortality in adult life may be due to socioeconomic factors (Leon et al.,

1996). Reduced size at birth may be a marker for poor maternal socioeconomic circumstances that may result in both dysregulation of developing biological systems and inadequate medical or nutritional resources for the child (Osmond et al., 1993). Although research examining sex differences with regard to these associations in humans has been mixed (Phillips et al., 2000; Reynolds et al., 2005), some animal studies have found female rats to be more sensitive than males to activation of the HPA axis (Weinstock et al., 1992), suggesting that relationships between birth size and cardiovascular risk may not be the same in men and women. In terms of the sex differences found in the present study, perhaps these perinatal complications have more serious and long-lasting biological effects on daughters than sons.

The mechanisms explored here may partly account for the observed sex difference in the relationship between childhood SES and metabolic syndrome in this and one previous study. However, these mechanisms do not explain the fact that our findings differ from those of two other studies. Although sample differences between the two sets of studies may be responsible for this difference, the nature of these differences remains unclear.

4.3 CURRENT SES

Although childhood and individual SES are linked conceptually and tend to correlate, childhood SES contributed independently to the presence of the metabolic syndrome in women in the present study. As expected, individual-level SES, as indexed by years of education, did predict the presence of the metabolic syndrome when examining the sample as a whole (Table 12). A one-standard deviation increase in individual-level SES, as indexed by years of education, predicted a 7% decrease in the likelihood of developing metabolic syndrome, after accounting

for the contribution of covariates. Interestingly, in sex-specific analyses (Table 13), subjects' own education predicted the presence of metabolic syndrome significantly in men, while no significant effect was found in women. These results partially support other cross-sectional findings in the literature showing individual SES to predict metabolic syndrome in adults (Brunner et al., 1997; Lawlor et al., 2002; Park et al., 2003).

4.4 NON-INTACT FAMILIES

The reduced economic well-being of children in single-parent families is well-documented (Bumpass, 2004). It is possible that, by excluding subjects from non-intact families, this investigation removed a number of lower childhood SES individuals from the analyses. To examine this possibility, current and parental SES information between subjects from intact families and individuals who had only one parental figure present at age 5 were compared. Because it is not known which of the parents was present at age 5, only highest parental education levels for the household were utilized in the analyses. As seen in Table 106, all current and parental SES measures differ significantly between the intact and non-intact family subjects. In order to test whether this would change predictive value of childhood SES in the final analyses, additional logistic regression analyses were performed on the entire sample (n=903; includes both intact and non-intact families), using only highest parental household education (years and level) as independent predictor variables. No significant differences were detected after analyzing all subjects, as opposed to the participants whose families had both parents present at age 5. Utilizing non-intact families slightly strengthened the predictive value of parental education for developing the metabolic syndrome in women (OR = 0.71; 95% CI =

0.57, 0.87; p = 0.001), and this remained true after controlling for subjects' own educational levels (OR = 0.73; 95% CI = 0.59, 0.91; p = 0.006). In sum, the exclusion of non-intact families from the initial analyses did not significantly change the final results.

4.5 RACE

Race did not act as a predictor for developing the metabolic syndrome in this study. It may be that racial differences in the development of the syndrome do not exist, or it may be that our study did not have a high enough proportion of African American participants to address this issue meaningfully. Table 104 shows the racial breakdown of subjects having met NCEP criteria for the metabolic syndrome. Only 10 African American males and 13 African American females out of 186 individuals met criteria for the metabolic syndrome. This small proportion of individuals does not allow us to make accurate inferences about whether race influences the development of the metabolic syndrome in this study.

Because African American individuals in the United States tend to be over-represented in lower SES groups (Dressler, Oths, & Gravlee, 2005), it is possible that our restriction of analyses to intact families caused a critical loss of African American participants. By excluding non-intact families, a slight shift from 87% white and 13% black to 89% white and 11% black occurred. The addition of non-intact families adds another 19 people to the total having metabolic syndrome (205 out of 903); these include 5 black males and 2 black females. A childhood SES by race interaction term was entered in supplementary analyses of both intact and non-intact families, but no significant interaction was found. Thus, despite additional analyses including non-intact families, race did not predict the metabolic syndrome in this study.

5.0 CONCLUSIONS

Childhood SES predicts metabolic syndrome in a biracial, asymptomatic sample of women, implicating childhood socioeconomic factors in the development of disease in adulthood. These results suggest that parental education levels are important in determining vulnerability to developing the metabolic syndrome, which is associated with development of type 2 diabetes mellitus and coronary heart disease. The association between childhood SES and metabolic syndrome is independent of individual SES in women, suggesting that the socioeconomic characteristics of one's childhood provide an added risk or benefit beyond individual SES. This association was dependent upon sex; childhood SES interacted significantly with sex in predicting the metabolic syndrome. Age was associated with the metabolic syndrome in males but not females. The association between parental education and metabolic syndrome was independent of race; although African American subjects had significantly lower individual and parental education (Table 103), race was not itself associated with metabolic syndrome ($r = 0.02$, $p = 0.57$), nor did childhood SES interact with race. Finally, childhood SES remained a significant predictor of metabolic syndrome in women after accounting for both covariates and individual-level SES.

Similar associations were found for several of the cardiovascular risk factors that constitute the metabolic syndrome. In women, highest household parental education predicted systolic blood pressure, diastolic blood pressure, and triglycerides, and father's education was

related significantly to waist circumference after accounting for both covariates and individual SES. In men, higher parental education predicted lower HDL cholesterol in adulthood after controlling for covariates and participants' own educational attainment, but not in the expected direction.

Several mechanisms by which parental education might affect cardiovascular risk and the metabolic syndrome were discussed. Education promotes healthy lifestyles, and lower education may be related to the metabolic syndrome through poor nutritional and activity practices. Individuals in lower SES households might encounter greater levels of chronic stress, resulting in negative emotional states that may predict the development of the metabolic syndrome (Räikkönen, Matthews, & Salomon, 2003). Childhood SES may also contribute to adult health through other pathways, such as poor maternal socioeconomic circumstances that may result in dysregulation of a range of metabolic and endocrine systems during fetal growth, as well as predict material deprivation of the child in early life (Osmond et al., 1993).

While the current SES of an individual predicts exposure to certain health-impairing conditions, conditions in childhood appear to contribute additional risk beyond that which is accounted for by one's current socioeconomic status. Specifically for this sample, childhood SES proved to be a risk factor in women for development of the metabolic syndrome. Future investigations should focus on elucidating the mechanisms by which childhood SES is associated with metabolic syndrome and its associated risk factors. Targeting preventive measures to individuals at greater risk of developing the metabolic syndrome might improve the effectiveness of interventions aimed at reducing CHD morbidity.

6.0 TABLES

Table 1: *Demographic and cardiovascular risk factor characteristics for the total sample and for male and female participants separately*

	Total Sample N=843	Males N=410	Females N=433	Test Statistic
Age, years	44.2 (6.9)	43.9 (7.0)	44.6(6.7)	F_{843}=1.18
Race (%AA)	10.8	9.5	12.0	χ^2=1.36
Employed (%)	78.9	82.7	75.3	χ^2=6.91**
Smoker (%)	16.0	18.8	13.4	χ^2=4.54*
Married (%)	64.3	64.1	64.4	χ^2=0.01
Subj. Ed., yrs	16.1 (2.9)	16.2 (2.6)	16.0 (3.1)	F_{843}=1.36
Subj. Ed., level	5.7 (1.0)	5.8 (1.0)	5.7 (1.1)	F_{843}=2.70
Family Income	5.5 (2.0)	5.4 (2.0)	5.6 (2.1)	F_{843}=0.69
Father Ed., yrs.	13.0 (3.6)	13.1 (3.5)	12.9 (3.8)	F_{843}=0.55
Mother Ed., yrs.	12.7 (2.7)	12.7 (2.6)	12.6 (2.9)	F_{843}=0.63
Parent Ed., yrs.	13.8 (3.3)	13.8 (3.1)	13.8 (3.4)	F_{843}=0.03
Parent Ed., level	4.7 (1.4)	4.7 (1.4)	4.7 (1.4)	F_{843}=1.18
SBP, mm Hg	116.5 (13.7)	120.1 (12.8)	112.5 (13.3)	F_{843}=91.3*
DBP, mm Hg	78.1 (9.4)	81.3 (9.0)	75.2 (8.7)	F_{843}=99.7*
Glucose, mg/dL	95.7 (16.8)	99.0 (20.4)	92.5 (11.5)	F_{843}=34.8**
HDL, mg/dL	52.6 (14.4)	45.8 (11.4)	59.0 (14.1)	F_{843}=221.4**
Waist circ., cm	91.2 (15.5)	98.3 (13.3)	84.4 (14.4)	F_{843}=210.1**
Triglyceride,mg/dL	122.9 (84.1)	148.6 (101.6)	98.5 (52.7)	F_{843}=82.2**

Family Income (per year) coded as: 1 = less than $10,000, 2 = $10,000-$14,999, 3 = $15,000 - $24,999, 4 = $25,000 - $34,999, 5 = $35,000 - $49,999, 6 = $50,000 - $64,999, 7 = $65,000 - $80,000, 8 = greater than $80,000. Subject and Parental Education level coded as: 1 = <7th grade, 2 = 9th grade, 3 = 10 or 11th grade, 4 = HS grad, 5 = 1-3 yrs college, 6 = college grad, 7 = graduate degree

**P<.01, *P<.05, ^P<.10

Table 2: *Percentage of participants meeting criteria for individual metabolic syndrome risk factors and full syndrome*

	NCEP – sample %	NCEP- male/female %	Test Statistic	IDF – sample %	IDF – male/female %	Test Statistic
No criteria	31.6	22.1/40.6	χ^2=20.5**	35.6	23.4/47.1	χ^2=38.9**
1 criterion	25.2	22.9/27.5	χ^2=0.93	32.4	30.2/34.4	χ^2=2.28
2 criteria	21.1	24.1/18.2	χ^2=2.96^	19.6	26.3/13/2	χ^2=15.8**
3 criteria	13.0	17.3/9.0	χ^2=4.44*	9.3	15.1/3.7	χ^2=27.1**
4 criteria	6.5	10.0/3.2	χ^2=14.4**	3.2	4.9/1.6	χ^2=6.26*
5 criteria	2.5	3.6/1.4	χ^2=1.60	-	-	-
M.S.	22.0	30.9/13.6	χ^2=16.5**	26.1	35.4/17.3	χ^2=22.3*

M.S. – Metabolic Syndrome.
**P<.01, *P<.05, ^P<.10

Table 3: *Univariate correlations between subject and parental SES for total sample*

SES measure	Parent Ed. Years r	Father's Ed. Years r	Father's Ed. Level r	Mother's Ed. Years r	Mother's Ed. Level r
Subj. Ed. Years	0.32**	0.29**	0.29**	0.29**	0.27**
Subj. Ed. Level	0.30**	0.28**	0.29**	0.27**	0.25**
Family Income	0.07*	0.06	0.08*	0.12**	0.11**

**P<.01, *P<.05

Table 4: *Univariate correlations between subject and parental SES by sex*

SES measure	Parent Ed. Years r m/f	Father's Ed. Years r m/f	Father's Ed. Level r m/f	Mother's Ed. Years r m/f	Mother's Ed. Level r m/f
Subj. Ed. Years	0.26**/0.37**	0.24**/0.33**	0.26**/0.32**	0.24**/0.33**	0.25**/0.28**
Subj. Ed. Level	0.26**/0.33**	0.26**/0.30**	0.28**/0.29**	0.23**/0.30**	0.24**/0.26**
Family Income	0.05/0.08	0.06/0.06	0.06/0.10*	0.07/0.16**	0.07/0.16**

**P<.01, *P<.05

Table 5: *Correlations between race and parental and subject SES*

SES measure	Race r_{pb}
Subject Education (years)	-0.212**
Subject Education (level)	-0.218**
Family Income	-0.224**
Highest Parental Ed. (years)	-0.109**
Father's Education (years)	-0.135**
Father's Education (level)	-0.146**
Mother's Education (years)	-0.069*
Mother's Education (level)	-0.093**

**P<.01, *P<.05; Race coded 0-Caucasian, 1-African American

Table 6: *Univariate correlations between cardiovascular risk factors, metabolic syndrome, and parental and subject SES*

	Subject Education (years) *r*	Family Income *r*	Highest Parental Ed. (years) *r*	Father's Education (years) *r*	Mother's Education (years) *r*
SBP, mm Hg	-.110**	-.030	-.147**	-.138**	-.138**
DBP, mm Hg	-.109**	-.013	-.136**	-.124**	-.112**
Glucose, mg/dL	-.089**	.011	-.029	-.039	-.050
HDL, mg/dL	.026	.009	-.026	-.029	-.042
Waist Circumference, cm	-.130**	-.068	.074*	-.086*	-.068*
Triglyceride, mg/dL	-.060	-.041	-.087*	-.087**	-.065
Metabolic syndrome (NCEP)	†-.086*	†-.039	†-.063	†-.078*	†-.080*
Metabolic syndrome (IDF)	†-.121**	†-.023	†-.099**	†-.100**	†-.105**

**P<.01, *P<.05, †r_{pb} = point biserial correlation.

Table 7: *Univariate correlations between cardiovascular risk factors, metabolic syndrome, and parental and subject SES by sex*

	Subject Education (years) *r*	Family Income *r*	Highest Parental Ed. (years) *r*	Father's Education (years) *r*	Mother's Education (years) *r*
SBP, mm Hg	-.11*/-.15**	-.02/-.02	-.05/-.24**	-.07/-.22**	-.06/-.23**
DBP, mm Hg	-.13**/-.13**	-.01/.01	-.04/-.23**	-.05/-.21**	-.04/-.20**
Glucose, mg/dL	-.14**/-.06	-.07/.13**	-.10*/.06	-.13**/.05	-.10*/-.00
HDL, mg/dL	-.02/.10	-.04/.02	-.12*/.03	-.11*/.04	-.09/.00
Waist Circumference, cm	-.14**/-.19**	-.09/-.04	-.01/-.14**	-.02/-.17**	-.03/-.13**
Triglyceride, mg/dL	-.03/-.17**	-.03/-.04	-.02/-.24**	-.04/-.21	.01/-.24**
Metabolic syndrome (NCEP)	†-.06/-.14**	†-.03/-.04	†.01/-.15**	†-.04/-.14**	†-.04/-.14**
Metabolic syndrome (IDF)	†-.13**/-14**	†-.03/-.01	†-.05/-.16**	†-.07/-.15**	†-.07/-.15**

**P<.01, *P<.05, †r_{pb} = point biserial correlation.

Table 8: *Univariate correlations between cardiovascular risk factors, metabolic syndrome, and demographic indicators*

	Age r	Sex r_{pb}	Race r_{pb}
SBP, mm Hg	.183**	-.301**	.137**
DBP, mm Hg	.144**	-.323**	.105**
Glucose, mg/dL	.149**	-.170**	.023
HDL, mg/dL	.129**	.455**	-.003
Waist Circumference, cm	.073*	-.442**	.073*
Triglyceride, mg/dL	.060	-.306**	-.111**
Metabolic syndrome (NCEP)	†.084*	†-.165**	†.019
Metabolic syndrome (IDF)	†.102**	†-.205**	†.028

**P<.01, *P<.05; Sex coded 0-male, 1-female; Race coded 0-Caucasian, 1-African American, $^{\dagger}r_{pb}$ = point biserial correlation

Table 9: *Univariate correlations between cardiovascular risk factors, metabolic syndrome, and demographic indicators by sex*

	Sex	Race
	r_{pb} m/f	r_{pb} m/f
SBP, mm Hg	.202**/.207**	.086/.219**
DBP, mm Hg	.151**/.180**	.071/.176**
Glucose, mg/dL	.156**/.165**	.063/-.005
HDL, mg/dL	.078/.163**	.065/-.087
Waist Circumference, cm	.150**/.056	.039/.153**
Triglyceride, mg/dL	.055/.128**	-.123**/-.090
Metabolic syndrome (NCEP)	†.153**/.013	†-.058/.126**
Metabolic syndrome (IDF)	†.161**/.056	†-.031/.113*

**P<.01, *P<.05; Race coded 0-Caucasian, 1-African American, $^{\dagger}r_{pb}$ = point biserial correlation

Table 10: *Predictive analyses of metabolic syndrome by covariates and highest parental education years***.**

Logistic Regression	b	Odds Ratio (95% CI)	p
DV: Metabolic syndrome (NCEP)			
Step 1:			
Sex	-1.17	0.31 (0.22, 0.45)	0.000
Age	0.04	1.04 (1.02, 1.07)	0.002
Race	0.24	1.27 (0.75, 2.17)	0.370
Step 2:			
Parental Ed. Years	-0.01	0.99 (0.92, 1.06)	0.798
Step 3:			
Parental Ed. Years by Sex	-0.13	0.88 (0.78, 0.99)	0.027

Table 11: *Sex-Specific predictive analyses of metabolic syndrome by covariates and highest parental education years*

Logistic Regression	b f/m	Odds Ratio (95% CI) f/m	p f/m
DV: Metabolic syndrome (NCEP)			
Step 1:			
Age	0.01/0.06	0.88(0.96,1.05)/1.06 (1.03,1.10)	0.877/0.000
Race	0.73/-0.24	2.07(1.01,4.24)/0.79 (0.36,1.70)	0.048/0.541
Step 2:			
Highest Parental Ed. Years	-0.15/-0.01	0.86(0.78,0.95)/0.99 (0.93,1.07)	0.003/0.874

Table 12: *Predictive analyses of metabolic syndrome by covariates, subject education level, and highest parental education years*

Logistic Regression	b	Odds Ratio (95% CI)	p
DV: Metabolic syndrome (NCEP)			
Step 1:			
Sex	-1.19	0.30 (0.21, 0.44)	0.000
Age	0.04	1.04 (1.02, 1.07)	0.002
Race	0.04	1.11 (0.64, 1.91)	0.719
Step 2:			
Subject Ed. Years	-0.07	0.93 (0.87, 0.99)	0.037
Step 3:			
Parental Ed. Years	0.01	1.01 (0.94, 1.08)	0.901
Step 4:			
Parental Ed. Years by Sex	-0.13	0.88 (0.78, 0.99)	0.031

Table 13: *Sex-Specific predictive analyses of metabolic syndrome by covariates, subject education level, and highest parental education years*

Logistic Regression	b	Odds Ratio (95% CI)	p
DV:Metabolic syndrome (NCEP)			
Step 1:			
Age	0.01/0.06	1.00(0.96, 1.05)/ 1.06(1.02,1.10)	0.861/0.003
Race	0.66/-0.76	1.94(0.94,4.03)/ 0.47(0.18,1.21)	0.075/0.117
Step 2:			
Subject Ed. Years	-0.05/-0.25	0.95(0.86,1.06)/ 0.78(0.60,0.99)	0.348/0.050
Step 3:			
Highest Parental Ed. Years	-0.14/0.06	0.87(0.79,0.97)/1.06(0.89,1.27)	0.008/0.527

Table 14: *Predictive analyses of metabolic syndrome by covariates and highest parental education years***.**

Logistic Regression	b	Odds Ratio (95% CI)	p
DV: Metabolic syndrome (IDF)			
Step 1:			
Sex	0.04	1.04 (1.01, 1.06)	0.005
Age	-1.05	0.35 (0.25, 0.49)	0.000
Race	0.24	1.26 (0.77, 2.09)	0.360
Step 2:			
Parental Ed. Years			
Step 3:	-0.02	0.98 (0.92, 1.05)	0.555
Parental Ed. Years by Sex			
	-0.10	0.90 (0.81, 1.01)	0.065

Table 15: *Sex-Specific predictive analyses of metabolic syndrome by covariates and highest parental education years*

Logistic Regression	b f/m	Odds Ratio (95% CI) f/m	p f/m
DV: Metabolic syndrome (IDF)			
Step 1:			
Age	0.01/0.05	1.01(0.97,1.05) / 1.05(1.02,1.08)	0.566/0.002
Race	0.64/-0.20	1.89(0.96,3.72) / 0.82(0.40,1.70)	0.065/0.597
Step 2:			
Highest Parental Ed. Years	-0.12/-0.02	0.88(0.81,0.96) / 0.98(0.92,1.05)	0.004/0.628

Table 16: *Predictive analyses of metabolic syndrome by covariates, subject education level, and highest parental education years*

Logistic Regression	b	Odds Ratio (95% CI)	p
DV: Metabolic syndrome (IDF)			
Step 1:			
Sex	-1.08	0.40 (0.24, 0.48)	0.000
Age	0.03	1.04 (1.02, 1.06)	0.006
Race	0.05	1.05 (0.63, 1.77)	0.844
Step 2:			
Subject Ed. Years	-0.10	0.91 (0.85, 0.97)	0.004
Step 3:			
Parental Ed. Years	-0.01	0.99 (0.93, 1.07)	0.949
Step 4:			
Parental Ed. Years by Sex	-0.60	0.91 (0.81, 1.01)	0.079

Table 17: *Sex-Specific predictive analyses of metabolic syndrome by covariates, subject education level, and highest parental education years*

Logistic Regression	b	Odds Ratio (95% CI)	p
DV:Metabolic syndrome (IDF)			
Step 1:			
Age	0.01/0.05	1.01(0.97,1.05) / 1.05(1.02,1.08)	0.543/0.003
Race	0.54/-0.51	1.72(0.87,3.43) / 0.60(0.28,1.30)	0.121/0.199
Step 2:			
Subject Ed. Years	-0.08/-0.12	0.92(0.84,1.02) / 0.89(0.81,0.97)	0.102/0.009
Step 3:			
Highest Parental Ed. Years	-0.10/-0.01	0.90(0.82,0.99) /1.00(0.93,1.08)	0.024/0.918

Table 18: *Predictive analyses of metabolic syndrome by covariates and mother's education years***.**

Logistic Regression	b	Odds Ratio (95% CI)	p
DV: Metabolic syndrome (NCEP)			
Step 1:			
Sex	-1.15	0.32 (0.22, 0.46)	0.000
Age	0.04	1.04 (1.02, 1.07)	0.002
Race	0.29	1.33 (0.78, 2.26)	0.292
Step 2:			
Mother's Ed. Years			
Step 3:	-0.03	0.97 (0.90, 1.06)	0.529
Mother's Ed. Years by Sex			
	-0.12	0.89 (0.78, 1.01)	0.080

Table 19: *Sex-Specific predictive analyses of metabolic syndrome by covariates and mother's education years*

Logistic Regression	b f/m	Odds Ratio (95% CI) f/m	p f/m
DV: Metabolic syndrome (NCEP)			
Step 1:			
Age	0.01/0.06	1.00(0.96,1.05)/ 1.06(1.03,1.10)	0.893/0.000
Race	0.82/-0.24	2.27(1.10,4.66) / 0.79(0.36,1.70)	0.026/0.542
Step 2:			
Mother's Ed. Years			
	-0.16/-0.02	0.86(0.77,0.95) / 0.98(0.90,1.07)	0.004/0.616

Table 20: *Predictive analyses of metabolic syndrome by covariates, subject education level, and mother's education years*

Logistic Regression	b	Odds Ratio (95% CI)	p
DV: Metabolic syndrome (NCEP)			
Step 1:			
Sex	-1.17	0.31 (0.22, 0.45)	0.000
Age	0.04	1.04 (1.01, 1.07)	0.003
Race	0.13	1.14 (0.66, 1.98)	0.642
Step 2:			
Subject Ed. Years	-0.07	0.93 (0.87, 0.99)	0.031
Step 3:			
Mother's Ed. Years	-0.01	0.99 (0.91, 1.08)	0.794
Step 4:			
Mother's Ed. Years by Sex	-0.12	0.89 (0.78, 1.02)	0.086

Table 21: *Sex-Specific predictive analyses of metabolic syndrome by covariates, subject education level, and mother's education years*

Logistic Regression	b	Odds Ratio (95% CI)	p
DV:Metabolic syndrome (NCEP)			
Step 1:			
Age	0.01/0.06	1.00(0.96,1.05)/ 1.06(1.03,1.10)	0.881/0.000
Race	0.74/0.50	2.10(0.96,4.36)/ 0.61(0.27,1.37)	0.049/0.232
Step 2:			
Subject Ed. Years			
Step 3:	-0.06/-0.10	0.95(0.85,1.05)/ 0.91(0.83,0.99)	0.285/0.041
Mother's Ed. Years			
	-0.14/-0.01	0.87(0.78,0.97)/ 0.99(0.91,1.09)	0.011/0.944

Table 22: *Predictive analyses of metabolic syndrome by covariates and mother's education years*

Logistic Regression	b	Odds Ratio (95% CI)	p
DV: Metabolic syndrome (IDF)			
Step 1:			
Sex	-1.04	0.35 (0.25, 0.49)	0.000
Age	0.03	1.03 (1.01, 1.06)	0.007
Race	0.27	1.31 (0.80, 2.17)	0.287
Step 2:			
Mother's Ed. Years			
Step 3:	-0.04	0.96 (0.89, 1.04)	0.325
Mother's Ed. Years by Sex			
	-0.09	0.91 (0.81, 1.03)	0.149

Table 23: *Sex-Specific predictive analyses of metabolic syndrome by covariates and mother's education years*

Logistic Regression	b f/m	Odds Ratio (95% CI) f/m	p f/m
DV:Metabolic syndrome (IDF)			
Step 1:			
Age	0.01/0.05	1.01(0.97,1.05)/1.05(1.02,1.08)	0.596/0.003
Race	0.72/-0.19	2.06(1.05,4.07)/0.82(0.40,1.70)	0.037/0.603
Step 2:			
Mother's Ed. Years			
	-0.14/-0.04	0.87(0.79,0.96)/0.96(0.89,1.05)	0.005/0.368

Table 24: *Predictive analyses of metabolic syndrome by covariates, subject education level, and mother's education years*

Logistic Regression	b	Odds Ratio (95% CI)	p
DV: Metabolic syndrome (IDF)			
Step 1:			
Sex	-1.07	0.34 (0.25, 0.48)	0.000
Age	0.03	1.03 (1.01, 1.06)	0.009
Race	0.08	1.08 (0.64, 1.83)	0.762
Step 2:			
Subject Ed. Years	-0.10	0.91 (0.85, 0.97)	0.003
Step 3:			
Mother's Ed. Years	-0.02	0.98 (0.91, 1.07)	0.675
Step 4:			
Mother's Ed. Years by Sex	-0.09	0.91 (0.81, 1.04)	0.155

Table 25: *Sex-Specific predictive analyses of metabolic syndrome by covariates, subject education level, and mother's education years*

Logistic Regression	b	Odds Ratio (95% CI)	p
DV:Metabolic syndrome (IDF)			
Step 1:			
Age	0.01/0.05	1.01(0.97,1.05)/ 1.05(1.01,1.08)	0.578/0.004
Race	0.61/-0.51	1.84(0.92,3.68)/ 0.60(0.28,1.30)	0.084/0.198
Step 2:			
Subject Ed. Years			
Step 3:	-0.08/-0.12	0.92(0.84,1.01)/ 0.89(0.81,0.97)	0.081/0.010
Mother's Ed. Years			
	-0.12/-0.02	0.89(0.81,0.99)/ 0.99(0.91,1.07)	0.025/0.729

Table 26: *Predictive analyses of metabolic syndrome by covariates and father's education years*

Logistic Regression	b	Odds Ratio (95% CI)	p
DV: Metabolic syndrome (NCEP)			
Step 1:			
Sex	-1.16	0.31 (0.22, 0.45)	0.000
Age	0.04	1.04 (1.02, 1.07)	0.002
Race	0.24	1.27 (0.75, 2.16)	0.379
Step 2:			
Father's Ed. Years			
Step 3:	-0.02	0.98 (0.92, 1.04)	0.509
Father's Ed. Years by Sex			
	-0.09	0.92 (0.83, 1.01)	0.088

Table 27: *Sex-Specific predictive analyses of metabolic syndrome by covariates and father's education years*

Logistic Regression	b f/m	Odds Ratio (95% CI) f/m	p f/m
DV: Metabolic syndrome (NCEP)			
Step 1:			
Age	0.01/0.06	0.76(0.96,1.05)/1.06(1.03,1.10)	0.761/0.000
Race	0.74/-0.28	2.10(1.03,4.28)/ 0.76(0.35,1.65)	0.041/0.482
Step 2:			
Father's Ed. Years			
	-0.11/-0.02	0.89(0.82,0.97)/ 0.98(0.92,1.04)	0.007/0.526

Table 28: *Predictive analyses of metabolic syndrome by covariates, subject education level, and father's education years*

Logistic Regression	b	Odds Ratio (95% CI)	p
DV: Metabolic syndrome (NCEP)			
Step 1:			
Sex	-1.18	0.31 (0.21, 0.44)	0.000
Age	0.04	1.04 (1.02, 1.07)	0.002
Race	0.09	1.10 (0.64, 1.91)	0.730
Step 2:			
Subject Ed. Years	-0.07	0.93 (0.87, 0.99)	0.033
Step 3:			
Father's Ed. Years	-0.01	0.99 (0.93, 1.05)	0.743
Step 4:			
Father's Ed. Years by Sex	-0.09	0.92 (0.83, 1.02)	0.098

Table 29: *Sex-Specific predictive analyses of metabolic syndrome by covariates, subject education level, and father's education years*

Logistic Regression	b	Odds Ratio (95% CI)	p
DV:Metabolic syndrome (NCEP)			
Step 1:			
Age	0.01/0.06	1.01(0.96,1.05)/ 1.03(1.02,1.10)	0.763/0.000
Race	0.67/-0.51	1.95(0.95,4.02)/0.60(0.26,1.36)	0.070/0.219
Step 2:			
Subject Ed. Years			
Step 3:	-0.06/-0.09	0.94(0.85,1.04/ 0.91(0.83,0.99)	0.249/0.048
Father's Ed. Years			
	-0.10/-0.01	0.90(0.83,0.98)/ 0.99(0.93,1.06)	0.019/0.782

Table 30: *Predictive analyses of metabolic syndrome by covariates and father's education years***.**

Logistic Regression	b	Odds Ratio (95% CI)	p
DV: Metabolic syndrome (IDF)			
Step 1:			
Sex	0.04	1.04 (1.01, 1.06)	0.005
Age	-1.05	0.35 (0.25, 0.49)	0.000
Race	0.22	1.25 (0.76, 2.07)	0.384
Step 2:			
Father's Ed. Years			
Step 3:	-0.03	0.98 (0.92, 1.04)	0.409
Father's Ed. Years by Sex			
	-0.07	0.93 (0.85, 1.02)	0.128

Table 31: *Sex-Specific predictive analyses of metabolic syndrome by covariates and father's education years*

Logistic Regression	b f/m	Odds Ratio (95% CI) f/m	p f/m
DV:Metabolic syndrome (IDF)			
Step 1:			
Age	0.01/0.05	1.01(0.98,1.06)/1.05(1.02,1.08)	0.485/0.003
Race	0.65/-0.23	1.90(0.97,3.74)/ 0.79(0.38,1.66)	0.060/0.537
Step 2:			
Father's Ed. Years			
	-0.10/-0.03	0.91(0.84,0.98)/ 0.97(0.92,1.04)	0.009/0.403

Table 32: *Predictive analyses of metabolic syndrome by covariates, subject education level, and father's education years*

Logistic Regression	b	Odds Ratio (95% CI)	p
DV: Metabolic syndrome (IDF)			
Step 1:			
Sex	-1.08	0.34 (0.24, 0.48)	0.000
Age	0.03	1.04 (1.01, 1.06)	0.007
Race	0.04	1.05 (0.62, 1.76)	0.869
Step 2:			
Subject Ed. Years	-0.09	0.91 (0.85, 0.97)	0.003
Step 3:			
Father's Ed. Years	-0.01	0.99 (0.93, 1.05)	0.668
Step 4:			
Father's Ed. Years by Sex	-0.07	0.94 (0.85, 1.03)	0.164

Table 33: *Sex-Specific predictive analyses of metabolic syndrome by covariates, subject education level, and father's education years*

Logistic Regression	b	Odds Ratio (95% CI)	p
DV:Metabolic syndrome (IDF)			
Step 1:			
Age	0.01/0.05	1.01(0.97,1.06)/ 1.05(1.01,1.08)	0.485/0.005
Race	0.54/-0.53	1.72(0.87,3.41)/ 0.59(0.27,1.28)	0.119/0.183
Step 2:			
Subject Ed. Years			
Step 3:	-0.09/-0.12	0.92(0.84,1.01)/ 0.89(0.82,0.97)	0.071/0.011
Father's Ed. Years			
	-0.09/-0.01	0.92(0.85,1.00)/ 0.99(0.93,1.05)	0.038/0.717

Table 34: *Predictive analyses of Systolic Blood Pressure by highest parental education years*

Linear Regression	ΔR^2 (p)	r_p	Unstandardized b	p
DV: SBP				
Step 1:	0.149 (0.00)			
Age		0.159	0.287	0.000
Sex		-0.336	-8.672	0.000
Race		0.136	5.571	0.000
Step 2:	0.007 (0.01)			
Parental Ed. Years		0.012	0.067	0.740
Step 3:	0.007 (0.01)			
Parental Ed. by Sex		-0.09	-0.698	0.008

Table 35: *Sex-specific predictive analyses of Systolic Blood Pressure by highest parental education years*

Linear Regression	ΔR^2 (p) m/f	r_p m/f	Unstandardized b m/f	p m/f
DV: SBP				
Step 1:	0.077(0.00)/0.030(0.00)			
Age		0.179/0.200	0.342 / 0.380	0.000/ 0.000
Race		0.205/0.099	8.068 / 4.398	0.000/ 0.040
Step 2:	0.028(0.00)/0.000(0.82)			
Parental Ed. Years		-0.184/0.001	-0.695 / -0.006	0.000/ 0.977

Table 36. *Predictive analyses of Systolic Blood Pressure by subject education years and highest parental education years*

Linear Regression	ΔR^2 (p)	r_p	Unstandardized b	p
DV: SBP				
Step 1:	0.147 (0.00)			
Age		0.178	0.337	0.000
Sex		0.323	-8.557	0.000
Race		0.138	5.824	0.000
Step 2:	0.003 (0.01)			
Subject Ed. Years		-0.031	-0.147	0.363
Step 3:	0.006 (0.02)			
Parental Ed. Years		0.009	0.052	0.801
Step 4:	0.007 (0.01)			
Parent Ed. by Sex		-0.090	-0.699	0.009

Table 37. *Sex-specific predictive analyses of Systolic Blood Pressure by subject education years and highest parental education years*

Linear Regression	ΔR^2 (p)	r_p	Unstandardized b	P
DV: SBP				
Step 1:	0.077(0.00)/0.030 (0.00)			
Age		0.178 / 0.195	0.340 / 0.370	0.000 / 0.000
Race		0.200 / 0.078	7.905 / 3.589	0.000 / 0.107
Step 2:	0.002(0.31)/0.002 (0.40)			
Subject Ed. Years		-0.032 / -0.064	-0.140 / -0.329	0.498 / 0.187
Step 3:	0.026(0.00)/0.000 (0.67)			
Parental Ed. Years		-0.162 / 0.013	-0.650 / 0.057	0.001 / 0.789

Table 38: *Predictive analyses of Systolic Blood Pressure by mother's education years*

Linear Regression	ΔR^2 (p)	r_p	Unstandardized b	p
DV: SBP				
Step 1:	0.149 (0.00)			
Age		0.187	0.356	0.000
Sex		-0.330	-8.789	0.000
Race		0.165	6.859	0.000
Step 2:	0.008 (0.01)			
Mother Ed. Years		-0.006	-0.044	0.855
Step 3:	0.008 (0.01)			
Mother Ed. by Sex		-0.086	-0.803	0.011

Table 39: *Sex-specific predictive analyses of Systolic Blood Pressure by mother's education years*

Linear Regression	ΔR^2 (p) m/f	r_p m/f	Unstandardized b m/f	p m/f
DV: SBP				
Step 1:	0.079(0.00)/0.030(0.00)			
Age		0.175 / 0.199	0.335 / 0.376	0.000/ 0.000
Race		0.221 / 0.099	8.742 / 4.379	0.000/ 0.041
Step 2:	0.034(0.00)/0.000(0.84)			
Mother Ed. Years		-0.186/0.010	-0.842 / -0.051	0.000/ 0.834

Table 40: *Predictive analyses of Systolic Blood Pressure by subject education years and mother's education years*

Linear Regression	ΔR^2 (p)	r_p	Unstandardized b	p
DV: SBP				
Step 1:	0.147 (0.00)			
Age		0.175	0.331	0.000
Sex		-0.323	-8.563	0.000
Race		0.146	6.153	0.000
Step 2:	0.003 (0.07)			
Subject Ed. Years		-0.032	-0.148	0.357
Step 3:	0.007 (0.01)			
Mother Ed. Years		0.003	0.024	0.923
Step 4:	0.007 (0.01)			
Mother Ed. by Sex		-0.091	-0.839	0.009

Table 41: *Sex-specific predictive analyses of Systolic Blood Pressure by subject education years and mother's education years*

Linear Regression	ΔR^2 (p)	r_p	Unstandardized b	P
DV: SBP				
Step 1:	0.079(0.00)/0.030 (0.00)			
Age		0.173 / 0.193	0.332 /0.366	0.000 / 0.000
Race		0.214 / 0.078	8.518 /3.577	0.000 / 0.109
Step 2:	0.002(0.30)/0.002 (0.41)			
Mother Ed. Years		-0.039 / -0.062	-0.167 / -0.320	0.411 / 0.200
Step 3:	0.032(0.00)/0.000 (0.71)			
Mother Ed. Years		-0.166 / 0.003	-0.786 / 0.016	0.000 / 0.949

Table 42: *Predictive analyses of Systolic Blood Pressure by father's education years*

Linear Regression	ΔR^2 (p)	r_p	Unstandardized b	p
DV: SBP				
Step 1:	0.149 (0.00)			
Age		0.191	0.365	0.000
Sex		-0.331	-8.825	0.000
Race		0.155	6.484	0.000
Step 2:	0.004 (0.05)			
Father Ed. Years		-0.008	-0.040	0.824
Step 3:	0.004 (0.05)			
Father Ed. by Sex		-0.074	-0.519	0.028

Table 43: *Sex-specific predictive analyses of Systolic Blood Pressure by father's education years*

Linear Regression	ΔR^2 (p) m/f	r_p m/f	Unstandardized b m/f	p m/f
DV: SBP				
Step 1:	0.077(0.00)/0.029(0.00)			
Age		0.186 / 0.196	0.356 / 0.373	0.000/ 0.000
Race		0.205 / 0.096	8.129 / 4.282	0.000/ 0.047
Step 2:	0.016(0.01)/0.000(0.97)			
Father Ed. Years		-0.159/0.016	-0.542 / -0.060	0.001/ 0.742

Table 44: *Predictive analyses of Systolic Blood Pressure by subject education years and father's education years*

Linear Regression	ΔR^2 (p)	r_p	Unstandardized b	p
DV: SBP				
Step 1:	0.148 (0.00)			
Age		0.179	0.341	0.000
Sex		-0.325	-8.642	0.000
Race		0.136	5.765	0.000
Step 2:	0.003 (0.07)			
Subject Ed. Years		-0.040	-0.186	0.246
Step 3:	0.004 (0.04)			
Father Ed. Years		0.005	0.028	0.880
Step 4:	0.005 (0.03)			
Father Ed. by Sex		-0.075	-0.521	0.030

Table 45: *Sex-specific predictive analyses of Systolic Blood Pressure by subject education years and father's education years*

Linear Regression	ΔR^2 (p)	r_p	Unstandardized b	P
DV: SBP				
Step 1:	0.077(0.00)/0.029 (0.00)			
Age		0.183 / 0.191	0.352 / 0.363	0.000 / 0.000
Race		0.198 / 0.076	7.888 / 3.531	0.000 / 0.114
Step 2:	0.002(0.31)/0.002 (0.41)			
Father Ed. Years		-0.048 / -0.061	-0.208 / -0.312	0.308 / 0.208
Step 3:	0.014(0.01)/0.000 (0.90)			
Father Ed. Years		-0.138 / -0.004	-0.491 / -0.016	0.004 / 0.933

Table 46: *Predictive analyses of Diastolic Blood Pressure by highest parental education years*

Linear Regression	ΔR^2 (p)	r_p	Unstandardized b	p
DV: DBP				
Step 1:	0.139 (0.00)			
Age		0.148	0.192	0.000
Sex		-0.339	-6.197	0.000
Race		0.121	3.436	0.000
Step 2:	0.007 (0.01)			
Parental Ed. Years		-0.002	-0.007	0.962
Step 3:	0.005 (0.04)			
Parental Ed. by Sex		-0.087	-0.467	0.010

Table 47: *Sex-specific predictive analyses of Diastolic Blood Pressure by highest parental education years*

Linear Regression	ΔR^2 (p) m/f	r_p m/f	Unstandardized b m/f	p m/f
DV: DBP				
Step 1:	0.046(0.00)/0.017(0.04)			
Age		0.179 / 0.149	0.188 / 0.196	0.002/ 0.002
Race		0.205 / 0.081	4.127 / 2.517	0.001/ 0.094
Step 2:	0.026(0.00)/0.000(0.82)			
Parental Ed. Years		-0.184/0.004	-0.467 / -0.013	0.000/ 0.931

Table 48: *Predictive analyses of Diastolic Blood Pressure by subject education years and highest parental education years*

Linear Regression	ΔR^2 (p)	r_p	Unstandardized b	p
DV: DBP				
Step 1:	0.142 (0.00)			
Age		0.140	0.181	0.000
Sex		-0.341	-6.252	0.000
Race		0.103	2.947	0.003
Step 2:	0.005 (0.02)			
Subject Ed. Years		-0.050	-0.160	0.150
Step 3:	0.005 (0.02)			
Parental Ed. Years		0.003	0.012	0.933
Step 4:	0.005 (0.02)			
Parent Ed. by Sex		-0.078	-0.418	0.024

Table 49: *Sex-specific predictive analyses of Diastolic Blood Pressure by subject education years and highest parental education years*

Linear Regression	ΔR^2 (p)	r_p	Unstandardized b	P
DV: DBP				
Step 1:	0.046(0.00) / 0.017 (0.04)			
Age		0.147 / 0.141	0.187 / 0.186	0.002 / 0.003
Race		0.154 / 0.054	4.032 / 1.722	0.001 / 0.269
Step 2:	0.002(0.39)/0.006 (0.13)			
Subject Ed. Years		-0.029 / -0.090	-0.083 / -0.324	0.544 / 0.064
Step 3:	0.024(0.00)/0.000 (0.92)			
Parental Ed. Years		-0.164 / 0.016	-0.440 / 0.049	0.000 / 0.741

Table 50: *Predictive analyses of Diastolic Blood Pressure by mother's education years*

Linear Regression	ΔR^2 (p)	r_p	Unstandardized b	p
DV: DBP				
Step 1:	0.140 (0.00)			
Age		0.150	0.194	0.000
Sex		-0.341	-6.244	0.000
Race		0.133	3.679	0.000
Step 2:	0.006 (0.02)			
Mother Ed. Years		-0.004	-0.019	0.907
Step 3:	0.004 (0.05)			
Mother Ed. by Sex		-0.073	-0.463	0.032

Table 51: *Sex-specific predictive analyses of Diastolic Blood Pressure by mother's education years*

Linear Regression	ΔR^2 (p) m/f	r_p m/f	Unstandardized b m/f	p m/f
DV: DBP				
Step 1:	0.049(0.00)/0.017(0.04)			
Age		0.150 / 0.149	0.192 / 0.196	0.000/ 0.002
Race		0.181 / 0.080	4.737 / 2.494	0.001/ 0.097
Step 2:	0.024(0.00)/0.000(0.87)			
Mother Ed. Years		-0.158/0.008	-0.475 / -0.027	0.001/ 0.872

Table 52: *Predictive analyses of Diastolic Blood Pressure by subject education years and mother's education years*

Linear Regression	ΔR^2 (p)	r_p	Unstandardized b	p
DV: DBP				
Step 1:	0.143 (0.00)			
Age		0.141	0.182	0.000
Sex		-0.342	-6.272	0.000
Race		0.112	3.221	0.001
Step 2:	0.005 (0.02)			
Subject Ed. Years		-0.055	-0.175	0.112
Step 3:	0.004 (0.05)			
Mother Ed. Years		0.005	0.023	0.892
Step 4:	0.005 (0.04)			
Mother Ed. by Sex		-0.071	-0.448	0.041

Table 53: *Sex-specific predictive analyses of Diastolic Blood Pressure by subject education years and mother's education years*

Linear Regression	ΔR^2 (p)	r_p	Unstandardized b	P
DV: DBP				
Step 1:	0.049(0.00)/0.017(0.04)			
Age		0.173 / 0.142	0.190 / 0.186	0.002 / 0.003
Race		0.214 / 0.053	4.561 / 1.706	0.000 / 0.274
Step 2:	0.002(0.38)/0.006 (0.14)			
Mother Ed. Years		-0.039 / -0.087	-0.131 / -0.314	0.334 / 0.072
Step 3:	0.020(0.00)/0.000 (0.89)			
Mother Ed. Years		-0.166 / 0.011	-0.431 / 0.038	0.004 / 0.826

Table 54: *Predictive analyses of Diastolic Blood Pressure by father's education years*

Linear Regression	ΔR^2 (p)	r_p	Unstandardized b	p
DV: DBP				
Step 1:	0.139 (0.00)			
Age		0.152	0.197	0.000
Sex		-0.341	-6.244	0.000
Race		0.121	3.428	0.000
Step 2:	0.005 (0.03)			
Father Ed. Years		-0.003	-0.012	0.923
Step 3:	0.003 (0.11)			
Father Ed. by Sex		-0.077	-0.367	0.023

Table 55: *Sex-specific predictive analyses of Diastolic Blood Pressure by father's education years*

Linear Regression	ΔR^2 (p) m/f	r_p m/f	Unstandardized b m/f	p m/f
DV: DBP				
Step 1:	0.046(0.00)/0.017 (0.04)			
Age		0.157 / 0.146	0.199 / 0.193	0.001/ 0.002
Race		0.159 / 0.079	4.144 / 2.468	0.001/ 0.102
Step 2:	0.016(0.01)/0.000(0.71)			
Father Ed. Years		-0.163/0.009	-0.369 / -0.024	0.001/ 0.853

Table 56: *Predictive analyses of Diastolic Blood Pressure by subject education years and father's education years*

Linear Regression	ΔR^2 (p)	r_p	Unstandardized b	p
DV: DBP				
Step 1:	0.143 (0.00)			
Age		0.141	0.182	0.000
Sex		-0.342	-6.272	0.000
Race		0.112	3.221	0.004
Step 2:	0.005 (0.02)			
Subject Ed. Years		-0.055	-0.175	0.089
Step 3:	0.003 (0.07)			
Father Ed. Years		0.005	0.023	0.902
Step 4:	0.005 (0.06)			
Father Ed. by Sex		-0.071	-0.448	0.059

Table 57: *Sex-specific predictive analyses of Diastolic Blood Pressure by subject education years and father's education years*

Linear Regression	ΔR^2 (p)	r_p	Unstandardized b	P
DV: DBP				
Step 1:	0.046(0.00)/ 0.017 (0.04)			
Age		0.155 / 0.139	0.196 / 0.183	0.001 / 0.004
Race		0.152 / 0.053	4.001 / 1.703	0.001 / 0.276
Step 2:	0.002(0.41)/0.006 (0.13)			
Father Ed. Years		-0.043 / -0.089	-0.123 / -0.318	0.364 / 0.067
Step 3:	0.014(0.01)/0.000 (0.92)			
Father Ed. Years		-0.143 / 0.008	-0.339 / 0.022	0.003 / 0.868

Table 58: *Predictive analyses of Glucose by highest parental education years*

Linear Regression	ΔR^2 (p)	r_p	Unstandardized b	p
DV: Glucose				
Step 1:	0.077 (0.00)			
Age		0.176	0.004	0.000
Sex		-0.225	-0.063	0.000
Race		0.020	0.009	0.564
Step 2:	0.000 (0.82)			
Parental Ed. Years		-0.060	-0.004	0.077
Step 3:	0.005 (0.03)			
Parental Ed. by Sex		0.073	0.006	0.030

Table 59: *Sex-specific predictive analyses of Glucose by highest parental education years*

Linear Regression	ΔR^2 (p) m/f	r_p m/f	Unstandardized b m/f	p m/f
DV: Glucose				
Step 1:	0.034(0.00)/0.035 (0.00)			
Age		0.191 / 0.164	0.004 / 0.004	0.000/ 0.001
Race		-0.006/ 0.046	-0.002 / 0.023	0.894/ 0.344
Step 2:	0.003(0.21)/0.006(0.11)			
Parental Ed. Years		0.060/-0.077	0.002 / -0.004	0.207/ 0.113

Table 60: *Predictive analyses of Glucose by subject education years and highest parental education years*

Linear Regression	ΔR^2 (p)	r_p	Unstandardized b	p
DV: Glucose				
Step 1:	0.081 (0.00)			
Age		0.161	0.003	0.000
Sex		-0.245	-0.066	0.000
Race		0.000	0.000	0.990
Step 2:	0.012 (0.00)			
Subject Ed. Years		-0.117	-0.006	0.001
Step 3:	0.000 (0.94)			
Parental Ed. Years		-0.057	-0.004	0.099
Step 4:	0.006 (0.02)			
Parent Ed. by Sex		0.080	0.006	0.020

Table 61: *Sex-specific predictive analyses of Glucose by subject education years and highest parental education years*

Linear Regression	ΔR^2 (p)	r_p	Unstandardized b	P
DV: Glucose				
Step 1:	0.034(0.00)/0.035(0.00)			
Age		0.174 / 0.155	0.004 / 0.003	0.000 / 0.001
Race		-0.065 / 0.013	-0.007 / 0.007	0.689 / 0.790
Step 2:	0.005(0.12)/0.015(0.01)			
Subject Ed. Years		0.093 / -0.112	-0.004 / -0.007	0.034 / 0.021
Step 3:	0.008(0.06)/0.002 (0.30)			
Parental Ed. Years		0.057 / -0.051	0.004 / -0.003	0.056 / 0.297

Table 62: *Predictive analyses of Glucose by mother's education years*

Linear Regression	ΔR^2 (p)	r_p	Unstandardized b	p
DV: Glucose				
Step 1:	0.076 (0.00)			
Age		0.171	0.004	0.000
Sex		-0.224	-0.062	0.000
Race		0.018	0.008	0.601
Step 2:	0.001 (0.31)			
Mother Ed. Years		-0.068	-0.005	0.044
Step 3:	0.003 (0.07)			
Mother Ed. by Sex		-0.014	0.006	0.072

Table 63: *Predictive analyses of Glucose by father's education years*

Linear Regression	ΔR^2 (p)	r_p	Unstandardized b	p
DV: Glucose				
Step 1:	0.077 (0.00)			
Age		0.173	0.003	0.000
Sex		-0.226	-0.063	0.000
Race		0.017	0.008	0.618
Step 2:	0.000 (0.66)			
Father Ed. Years		-0.069	-0.004	0.042
Step 3:	0.006 (0.02)			
Father Ed. by Sex		0.080	0.006	0.018

Table 64: *Sex-specific predictive analyses of Glucose by father's education years*

Linear Regression	ΔR^2 (p) m/f	r_p m/f	Unstandardized b m/f	p m/f
DV: Glucose				
Step 1:	0.034(0.00)/0.035(0.00)			
Age		0.190 / 0.158	0.004 / 0.003	0.000/ 0.001
Race		-0.006/ 0.040	-0.002 / 0.020	0.907/ 0.411
Step 2:	0.003(0.21)/0.007(0.07)			
Father Ed. Years		0.059/ -0.088	0.002 / -0.004	0.213/ 0.070

Table 65: *Predictive analyses of Glucose by subject education years and father's education years*

Linear Regression	ΔR^2 (p)	r_p	Unstandardized b	p
DV: Glucose				
Step 1:	0.081 (0.00)			
Age		0.161	0.003	0.000
Sex		-0.245	-0.066	0.000
Race		0.000	-0.001	0.949
Step 2:	0.012 (0.00)			
Subject Ed. Years		-0.117	-0.005	0.001
Step 3:	0.000 (0.71)			
Father Ed. Years		-0.057	-0.004	0.037
Step 4:	0.007 (0.01)			
Father Ed. by Sex		0.080	0.006	0.012

Table 66: *Sex-specific predictive analyses of Glucose by subject education years and father's education years*

Linear Regression	ΔR^2 (p)	r_p	Unstandardized b	P
DV: Glucose				
Step 1:	0.034(0.00)/0.035 (0.00)			
Age		0.183 / 0.150	0.004 / 0.003	0.000 / 0.002
Race		-0.018 / 0.008	-0.007 / 0.004	0.711 / 0.869
Step 2:	0.005(0.12)/0.015 (0.01)			
Father Ed. Years		-0.096 / -0.112	-0.004 / -0.007	0.044 / 0.020
Step 3:	0.007(0.07)/0.004 (0.17)			
Father Ed. Years		0.085 / -0.066	0.003 / -0.003	0.074 / 0.072

Table 67: *Predictive analyses of HDL Cholesterol by highest parental education years*

Linear Regression	ΔR^2 (p)	r_p	Unstandardized b	p
DV: HDL				
Step 1:	0.219 (0.00)			
Age		0.118	0.226	0.000
Sex		0.453	12.938	0.000
Race		-0.023	-0.961	0.497
Step 2:	0.000 (0.82)			
Parental Ed. Years		-0.059	-0.356	0.082
Step 3:	0.004 (0.03)			
Parental Ed. by Sex		0.072	0.572	0.033

Table 68: *Sex-specific predictive analyses of HDL Cholesterol by highest parental education years*

Linear Regression	ΔR^2 (p) m/f	r_p m/f	Unstandardized b m/f	p m/f
DV: HDL				
Step 1:	0.034(0.01)/0.010 (0.11)			
Age		0.170 / 0.057	0.361 / 0.095	0.000/ 0.237
Race		-0.077/ 0.055	-3.336 / 2.190	0.103/ 0.254
Step 2:	0.003(0.22)/0.010(0.04)			
Parental Ed. Years		0.058/-0.102	0.239 / -0. 390	0.224/ 0.035

Table 69: *Predictive analyses of HDL Cholesterol by subject education years and highest parental education years*

Linear Regression	ΔR^2 (p)	r_p	Unstandardized b	p
DV: HDL				
Step 1:	0.221 (0.00)			
Age		0.120	0.227	0.001
Sex		0.457	13.068	0.000
Race		-0.008	-0.318	0.827
Step 2:	0.004 (0.05)			
Subject Ed. Years		0.068	0.322	0.049
Step 3:	0.000 (0.49)			
Parental Ed. Years		-0.065	-0.393	0.062
Step 4:	0.003 (0.06)			
Parent Ed. by Sex		0.066	0.520	0.056

Table 70: *Sex-specific predictive analyses of HDL Cholesterol by subject education years and highest parental education years*

Linear Regression	ΔR^2 (p)	r_p	Unstandardized b	P
DV: HDL				
Step 1:	0.034(0.01)/0.010(0.11)			
Age		0.174 / 0.063	0.368 / 0.099	0.000 / 0.220
Race		-0.065 / 0.063	-2.826 / 2.528	0.169 / 0.207
Step 2:	0.011(0.02)/0.000(0.90)			
Subject Ed. Years		0.093 / 0.031	0.450 / 0.136	0.049 / 0.544
Step 3:	0.000(0.64)/0.011 (0.03)			
Parental Ed. Years		0.057 / -0.092	0.099 / -0.416	0.635 / 0.029

Table 71: *Predictive analyses of HDL Cholesterol by mother's education years*

Linear Regression	ΔR^2 (p)	r_p	Unstandardized b	p
DV: HDL				
Step 1:	0.218 (0.00)			
Age		0.119	0.227	0.000
Sex		0.453	12.866	0.000
Race		-0.033	-1.375	0.331
Step 2:	0.000 (0.68)			
Mother Ed. Years		-0.043	-0.307	0.203
Step 3:	0.002 (0.18)			
Mother Ed. by Sex		0.045	0.426	0.181

Table 72: *Predictive analyses of HDL Cholesterol by father's education years*

Linear Regression	ΔR^2 (p)	r_p	Unstandardized b	p
DV: HDL				
Step 1:	0.220 (0.00)			
Age		0.119	0.228	0.000
Sex		0.454	12.966	0.000
Race		-0.024	-1.014	0.475
Step 2:	0.000 (0.94)			
Father Ed. Years		-0.049	-0.261	0.150
Step 3:	0.003 (0.06)			
Father Ed. by Sex		0.026	0.450	0.060

Table 73: *Predictive analyses of Waist Circumference by highest parental education years*

Linear Regression	ΔR^2 (p)	r_p	Unstandardized b	p
DV: WC				
Step 1:	0.216 (0.00)			
Age		0.091	0.188	0.007
Sex		-0.452	-13.920	0.000
Race		0.101	4.561	0.003
Step 2:	0.002 (0.13)			
Parental Ed. Years		0.013	0.083	0.708
Step 3:	0.003 (0.05)			
Parental Ed. by Sex		-0.063	-0.537	0.051

Table 74: *Sex-specific predictive analyses of Waist Circumference by highest parental education years*

Linear Regression	ΔR^2 (p) m/f	r_p m/f	Unstandardized b m/f	p m/f
DV: WC				
Step 1:	0.027(0.00)/0.025(0.01)			
Age		0.035 / 0.153	0.077 / 0.296	0.456/ 0.001
Race		0.139 / 0.051	6.242 / 2.308	0.003/ 0.295
Step 2:	0.012(0.02)/0.001(0.59)			
Parental Ed. Years		-0.111/0.026	-0.477 / 0.114	0.019/ 0.026

Table 75: *Predictive analyses of Waist Circumference by subject education years and highest parental education years*

Linear Regression	ΔR^2 (p)	r_p	Unstandardized b	p
DV: WC				
Step 1:	0.217 (0.00)			
Age		0.081	0.164	0.020
Sex		-0.461	-14.186	0.000
Race		0.078	3.522	0.025
Step 2:	0.013 (0.00)			
Subject Ed. Years		-0.114	-0.584	0.001
Step 3:	0.000 (0.60)			
Parental Ed. Years		0.023	0.149	0.510
Step 4:	0.002 (0.16)			
Parent Ed. by Sex		-0.048	-0.410	0.161

Table 76: *Sex-specific predictive analyses of Waist Circumference by subject education years and highest parental education years*

Linear Regression	ΔR^2 (p)	r_p	Unstandardized b	P
DV: WC				
Step 1:	0.027(0.00)/0.025 (0.01)			
Age		0.031 / 0.144	0.066 / 0.277	0.519 / 0.003
Race		0.123 / 0.016	5.494 / 0.740	0.009 / 0.745
Step 2:	0.025(0.00)/0.012 (0.02)			
Subject Ed. Years		-0.129 / -0.120	-0.649 / -0.638	0.006 / 0.013
Step 3:	0.003(0.21)/0.003 (0.28)			
Parental Ed. Years		-0.060 / 0.053	-0.271 / 0.235	0.208 / 0.277

Table 77: *Predictive analyses of Waist Circumference by mother's education years*

Linear Regression	ΔR^2 (p)	r_p	Unstandardized b	p
DV: WC				
Step 1:	0.213 (0.00)			
Age		0.088	0.181	0.009
Sex		-0.452	-13.933	0.000
Race		0.106	4.795	0.002
Step 2:	0.003 (0.07)			
Mother Ed. Years		-0.004	-0.034	0.896
Step 3:	0.002 (0.15)			
Mother Ed. by Sex		-0.049	-0.497	0.149

Table 78: *Sex-specific predictive analyses of Waist Circumference by mother's education years*

Linear Regression	ΔR^2 (p) m/f	r_p m/f	Unstandardized b m/f	p m/f
DV: WC				
Step 1:	0.028(0.00)/0.025(0.01)	0.033 / 0.147	0.073 / 0.284	0.481/ 0.002
Age		0.150 / 0.049	6.744 / 2.227	0.001/ 0.312
Race				
Step 2:	0.012(0.02)/0.000(0.99)	-0.110/0.000	-0.570 / 0.002	0.020/ 0.993
Mother Ed. Years				

Table 79: *Predictive analyses of Waist Circumference by subject education years and mother's education years*

Linear Regression	ΔR^2 (p)	r_p	Unstandardized b	p
DV: WC				
Step 1:	0.214 (0.00)	0.081	0.164	0.025
Age		-0.461	-14.186	0.000
Sex		0.078	3.522	0.019
Race				
Step 2:	0.013 (0.00)	-0.114	-0.584	0.001
Subject Ed. Years				
Step 3:	0.001 (0.42)	0.004	0.031	0.906
Mother Ed. Years				
Step 4:	0.001 (0.36)	-0.032	-0.322	0.355
Mother Ed. by Sex				

Table 80: *Sex-specific predictive analyses of Waist Circumference by subject education years and mother's education years*

Linear Regression	ΔR^2 (p)	r_p	Unstandardized b	P
DV: WC				
Step 1:	0.028(0.00)/0.025(0.01)			
Age		0.028 / 0.138	0.061 / 0.265	0.551 / 0.004
Race		0.130 / 0.015	5.859 / 0.692	0.006 / 0.761
Step 2:	0.025(0.00)/0.012 (0.02)			
Mother Ed. Years		-0.133 / -0.116	-0.656 / -0.611	0.005 / 0.017
Step 3:	0.004(0.17)/0.001 (0.61)			
Mother Ed. Years		-0.065 / 0.025	-0.346 / 0.130	0.174 / 0.609

Table 81: *Predictive analyses of Waist Circumference by father's education years*

Linear Regression	ΔR^2 (p)	r_p	Unstandardized b	p
DV: WC				
Step 1:	0.213 (0.00)			
Age		0.089	0.183	0.008
Sex		-0.453	-13.944	0.000
Race		0.097	4.387	0.004
Step 2:	0.004 (0.04)			
Father Ed. Years		0.009	0.053	0.787
Step 3:	0.005 (0.02)			
Father Ed. by Sex		-0.077	-0.585	0.023

Table 82: *Sex-specific predictive analyses of Waist Circumference by father's education years*

Linear Regression	ΔR^2 (p) m/f	r_p m/f	Unstandardized b m/f	p m/f
DV: WC				
Step 1:	0.027(0.00)/0.025(0.01)			
Age		0.033 / 0.151	0.071 / 0.293	0.485/ 0.002
Race		0.134 / 0.050	5.963 / 2.294	0.005/ 0.301
Step 2:	0.020(0.00)/0.000(0.67)			
Father Ed. Years		-0.142/0.020	-0.550 / 0.079	0.003/ 0.674

Table 83: *Predictive analyses of Waist Circumference by subject education years and father's education years*

Linear Regression	ΔR^2 (p)	r_p	Unstandardized b	p
DV: WC				
Step 1:	0.214 (0.00)			
Age		0.079	0.160	0.023
Sex		-0.462	-14.186	0.000
Race		0.075	3.416	0.030
Step 2:	0.013 (0.00)			
Subject Ed. Years		-0.111	-0.560	0.001
Step 3:	0.001 (0.28)			
Father Ed. Years		0.019	0.109	0.581
Step 4:	0.001 (0.07)			
Father Ed. by Sex		-0.062	-0.470	0.071

Table 84: *Sex-specific predictive analyses of Waist Circumference by subject education years and father's education years*

Linear Regression	ΔR^2 (p)	r_p	Unstandardized b	P
DV: WC				
Step 1:	0.027(0.00) / 0.025 (0.01)			
Age		0.027 / 0.142	0.057 / 0.274	0.574 / 0.003
Race		0.118 / 0.017	5.259 / 0.799	0.013 / 0.727
Step 2:	0.024(0.00)/0.012 (0.02)			
Father Ed. Years		-0.122 / -0.118	-0.599 / -0.621	0.010 / 0.015
Step 3:	0.009(0.04)/0.002 (0.38)			
Father Ed. Years		-0.100 / 0.043	-0.402 / 0.168	0.035 / 0.379

Table 85: *Predictive analyses of Triglycerides by highest parental education years*

Linear Regression	ΔR^2 (p)	r_p	Unstandardized b	p
DV: TG				
Step 1:	0.138 (0.00)			
Age		0.077	0.006	0.023
Sex		-0.145	-0.354	0.000
Race		0.121	-0.236	0.000
Step 2:	0.016 (0.00)			
Parental Ed. Years		-0.027	-0.006	0.430
Step 3:	0.007 (0.01)			
Parental Ed. by Sex		-0.088	-0.027	0.010

Table 86: *Sex-specific predictive analyses of Triglycerides by highest parental education years*

Linear Regression	ΔR^2 (p) m/f	r_p m/f	Unstandardized b m/f	p m/f
DV: TG				
Step 1:	0.035(0.00)/0.024 (0.01)			
Age		0.097 / 0.063	0.006 / 0.005	0.041/ 0.195
Race		-0.156/0.141	-0.207 / -0.274	0.001/ 0.003
Step 2:	0.059(0.00)/0.001(0.45)			
Parental Ed. Years		-0.248/0.037	-0.033 / -0.007	0.000/ 0.449

Table 87: *Predictive analyses of Triglycerides by subject education years and highest parental education years*

Linear Regression	ΔR^2 (p)	r_p	Unstandardized b	p
DV: TG				
Step 1:	0.135 (0.00)			
Age		0.070	0.005	0.043
Sex		-0.334	-0.347	0.000
Race		-0.158	0.260	0.000
Step 2:	0.016 (0.00)			
Subject Ed. Years		-0.194	-0.017	0.006
Step 3:	0.010 (0.00)			
Parental Ed. Years		-0.009	-0.002	0.801
Step 4:	0.007 (0.01)			
Parent Ed. by Sex		-0.090	-0.027	0.009

Table 88: *Sex-specific predictive analyses of Triglycerides by subject education years and highest parental education years*

Linear Regression	ΔR^2 (p)	r_p	Unstandardized b	P
DV: TG				
Step 1:	0.035(0.00) / 0.024 (0.01)			
Age		0.094 / 0.141	0.006 / 0.004	0.048 / 0.255
Race		-0.166 / 0.054	-0.222 / -0.327	0.000 / 0.001
Step 2:	0.026(0.00)/0.010 (0.04)			
Subject Ed. Years		-0.089 / -0.090	-0.013 / -0.021	0.011 / 0.040
Step 3:	0.040(0.00)/0.000 (0.76)			
Parental Ed. Years		-0.207 / 0.016	-0.028 / -0.003	0.000 / 0.759

Table 89: *Predictive analyses of Triglycerides by mother's education years*

Linear Regression	ΔR^2 (p)	r_p	Unstandardized b	p
DV: TG				
Step 1:	0.137 (0.00)			
Age		0.078	0.006	0.021
Sex		-0.341	-0.357	0.000
Race		-0.136	-0.222	0.000
Step 2:	0.013 (0.00)			
Mother Ed. Years		-0.005	-0.001	0.885
Step 3:	0.000 (0.00)			
Mother Ed. by Sex		-0.104	-0.038	0.002

Table 90: *Sex-specific predictive analyses of Triglycerides by mother's education years*

Linear Regression	ΔR^2 (p) m/f	r_p m/f	Unstandardized b m/f	p m/f
DV: TG				
Step 1:	0.035(0.00)/0.025(0.01)			
Age		0.090 / 0.069	0.006 / 0.006	0.057/ 0.153
Race		-0.139/0.139	-0.185 / -0.269	0.003/ 0.004
Step 2:	0.059(0.00)/0.000(0.87)			
Mother Ed. Years		-0.248/0.008	-0.039 / -0.002	0.000/ 0.868

Table 91: *Predictive analyses of Triglycerides by subject education years and mother's education years*

Linear Regression	ΔR^2 (p)	r_p	Unstandardized b	p
DV: TG				
Step 1:	0.135 (0.00)			
Age		0.070	0.005	0.036
Sex		-0.336	-0.348	0.000
Race		-0.151	-0.249	0.000
Step 2:	0.016 (0.00)			
Subject Ed. Years		-0.102	-0.017	0.003
Step 3:	0.006 (0.01)			
Mother Ed. Years		-0.019	-0.005	0.580
Step 4:	0.010 (0.00)			
Mother Ed. by Sex		-0.109	-0.039	0.002

Table 92: *Sex-specific predictive analyses of Triglycerides by subject education years and mother's education years*

Linear Regression	ΔR^2 (p)	r_p	Unstandardized b	P
DV: TG				
Step 1:	0.035(0.00) / 0.025 (0.01)			
Age		0.173 / 0.061	0.006 / 0.005	0.066 / 0.211
Race		0.214 / -0.162	-0.204 / -0.326	0.001 / 0.001
Step 2:	0.026(0.00)/0.010 (0.04)			
Mother Ed. Years		-0.039 / -0.100	-0.014 / -0.023	0.042 / 0.039
Step 3:	0.041(0.00)/0.000 (0.78)			
Mother Ed. Years		-0.166 / 0.013	-0.034 / 0.003	0.000 / 0.787

Table 93: *Predictive analyses of Triglycerides by father's education years*

Linear Regression	ΔR^2 (p)	r_p	Unstandardized b	p
DV: TG				
Step 1:	0.138 (0.00)			
Age		0.076	0.006	0.026
Sex		-0.343	-0.359	0.000
Race		-0.147	-0.240	0.000
Step 2:	0.016 (0.00)			
Father Ed. Years		-0.055	-0.011	0.107
Step 3:	0.002 (0.12)			
Father Ed. by Sex		-0.053	-0.014	0.119

Table 94: *Sex-specific predictive analyses of Triglycerides by father's education years*

Linear Regression	ΔR^2 (p) m/f	r_p m/f	Unstandardized b m/f	p m/f
DV: TG				
Step 1:	0.035(0.00)/0.024(0.01)			
Age		0.105/0.053	0.007 / 0.004	0.026/ 0.277
Race		-0.151/0.148	-0.203 / -0.288	0.001/ 0.002
Step 2:	0.043(0.00)/0.006(0.12)			
Father Ed. Years		-0.210/0.075	-0.025 / -0.012	0.000/ 0.121

Table 95: *Predictive analyses of Triglycerides by subject education years and father's education years*

Linear Regression	ΔR^2 (p)	r_p	Unstandardized b	p
DV: TG				
Step 1:	0.136 (0.00)			
Age		0.068	0.005	0.051
Sex		-0.338	-0.352	0.000
Race		-0.161	-0.265	0.000
Step 2:	0.016 (0.00)			
Subject Ed. Years		-0.102	-0.019	0.003
Step 3:	0.011 (0.00)			
Father Ed. Years		-0.039	-0.008	0.266
Step 4:	0.002 (0.12)			
Father Ed. by Sex		-0.054	-0.015	0.120

Table 96: *Sex-specific predictive analyses of Triglycerides by subject education years and father's education years*

Linear Regression	ΔR^2 (p)	r_p	Unstandardized b	P
DV: TG				
Step 1:	0.035(0.00)/0.024 (0.01)			
Age		0.100 / 0.045	0.006 / 0.004	0.035 / 0.350
Race		-0.165 / -0.166	-0.222 / -0.336	0.001 / 0.001
Step 2:	0.027(0.00)/0.010 (0.04)			
Father Ed. Years		-0.113 / -0.089	-0.017 / -0.020	0.017 / 0.068
Step 3:	0.027(0.00)/0.003 (0.23)			
Father Ed. Years		-0.171 / -0.058	-0.021 / -0.010	0.000 / 0.233

Table 97: *Comparison of SES indicators between subjects with and without the metabolic syndrome (NCEP)*

	Metabolic syndrome absent (n=657) Mean (SD)	Metabolic Syndrome present (n=186) Mean (SD)
Family Income (1-8)	5.55 (2.03)	5.43 (2.12)
Subject Education (years)	16.19 (2.90)	15.52 (2.78)*
Subject Education (level)	5.76 (1.04)	5.51 (1.01)*
Father's Education (years)	13.13 (3.65)	12.36 (3.63)*
Father's Education (level)	4.39 (1.60)	4.06 (1.59)*
Mother's Education (years)	12.75(2.74)	12.16 (2.74)*
Mother's Education (level)	4.24 (1.31)	3.98 (1.33)*
Highest Parental Education (years)	13.90 (3.27)	13.35 (3.21)
Highest Parental Education (level)	4.75 (1.39)	4.50 (1.37)

P<.01**, P<.05*

Table 98: *Comparison of SES indicators between men and women with the metabolic syndrome (NCEP)*

	Metabolic syndrome males (n=127) Mean (SD)	Metabolic Syndrome females (n=59) Mean (SD)
Family Income (1-8)	5.35 (2.16)	5.31 (2.07)
Subject Education (years)	15.90 (2.47)	14.74 (3.22)*
Subject Education (level)	5.64 (0.95)	5.26 (1.10)*
Father's Education (years)	12.85 (3.80)	11.37 (3.06)*
Father's Education (level)	4.23 (1.64)	3.70 (1.44)*
Mother's Education (years)	12.53(2.63)	11.41 (2.86)*
Mother's Education (level)	4.14 (1.31)	3.65 (1.34)*
Highest Parental Education (years)	13.86 (3.32)	12.30 (2.73)**
Highest Parental Education (level)	4.69 (1.42)	4.13 (1.17)*

P<.01**, P<.05*

Table 99: *Comparison of SES indicators between women with and without the metabolic syndrome (NCEP)*

	Metabolic syndrome absent (n=374) Mean (SD)	Metabolic Syndrome present (n=59) Mean (SD)
Family Income (1-8)	5.61 (2.06)	5.31 (2.07)
Subject Education (years)	16.11 (3.06)	14.74 (3.22)**
Subject Education (level)	5.71 (1.09)	5.26 (1.10)**
Father's Education (years)	13.11 (3.83)	11.34 (3.06)**
Father's Education (level)	4.40 (1.62)	3.70 (1.44)**
Mother's Education (years)	12.75(2.85)	11.41 (2.86)**
Mother's Education (level)	2.86 (2.07)	2.02 (1.64)**
Highest Parental Education (years)	13.97 (3.47)	12.30 (2.73)**
Highest Parental Education (level)	4.77 (1.42)	4.13 (1.17)**

P<.01**, P<.05*

Table 100: *Comparison of SES indicators between men with and without the metabolic syndrome (NCEP)*

	Metabolic syndrome absent (n=283) Mean (SD)	Metabolic Syndrome present (n=127) Mean (SD)
Family Income (1-8)	5.49 (2.00)	5.35 (2.16)
Subject Education (years)	16.28 (2.69)	15.90 (2.47)
Subject Education (level)	5.82 (0.97)	5.63 (0.95)
Father's Education (years)	13.15 (3.41)	12.85 (3.80)
Father's Education (level)	4.40 (1.58)	4.23 (1.64)
Mother's Education (years)	12.75(2.60)	12.53 (2.63)
Mother's Education (level)	2.87 (2.03)	4.14 (1.31)
Highest Parental Education (years)	13.82 (3.00)	13.86 (3.32)
Highest Parental Education (level)	4.73 (1.37)	4.69 (1.42)

P<.01**, P<.05*

Table 101: *Percentage of participants with metabolic syndrome and risk factors stratified by subjects' educational levels.*

	< H.S. Grad	H.S. Grad	1- 3 Years College	College Degree	Graduate Degree
	m/f	m/f	m/f	m/f	m/f
Metabolic Syndrome (NCEP)	1.1%/-	12.8%/32.6%	24.5%/26.1%	44.7%/23.9%	17.0%/17.4%
SBP ≥ 130 mmHg	1.1%/-	16.3%/30.2%	26.9%/27.9%	36.5%/18.6%	19.2%/23.3%
DBP ≥ 85 mmHg	2.5%/-	10.8%/26.6%	23.6%/32.8%	45.2%/21.9%	17.8%/18.8%
Gluc. ≥110 mg/dL	4.4%/-	17.8%/23.1%	22.2%/23.1%	37.8%/38.5%	17.8%/15.4%
HDL <40 (m), <50 (f) mg/dL	-/-	11.6%/26.1%	29.5%/26.9%	37.2%/25.2%	21.7%/21.8%
WC>102(m) >88(f) cm	2.7%/-	14.4%/26.7%	24.0%/25.3%	39.7%/27.3%	19.2%/20.7%
Triglyceride ≥ 150 mg/dL	1.3%/-	15.1%/26.3%	18.9%/22.8%	40.9%/38.6%	23.3%/12.3%

Table 102: *Percentage of participants with metabolic syndrome and risk factors stratified by parents' educational levels.*

	< H.S. Grad	H.S. Grad	1- 3 Years College	College Degree	Graduate Degree
	m/f	m/f	m/f	m/f	m/f
Metabolic Syndrome (NCEP)	9.7%/10.8%	47.9%/67.4%	7.4%/8.7%	24.5%/10.9%	10.6%/2.2%
SBP ≥ 130 mmHg	11.5%/16.3%	45.2%/69.8%	13.5%/9.3%	20.2%/2.3%	9.6%/2.3%
DBP ≥ 85 mmHg	10.2%/12.5%	49.7%/67.2%	11.5%/4.7%	20.4%/12.5%	8.3%/3.1%
Gluc. ≥110 mg/dL	6.7%/3.8%	51.1%/46.2%	13.3%/11.5%	22.2%/26.9%	6.7%/11.5%
HDL <40 (m), <50 (f) mg/dL	7.0%/8.4%	49.6%/56.3%	9.3%/14.3%	24.0%/9.2%	10.1%/11.8%
WC>102(m) >88(f) cm	9.6%/9.3%	47.9%/58.7%	9.6%/10.0%	20.5%/12.7%	12.3%/9.3%
Triglyceride ≥ 150 mg/dL	8.2%/15.8%	52.2%/61.4%	8.8%/8.8%	24.5%/8.8%	6.3%/5.3%

Table 103: *Comparison of SES indicators between Caucasians and African Americans.*

	Caucasian (n=752) Mean (SD)	African American (n=91) Mean (SD)
Family Income (1-8)	5.67 (1.98)	4.17 (2.00)**
Subject Education (years)	16.22 (2.83)	14.23 (2.76)**
Subject Education (level)	5.77 (1.03)	5.03 (0.99)**
Father's Education (years)	13.12 (3.62)	11.53 (3.52)**
Father's Education (level)	4.39 (1.60)	3.63 (1.55)**
Mother's Education (years)	12.68(2.67)	12.07 (3.12)*
Mother's Education (level)	4.22 (1.28)	3.83 (1.46)**
Highest Parental Education (years)	13.90 (3.21)	12.74 (3.37)**
Highest Parental Education (level)	4.75 (1.38)	4.20 (1.37)**

P<.01**, P<.05*

Table 104: *Comparison of SES indicators between Caucasian versus African Americans with the metabolic syndrome (NCEP).*

	Metabolic syndrome Caucasians (n=163) Mean (SD)	Metabolic Syndrome African Americans (n=23) Mean (SD)
Family Income (1-8)	5.52 (2.05)	4.00 (2.28)**
Subject Education (years)	15.84 (2.75)	13.23 (1.82)**
Subject Education (level)	5.63 (1.01)	4.71 (0.59)**
Father's Education (years)	12.69 (3.57)	10.00 (3.29)**
Father's Education (level)	4.18 (1.59)	3.18 (1.33)*
Mother's Education (years)	12.39 (2.56)	10.52 (3.52)**
Mother's Education (level)	4.09 (1.25)	3.18 (1.63)**
Highest Parental Education (years)	13.65 (3.09)	11.18 (3.30)**
Highest Parental Education (level)	4.62 (1.35)	3.71 (1.21)**

P<.01**, P<.05*

Table 105: *Comparison of SES indicators between African Americans with and without the metabolic syndrome (NCEP)*

	Non-metabolic syndrome African Americans (n=68) Mean (SD)	Metabolic Syndrome African Americans (n=23) Mean (SD)
Family Income (1-8)	4.19 (1.96)	4.00 (2.28)
Subject Education (years)	14.50 (2.04)	13.23 (1.82)
Subject Education (level)	5.11 (1.07)	4.71 (0.59)
Father's Education (years)	11.92 (3.55)	10.00 (3.29)*
Father's Education (level)	3.76 (1.60)	3.18 (1.33)
Mother's Education (years)	12.50 (2.94)	10.52 (3.52)*
Mother's Education (level)	4.01 (1.40)	3.18 (1.63)*
Highest Parental Education (years)	13.16 (3.35)	11.18 (3.30)*
Highest Parental Education (level)	4.35 (1.40)	3.71 (1.21)

P<.01**, P<.05*

Table 106: *Comparison of SES indicators between subjects from intact versus non-intact families*

	Both parents present (n=843) Mean (SD)	One parent present (n= 60) Mean (SD)
Family Income (1-8)	5.51 (2.04)	4.17 (2.16)**
Subject Education (years)	16.01 (2.89)	14.92 (3.18)**
Subject Education (level)	5.69 (1.05)	5.25 (1.08)**
Highest Parental Education (years)	13.78 (3.25)	12.30 (2.48)**
Highest Parental Education (level)	4.70 (1.38)	3.83 (1.41)**

P<.01**, P<.05*

7.0 BIBLIOGRAPHY

Adler, N.E., Boyce, T., Chesney, M.A., Cohen, S., Folkman, S., Kahn, R.L., et al. (1994). Socioeconomic Status and Health: The Challenge of the Gradient. *American Psychologist*, 49, 15-24.

Anderson, N.B., & Armstead, C.A. (1995). Toward understanding the association of socioeconomic status and health: a new challenge for the biopsychosocial approach. *Psychosomatic Medicine*, 57, 213-225.

Backlund, E., Sorlie, P.D., & Johnson, N.J. (1996). The Shape of the Relationship Between Income and Mortality in the United States: Evidence from the National Longitudinal Mortality Study. *Annals of Epidemiology*, 6, 12-20.

Bao, W., Srinivasan, S.R., & Berenson, G.S. (1996). Persistent elevation of plasma insulin levels is associated with increased cardiovascular risk in children and young adults. The Bogalusa Heart Study. *Circulation*, 93, 54-59.

Berenson, G.S., Wattigney, W.A., Tracy, R.E., Newman, W.P., Srinivasan, S.R., Webber, L.S., et al. (1992). Atherosclerosis of the aorta and coronary arteries and cardiovascular risk factors in persons aged 6 to 30 years and studied at necropsy: The Bogalusa Heart Study. *American Journal of Cardiology*, 70, 851-858.

Behrman, J.R., & Taubman, P. (1990). The intergenerational correlation between children's adult earnings and their parents' income: Results from the Michigan Panel Survey of Income Dynamics. *Review of Income and Wealth*, 36, 115-127.

Black, P.H. (2003). The inflammatory response is an integral part of the stress response: Implications for atherosclerosis, insulin resistance, type II diabetes and metabolic syndrome X. *Brain, Behavior, and Immunity*, 17, 350-364.

Blane, D., Hart, C.L., Davey Smith, G., Gillis, C.R., Hole, D. J., & Hawthorne, V. M. (1996). Association of cardiovascular disease risk factors with socioeconomic position during childhood and during adulthood. *BMJ*, 313, 1434-1438.

Bobak M, Hertzman C, Skodova Z, & Marmot M. (1999). Socioeconomic status and cardiovascular risk factors in the Czech Republic. *International Journal of Epidemiology*, 28, 46-52.

Brunner, E.J., Marmot, M.J., White, I.R. O'Brien, J.R., Etherington, M.D., Slavin, B.M., et al. (1993). Gender and employment grade differences in blood cholesterol, apolipoproteins and haemostatic factors in the Whitehall II study. *Atherosclerosis,* 102, 195–207.

Brunner, E., Davey Smith, G., Marmot, M., Canner, R., Beksinska, M., O'Brien, J. (1996). Childhood social circumstances and psychosocial and behavioural factors as determinants of plasma fibrinogen. *Lancet*, 347, 1008-1013.

Brunner, E.J., Marmot, M.J., Nanchahal,K., Shipley, M.J., Stansfield, S.A., Juneja, M., et al. (1997). Social Inequality in coronary risk: central obesity and the metabolic syndrome. Evidence form the Whitehall II study. *Diabetologia*, 40, 1341-1349.

Brunner, E., Shipley, M.J., Blane, D., Davey Smith, G., & Marmot, M.G. (1999). When does cardiovascular risk start? Past and present socioeconomic circumstances and risk factors in adulthood. *Journal of Epidemiology and Childhood Health*, 5, 3757-764.

Centers for Disease Control and Prevention. (1996). Physical Activity and Health – A Report of the Surgeon General.

Bumpass, L.L. (2004). Social change and the American family. Annals of the New York *Academy of Sciences*, 1038, 213-219.

Chen, E., Matthews, K.A., & Boyce, W.T. (2002). Socioeconomic Differences in Children's Health: How and Why do these Relationships Change with Age? *Psychological Bulletin*, 128, 295-329.

Clausen, J.O., Ibsen, H., Ibsen, K.K., & Borch-Johnsen, K. (1996). Association of body mass index, blood pressure and serum levels of triglycerides and high-density lipoprotein cholesterol in childhood with the insulin sensitivity index in young adulthood: a 13-year follow-up. *Journal of Cardiovascular Risk*, 3, 427-433.

Covey, L.S. & Wynder, E.L. (1981). Smoking Habits and Occupational Status. *Journal of Occupational Medicine*, 23, 537-542.

Dahl, E. (1993). Social Inequality in Health – The Role of the Healthy Worker Effect. *Social Science and Medicine,* 36, 1077-1086.

Dallongeville, J., Cottel, D., Ferrieres, J., Arveiler, D., Bingham, A., Ruidavets, J.B., et al. (2005). Household income is associated with the risk of metabolic syndrome in a sex-specific manner. *Diabetes Care*, 28, 409-415.

Davey Smith, G., Hart, C., Blane, D., & Hole, D. (1998). Adverse socioeconomic conditions in childhood and cause specific adult mortality: prospective observational study. *BMJ,* 316, 1631-1635.

Davey Smith, G. & Hart, C. (2002). Life-course socioeconomic and behavioral influences on cardiovascular disease mortality: The Collaborative Study. *American Journal of Public Health,* 92, 1295-98.

Davey Smith, G., Ben-Shlomo, Y., & Lynch, J. (2002). Life course approaches to inequalities in coronary heart disease risk. In S.A. Stansfield & M.G. Marmot (Eds.), *Stress and the Heart*. Pp. 20-49. London: BMJ Books.

Dedman, D.J., Gunnell, D., Davey Smith, G., & Frankel, S. (2001). Childhood housing conditions and later mortality in the Boyd Orr cohort. *Journal of Epidemiology & Childhood Health,* 55, 10-15.

Dekkers, J.C., Sneider, H., van den Oord, E.J.C.G., & Treiber, F.A. (2002). Moderators of blood pressure development from childhood to adulthood: A 10-year longitudinal study. *Journal of Pediatrics,* 141, 770-779.

Diez-Roux, A.V., Nieto, F.J., Tyroler, H.A., Crum, L.D., & Szklo, M**.** (1995). Social inequalities and atherosclerosis. The atherosclerosis risk in communities study. *American Journal of Epidemiology*, 141, 960-972.

Dobson, A.J., Gibberd, R.W., Leeder S.R., & O'Connell, D.L. (1985). Occupational Differences in Ischemic Heart Disease Mortality and Risk Factors in Australia. *American Journal of Epidemiology,* 122, 283-290.

Donahue, R.P., Orchard, T.J., Kuller, L.H., & Drash, A.L. (1985). Lipids and lipoproteins in a young adult population. The Beaver County Lipid Study. *American Journal of Epidemiology,* 122, 458-467.

Dressler, W.W., Oths, K.S. & Gravlee, C.C. (2005). Race and Ethnicity in Public Health: Models to Explain Health Disparities. *Annual Review of Anthropology*, 34, 231-252.

Ebrahim, S., Montaner, D., & Lawlor, D.A. (2004). Clustering of risk factors and social class in childhood and adulthood in British women's heart and health study: cross sectional analysis. *BMJ*, 328, 861-865.

Elo, I.T., & Preston, S.H. (1996). Educational differentials in mortality: United States, 1979–85. *Social Science and Medicine*, 42**,** 47–57.

Evenson, K.R., Wilcox, S., Pettinger, M., Brunner, R., King, A.C., & McTiernan, A. (2002). Vigorous Leisure Activity Through Women's Adult Life – The Women's Health Initiative Observational Cohort Study. *American Journal of Epidemiology*, 156, 945-953.

Flegal, K.M., Carroll, M.D., Ogden, C.L., & Johnson, C.L. (2002). Prevalence and trends in obesity among US adults, 1999–2000. *JAMA*, 288, 1723–1727.

Ford, E.S., Merritt, R.K., Heath, G.W., Powell, K.E., Washburn, R.A., Kriska, A., et al. (1991). Physical Activity Behaviors in Lower and Higher Socioeconomic Status Populations. *American Journal of Epidemiology*, 133, 1246-1256.

Ford, E.S., Giles, W.H., & Dietz, W.H. (2002). Prevalence of the metabolic syndrome among US adults: findings from the third National Health and Nutrition Examination Survey. *JAMA*, 287, 356-359.

Ford, E.S., Giles, W.H., & Mokdad, A.H. (2004). Increasing Prevalence of the Metabolic Syndrome Among U.S. Adults. *Diabetes Care*, 27, 2444-2449.

Frankel, S., Smith, G.D., & Gunnell, D. (1999). Childhood socioeconomic position and adult cardiovascular mortality: the Boyd Orr Cohort. *American Journal of Epidemiology*, 150, 1081-1084.

Fuentes, R.M., Notkola, I.L., Shemeikka, S., Tuomilehto, J., & Nissinen, A. (2003). Tracking of body mass index during childhood: a 15-year prospective population-based family study in eastern Finland. *International Journal of Obesity Related Metabolic Disorders*, 27, 716-721.

Gallo, L.C. & Matthews, K.A. (2003). Understanding the association between socioeconomic status and physical health: do negative emotions play a role? *Psychology Bulletin*, 129, 10-51.

Galobardes, B., Lynch, J.W., & Davey Smith, G. (2004). Childhood socioeconomic circumstances and cause-specific mortality in adulthood: systematic review and interpretation. *Epidemiology Review*, 26, 7-21.

Garn, S.M., & Clark, D. (1975). Nutrition, Growth, development, and maturation: Findings of the Ten State Nutritional Survey of 1968-1970. *Pediatrics,* 56, 306-319.

Gillum, R.F., & Paffenbarger, R.S. Jr. (1978). Chronic disease in former college students. XVII. Sociocultural mobility as a precursor of coronary heart disease and hypertension. *American Journal of Epidemiology*, 108, 289-298.

Gliksmen, M.D., Kawachi, I., Hunter, D., Colditz, G.A., Manson, J.E. Stampfer, M. J., et al. (1995). Childhood socioeconomic status and risk of cardiovascular disease in middle aged US women: a prospective study. *Journal of Epidemiology and Childhood Health*, 49, 10-15.

Grundy, S.M. (1999). Hypertriglyceridemia, Insulin Resistance, and the Metabolic syndrome. *American Journal of Cardiology*, 83, 25F-29F.

Grundy, S.M., Cleeman, J.I., Daniels, S.R., Donato, K.A., Eckel, R.H., Franklin, B.A., et al. (2005). Diagnosis and Management of the Metabolic Syndrome: An American Heart Association/National Heart, Lung, and Blood Institute Scientific Statement. *Circulation*, 112, 1-6.

Guo, S.S., & Chumlea, W.C. (1999). Tracking of body mass index in children in relation to overweight in adulthood. *American Journal of Clinical Nutrition*, 70, 145S-8S.

Hart, C.L., & Davey Smith, G. (2003). Relation between number of siblings and adult mortality and stroke risk: 25 year follow up of men in the Collaborative study. *Journal of Epidemiology & Childhood Health*, 57, 385-391.

Hasle, H. (1990). Association between living conditions in childhood and myocardial infarction. *BMJ*, 300, 512-513.

Hay, D.R., & Foster, F.H. (1981). The Influence of Race, Religion, Occupation and Other Social Factors on Cigarette Smoking in New Zealand. *International Journal of Epidemiology*, 10, 41-43.

Heiss, G., Johnson, N.J., Reiland, S., Davis, C.E., & Tyroler, H.A. (1980). The epidemiology of plasma high-density lipoprotein cholesterol levels. The Lipid Research Clinics Program Prevalence Study. Summary. *Circulation*, 62, 116-136.

Hemmingsson T., & Lundberg, I. (2004). How far are socioeconomic differences in coronary heart disease hospitalization, all-cause mortality and cardiovascular mortality among adult Swedish males attributable to negative childhood circumstances and behaviour in adolescence? *International Journal of Epidemiology*, August 27 Epub.

Inchley, J.C., Currie, D.B., Todd, J.M., Akhtar, P.C., & Currie, C.E. (2005). Persistent socio-demographic differences in physical activity among Scottish schoolchildren 1990–2002. *The European Journal of Public Health,* 15, 386-388.

International Diabetes Federation. (2004). Backgrounder 1: The IDF consensus worldwide definition for the metabolic syndrome. *www.idf.org*, 1-7.

Jonas, B.S., Franks, P., & Ingram, D.D. (1997). Are symptoms of anxiety and depression risk factors for hypertension? Longitudinal evidence from the National Health and Nutrition Examination Survey I Epidemiologic Follow-up study. *Archives of Family Medicine*, 6, 43-49.

Jousilahti, P., Salomaa, V., Rasi, V., Vahtera, E., & Palosuo, T. (2003). Association of markers of systemic inflammation, C reactive protein, serum amyloid A, and fibrinogen, with socioeconomic status. *Journal of Epidemiology and Childhood Health*, 57, 730-733.

Kaplan, G.A. & Salonen, J.T. (1990). Socioeconomic conditions in childhood and ischemic heart disease in middle age. *BMJ*, 301, 1121-3.

Kaplan, G.A.& Keil, J.E. (1993). Special Report: Socioeconomic Factors and Cardiovascular Disease: A Review of the Literature. *Circulation*, 88, 1973-98.

Keil, J.E., Tyroler, H.A., Sandifer, S.H., & Boyle, E. Jr. (1977). Hypertension: effects of social class and racial admixture: the results of a cohort study in the black population of Charleston, South Carolina. *American Journal of Public Health*, 67,634-639.

Keil, J.E., Sutherland, S.E., Knapp, R.G., & Tyroler, H.A. (1992). Does Equal Socioeconomic Status in Black and White Men Mean Equal Risk of Mortality? *American Journal of Public Health*, 82, 1133-1136.

Kivimaki, M., Smith, G.D., Juonala, M., Ferrie, J.E., Keltikangas-Jarvinen, L., Elovainio, M., et al. (2006). Socioeconomic position in childhood and adult cardiovascular risk factors,

vascular structure, and function: cardiovascular risk in young Finns study. *Heart*, 92, 474-480.

Kitagawa, E.M. & Hauser, P.M. (1973). Differential mortality in the United States: a study in socioeconomic epidemiology. Cambridge, Mass.: Harvard University Press.

Ko, G.T., Chan, J.C., Yeung, V.T., Chow, C.C., Tsang, L.W., & Cockram, C.S. (2001). A low socio-economic status is an additional risk factor for glucose intolerance in high risk Hong Kong Chinese. *European Journal of Epidemiology*, 17, 289-295.

Koskinen, S. & Martelin, T. (1994). Why are socioeconomic mortality differences smaller among women than among men? *Social Science and Medicine*, 38, 1385–1396.

Krieger, N., Williams, D.R., & Moss, N.E. (1997). Measuring social class in U.S. health research: Concepts, methodologies, and guidelines. *Annual Review of Public Health*, 18, 341-378.

Krieger, N. (2000). Discrimination and Health. In: Berkman, L.F. & Kawachi, I. (Eds.). *Social Epidemiology*. New York: Oxford University Press.

Laaksonen, D.E., Lakka, H.M., Niskanen, L.K., Kaplan, G.A., Salonen, J.T., Lakka, T.A. (2002). Metabolic Syndrome and Development of Diabetes Mellitus: Application and Validation of Recently Suggested Definitions of the Metabolic Syndrome in a Prospective Cohort Study. *American Journal of Epidemiology*, 156, 1070-1077.

Lakka, H.M., Laaksonen, D.E., Lakka, T.A., Niskanen, L.K., Kumpusalo, E., Tuomilehto, J., et al. (2002). The metabolic syndrome and total and cardiovascular disease mortality in middle-aged men. *JAMA*, 288, 2709-2716.

Langnase, K., Mast, M., & Muller, M.J. (2002). Social class differences in overweight of prepubertal children in northwest Germany. *International Journal of Obesity Related Metabolic Disorders,* 26, 566-572.

Lapidus, L., Bengtsson, C., Hallstrom, T., & Bjorntorp, P. (1989). Obesity, adipose tissue distribution and health in women--results from a population study in Gothenburg, Sweden. *Appetite*, 13, 25-35.

Lawlor D.A., Ebrahim S., & Davey Smith G. (2002). Socioeconomic position in childhood and adulthood and insulin resistance: cross sectional survey using data from British women's heart and health study. *BMJ*, 325, 805-810.

Lehman, B.J., Taylor, S.E., Kiefe, C.I., & Seeman, T.E. (2005). Relation of childhood socioeconomic status and family environment to adult metabolic functioning in the CARDIA Study. *Psychosomatic Medicine*, 67, 846-854.

Leon, D.A., Koupilova, I., & Lithell, H.O. (1996). Failure to realize growth potential in utero and adult obesity in relation to blood pressure in 50 year old Swedish men. *BMJ*,312,401–

406.

Linn, S., Fulwood, R., Rifkind, B., Carroll, M., Muesing, R., Williams, O.D., et al. (1989). High density lipoprotein cholesterol levels among US adults by selected demographic and socioeconomic variables. The Second National Health and Nutrition Examination Survey 1976-1980. *American Journal of Epidemiology*,129, 281-94.

Liu, K., Cedre, L.B., Stamler, J., Dyer, A., Stamler, R., Nanas, S., et al. (1982). Relationship of education to major risk factors and death from coronary heart disease, cardiovascular diseases and all causes, findings of three Chicago epidemiologic studies. *Circulation*, 66, 1308-1314.

Luepker, R.V., Rosamond, W.D., Murphy, R., Sprafka, J.M., Folsom, A.R., McGovern, P.G., et al. (1993). Socioeconomic status and coronary heart disease risk factor trends. The Minnesota Heart Survey. *Circulation*, 88, 2172-2179.

Lynch, J. & Kaplan, G. (2000). Socioeconomic Position. In L.F. Berkman & I. Kawachi (Eds.), *Social Epidemiology* Pp. 13-35. Oxford, England: Oxford University Press.

Magarey, A.M., Daniels, L.A., Boulton, T.J., & Cockington, R.A. (2003). Predicting obesity in early adulthood from childhood and parental obesity. *International Journal of Obesity Related Metabolic Disorders,* 27, 505-513.

Marmot, M.G., Kogevinas, M., & Elston, M.A. (1987). Social/Economic status and disease. *Annual Review of Public Health*, 8, 111-135.

Marmot, M.G., Davey Smith, G., Stansfeld, S., Patel, C., North, F., Head, J., et al. (1991). Health inequalities among British Civil Servants: the Whitehall II study. *Lancet*, 337, 1387-1394.

Marmot, M., Shipley, M., Brunner, E., & Hemingway, H. (2001). Relative contribution of early life and adult socioeconomic factors to adult morbidity in the Whitehall II study. *Journal of Epidemiology and Childhood Health*. 55, 301-307.

Matthews, S., Manor, O., & Power, C. (1999). Social Inequalities in Health: Are There Gender Differences? *Social Science and Medicine*, 48, 49-60.

McCaffrey, J.M., Shen, B.J., Niaura, R., Muldoon, M.F., & Manuck, S.B. (In press). Blood pressure and the metabolic syndrome in normotensive and untreated hypertensive men.

McCarron, P., Smith, G.D., Okasha, M., & McEwen, J. (2000). Blood pressure in young adulthood and mortality from cardiovascular disease. *Lancet,* 355, 1430-1431.

McFarlane**,** S.I., Banerji, M., & Sowers, J.R. (2001). Insulin Resistance and Cardiovascular Disease. *Journal of Clinical Endocrinology and Metabolism*, 86**,** 713-718.

Modin, B. (2003). Born out of wedlock and never married--it breaks a man's heart. Social *Science & Medicine*, 57, 487-501..

Mulligan, C.B. (1999). Galton versus the Human Capital Approach to Inheritance. *Journal of Political Economy,* 107, S184-201.

National Institutes of Health. (2001). Third Report of the National Cholesterol Education Program Expert Panel on Detection, Evaluation, and Treatment of High Blood Cholesterol in Adults (Adult Treatment Panel III). Bethesda, Maryland: National Institutes of Health. NIH Publication 01-3670.

Nelson, M.J., Ragland, D.R., & Syme, S.L. (1992). Longitudinal prediction of adult blood pressure from juvenile blood pressure levels. *American Journal of Epidemiology*, 136, 633-645.

Niaura, R., Stoney, C.M., & Herbert, P.N. (1992). Lipids in psychological research: the last decade. *Biological Psychology*, 34, 1–43.

Nicklas, T.A., von Duvillard, S.P., & Berenson, G.S. (2002). Tracking of serum lipids and lipoproteins from childhood to dyslipidemia in adults: the Bogalusa Heart Study. *International Journal of Sports Medicine*, 23, S39-43.

Notkola, V., Punsar, S., Karvonen, M.J., & Haapakoski, J. (1985). Socio-economic conditions in childhood and mortality and morbidity caused by coronary heart disease in adulthood in rural Finland. *Social Science and Medicine*, 21, 517-523.

Osmond C., Barker, D.J.P., Winter, P.D., Fall, C.H.D., & Simmonds, S.J. (1993). Early growth and death from cardiovascular disease in women. *BMJ*, 307, 1519-1524.

Owen, N., Poulton, T., Hay, F.C., Mohamed-Ali, V., & Steptoe, A. (2003). Socioeconomic status, C-reactive protein, immune factors, and responses to acute mental stress. *Brain, Behavior, and Immunity*, 17, 286-295.

Pappas, G., Queen, S., Hadden, W., & Fisher, G. (1993). The increasing disparity in mortality between socioeconomic groups in the United States, 1960 and 1986. *New England Journal of Medicine*, 329, 103-109.

Park, Y., Zhu, S., Palaniappan, L., Heshka, S., Carnethon, M.R., & Heymsfield, S.B. (2003). The Metabolic syndrome: Prevalence and associated risk factor findings in the U.S. population from the third national Health and Nutrition Examination Survey, 1988-1994). *Archives of Internal Medicine*, 163, 427-435.

Parker, L., Lamont, D.W., Unwin, N., Pearce, M.S., Bennett, S.M.A., Dickinson, H.O., et al. (2003). A lifecourse study of risk for hyperinsulemia, dyslipidaemia and obesity (the central metabolic syndrome) at age 49-51 years. *Diabetic Medicine*, 20, 406-415.

Pensola, T. H., & Martikainen, P. (2003). Cumulative social class and mortality from various causes of adult men. *Journal of Epidemiology and Childhood Health*, 57, 745-751.

Phillips, D.I.W., Walker, B.R., Reynolds, R.M., Flanagan, D.E.H., Wood, P.J., Osmond, C., et al. (2000). Low birthweight and elevated plasma cortisol concentrations in adults from three populations. *Hypertension*, 35, 1301–1306.

Pierce, J.P., Fiore, M.C., Novotny, T.E., Hatzianndreu, E.J., & Davis, R.M. (1989). Trends in Cigarette Smoking in the United States. Educational Differences are Increasing. *JAMA*, 261, 56-60.

Poulton, R., Avshalom, C., Milne, B. J., Thompson, W. M., Taylor, A., Sears, M.R., et al. (2002). Association between children's experience of socioeconomic disadvantage and adult health: a life-course study. *Lancet*, 360, 1640-45.

Raikkonen, K., Hautanen, A., & Keltikangas-Jarvinen, L. (1994). Association of stress and depression with regional fat distribution in healthy middle-aged men. *Journal of Behavioral Medicine,* 17, 605-616.

Raikkonen, K., Keltikangas-Jarvinen, L., Adlercreutz, H., & Hautanen, A. (1996). Psychosocial stress and the insulin resistance syndrome. , 45, 1533-1538.

Räikkönen, K., Matthews, K. A., & Salomon, K. (2003). Hostility Predicts Metabolic Syndrome Risk Factors in Children and Adolescents. *Health Psychology*, 22, 279–286.

Reaven, G. (1999). Insulin resistance: A chicken that has come home to roost. *Annals of the New York Academy of Sciences*, 892, 45-57.

Reynolds, R., Walker, B.R., Syddall, H.E., Andrew, R., Wood, P.J., & Phillips, D.I.W. (2005). Is there a gender difference in the associations of birthweight and adult hypothalamic–pituitary–adrenal axis activity? *European Journal of Endocrinology*, Vol 152, Issue 2, 249-253.

Richards, J.C., Hof, A., & Alvarenga, M. (2000). Serum lipids and their relationships with hostility and angry affect and behaviors in men. *Health Psychology*, 19, 393–398.

Rose, G., & Marmot, M.G. (1981). Social Class and Coronary Heart Disease. *British Heart Journal,* 45, 13-19.

Rowe, D.C., Vesterdal, W.J., & Rodgers, J.L. (1999). Herrnstein's syllogism: Genetic and shared environmental influences on IQ, education, and income. *Intelligence*, 26, 405-423.

Sallis, J.F., Prochaska, J.J., & Taylor, W.C. (2000). A review of correlates of physical activity of children and adolescents. *Medicine & Science in Sports & Exercise*. 32, 5, 963-975.

Salonen, J.T. (1982). Socioeconomic Status and Risk of Cancer, Cerebral Stroke, and Death Due to Coronary Heart Disease and Any Disease: a Longitudinal Study in Eastern Finland. *Journal of Epidemiology and Childhood Health*, 36, 294-297.

Shen, B.J., Todaro, J.F., Niaura, R., McCaffery, J.M., Zhang, J., Spiro III, et al. Are metabolic risk factors one unified syndrome? Modeling the structure of the metabolic syndrome X. *American Journal of Epidemiology*, 157, 701-11.

Siegel, D., Kuller, L., Lazarus, N.B., Black, D., Feigal, D., Hughes, G., et al. (1987). Predictors of Cardiovascular Events and Mortality in the Systolic Hypertension in the Elderly Program Pilot Project. *American Journal of Epidemiology*, 126, 385-399.

Sobal, A. & Stunkard, A.J. (1989). Socioeconomic Status and Obesity: A Review of the Literature. *Psychological Bulletin*, 105, 260-275.

Solon, G. (1992). Intergenerational Income Mobility in the United States. *American Economic Review*, 82, 393-408.

Sorel, J.E., Ragland, D.R., Syme, S.L. & Davis, W.B. (1992). Educational status and blood pressure: the Second National Health and Nutrition Examination Survey, 1976-1980, and the Hispanic Health and Nutrition Examination Survey, 1982-1984. *American Journal of Epidemiology*, 135, 1339-1348.

Steenland, K., Henley, J., Calle, E., & Thun, M. (2004). Individual- and area-level socioeconomic status variables as predictors of mortality in a cohort of 179,383 persons. *American Journal of Epidemiology*, 159, 1047-1056.

Stronks, K., van de Mheen, H., van den Bos, J., & Mackenbach, J.P. (1995). Smaller Socioeconomic Inequalities in Health Among Women: the Role of Employment Status. *International Journal of Epidemiology*, 24, 559-568.

Thurston, R.C., Kubzansky, L.D., Kawachi, I., & Berkman, L.F. (2005). Is the association between socioeconomic position and coronary heart disease stronger in women than in men? *American Journal of Epidemiology*, 162, 57-65.

Tomlinson-Keasey, C. & Little, T.D. (1990). Predicting educational attainment, occupational achievement, intellectual skill, and personal adjustment among gifted men and women. *Journal of Educational Psychology*, 82, 442-455.

Vagero, D., & Leon, D. (1994). Effect of social class in childhood and adulthood on adult mortality. *Lancet*, 343, 1224-1225.

Van de Mheen, H., Stronks, K., van den Bos, J., & Mackenbach, J.P. (1997). The contribution of childhood environment to the explanation of socioeconomic inequalities in health in adult life: a retrospective study. *Social Science and Medicine*, 44, 13-24.

Vargas, C.M., Ingram, D.D., & Gillum, R.F. (2000). Incidence of hypertension and educational attainment: the NHANES I epidemiologic follow-up study. First National Health and Nutrition Examination Survey. *American Journal of Epidemiology*, 152, 272-278.

Vogels, E.A., Lagro-Janssen, A.L., & van Weel, C. (1999). Sex differences in cardiovascular disease: are women with low socioeconomic status at high risk? *British Journal of General Practice,* 49, 963–966.

Wamala, S.P., Lynch, J., & Kaplan, G.A. (2001). Women's exposure to early and later life socioeconomic disadvantage and coronary heart disease risk: the Stockholm Female Coronary Risk Study. *International Journal of Epidemiology*, 30, 275-284.

Wannamethee, G. S., Whincup, P. H., Shaper, G., & Walker, M. (1996). Influence of father's social class on cardiovascular disease in middle-aged men. *Lancet*, 348, 1259-1263.

Wattigney, W.A., Webber, L.S., Srinivasan, S.R., & Berenson, G.S. (1995). The emergence of clinically abnormal levels of cardiovascular disease risk factor variables among young adults: the Bogalusa Heart Study. *Preventive Medicine*, 24, 617-626.

Willms, J.D., Tremblay, M.S., & Katzmarzyk, P.T. (2003). Geographic and demographic variation in the prevalence of overweight Canadian children. *Obesity Research*, 11, 668-673.

Wilson TW, Kaplan GA, Kauhanen J, Cohen RD, Wu M, Salonen R, et al. (1993). Association between plasma fibrinogen concentration and five socioeconomic indices in the Kuopio Ischemic Heart Disease Risk Factor Study. *American Journal of Epidemiology,* 137, 292-300.

Wing, R.R., Matthews, K.A., Kuller, L.H., Meilahn, E.L., & Plantinga, P. (1991). Waist to hip ratio in middle-aged women. Associations with behavioral and psychosocial factors and with changes in cardiovascular risk factors. *Arteriosclerosis and Thrombosis*, 11, 1250-1257.

Winkleby, M.A., Robinson, T.N., Sunquist, J., & Kraemer, H.C. (1999). Ethnic variation in cardiovascular disease risk factors among children and young adults: findings from the Third National Health and Nutrition Examination Survey, 1988-1994. *JAMA*, 281, 1006-1013.

Winkleby, M.A., Kraemer, H.C., Ahn, D.K., & Varady, A.N. (1998). Ethnic and socioeconomic differences in cardiovascular disease risk factors: findings for women from the Third National Health and Nutrition Examination Survey, 1988-1994. *JAMA*, 280, 356-362.

World Health Organization. (1999). Definition, diagnosis and classification of diabetes mellitus and its complications. WHO Consultation, Geneva, 31-33.

Yan, L.L., Liu, K., Matthews, K.A., Daviglus, M.L., Ferguson, T.F., & Kiefe, C.I. (2003). Psychosocial Factors and Risk of Hypertension; The Coronary Artery Risk Development in Young Adults (CARDIA) Study. *JAMA*, 290, 2138-2148.

Zang, E.A. & Wynder, E.L. (1998). Smoking Trends in the United States between 1969 and 1995 Based on Patients Hospitalized with Non-Smoking Related Diseases. *Preventive Medicine*, 27, 854-861.

Zhang, Q. & Wang, Y. (2004). Trends in the Association between Obesity and Socioeconomic Status in U.S. Adults: 1971 to 2000. *Obesity Research*, 12, 1622-1632.