LIFECOURSE SOCIOECONOMIC POSITION & CARDIOVASCULAR HEALTH

by

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DEDICATION

This work is dedicated to my husband: thank you for your love and support and for putting up with me, insanity and all.

ACKNOWLEDGEMENTS

My name may be the one front and center on this document, but this dissertation would have never been possible without the guidance and support of many people. The faculty of this department has provided me with a tremendous graduate education that I highly value and believe is unparalleled in the world of social epidemiology. Several individuals deserve special attention for their contribution to this body of work, but the ideas enclosed, the number of people involved in collection and management of data, and the love and support required for a needy graduate student makes a very large village.

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ABSTRACT

Background: A major limitation of past work on the social patterning of cardiovascular disease has been the reliance of measures of socioeconomic position (SEP) assessed at a single point in time in adulthood. The purpose of this dissertation research was to move beyond this limitation to explore how individuals, neighborhoods of residence, and health interact with each other over time. **Methods:** Chapter 2 utilized the Multi-Ethnic Study of Atherosclerosis (MESA) to examine the relation between childhood SEP [CSEP], adult SEP [ASEP], and 20-year average exposure to neighborhood poverty [NSEP] with the prevalence of subclinical atherosclerosis, as assessed by common carotid intimal-medial thickness (IMT). Chapter 3, using MESA also, used latent trajectory class modeling to determine patterns of neighborhood poverty over a 20-year period and then examined how these patterns were related to the amount of IMT, and associated risk factors. Chapter 4 used the Alameda County Study to examine if health-related indicators were associated with socioeconomic changes in residential mobility. **Results:** After adjustment for age, CSEP and ASEP were both inversely and independently associated with IMT, with slightly stronger associations in women than men. NSEP was only associated with IMT in women. There was evidence of heterogeneity in ASEP- and NSEP by race/ethnicity. Distinctive clusters of neighborhood poverty were found over the 20-year period; 5 out of 6 stable and one upwardly mobile. Generally, and mostly in women, higher stable neighborhood poverty (SNP) was associated with worse

cardiovascular outcomes than lower SNP. All medical conditions showed non-significant decreased odds of moving to a 'better' neighborhood and a 'worse' neighborhood (based on changes in census tract median family income between study waves). BMI was the only health indicator to be associated with both increased odds of moving to a better neighborhood and decreased odds of moving to a worse neighborhood. **Conclusion:** Lower SEP at multiple points in the lifecourse and higher neighborhood poverty over 20 years was related to worse cardiovascular health. If health selection is occurring, it is most likely to be an indirect result of the socioeconomic consequences of extreme weight or a general increased mobility of healthy individuals.

CHAPTER 1

INTRODUCTION

Specific Aims

Health has long been tied to a person's socioeconomic position within a society. The attendant rights, duties, and lifestyles of a person's relative socioeconomic rank translating into health through complex biological processes not completely understood. Individuals of low income or low education having higher morbidity and mortality, particularly from cardiovascular diseases, than persons of high income or education [1]. More recent work has also shown that the socioeconomic environment in which a person lives may add additional harm or benefit to a person's cardiovascular health over and above their own personal socioeconomic position (SEP) [2-8].

A major limitation of past work on the social patterning of cardiovascular disease (CVD) has been the reliance of measures of individual-level or area-level, particularly neighborhood, SEP assessed at a single point in time in adulthood. Risk of chronic disease is thought to operate throughout the life course, so the use of a single point in time measure may result in underestimates of area effects [9-10]. The purpose of this dissertation research is to move beyond single point in time assessments of neighborhood socioeconomic position (SEP) and to explore how individuals, neighborhoods of residence, and health interact with each other over time. Using data from two large population samples, I will examine the effects of lifecourse measures of SEP and

neighborhood characteristics on atherosclerosis as well as the relationship between cardiovascular health and neighborhood conditions over time.

The specific aims are:

Aim 1: Lifecourse socioeconomic positions and subclinical atherosclerosis

To examine independent contributions of lifecourse individual and neighborhood measures with the prevalence of subclinical atherosclerosis.

Hypothesis 1a. *Higher exposure to childhood SEP, adulthood SEP, and cumulative neighborhood SEP (defined as average exposure to neighborhood census tract poverty over a 20-year period) will be associated with lower subclinical atherosclerosis (as measured by mean common carotid Intima Medial Thickness [IMT]).*

Hypothesis 1b. *All three measures will be independently related to subclinical atherosclerosis.*

Hypothesis 1c. *Associations will not differ by gender or race/ethnicity.*

Aim 2: Trajectories of Neighborhood Poverty and Carotid Atherosclerosis

To use a social mobility model to examine the relationship between different trajectories of neighborhood poverty with the prevalence of sub clinical atherosclerosis, and associated risk factors.

Hypothesis 2a. *There will be significantly different latent trajectory classes of neighborhood census tract poverty over the twenty year period.*

Hypothesis 2b. *Groups with high stable neighborhood census tract poverty over time will have a higher mean IMT and a poorer cardiovascular risk profile than group(s) with low stable percent poverty over time (high stable > middle stable > low stable).*

Hypothesis 2c. *Downward social mobility (increased neighborhood census tract poverty over time) will be associated with an increased mean IMT and a poorer cardiovascular risk profile.*

Hypothesis 2d. *Upward social mobility (decreased neighborhood census tract poverty over time) will be associated with a decreased mean IMT and a better cardiovascular risk profile.*

Aim 3: Health Predictors of Social Mobility

To investigate the role of disease into self-selection of people into disadvantaged neighborhoods.

Hypothesis 3a. *Presence of health-related indicators (heart trouble, stroke, diabetes, body mass index, and current smoking) will be associated with increased odds of dying (as compared to not moving).*

Hypothesis 3b. *Presence of health-related indicators (heart trouble, stroke, diabetes, body mass index, and current smoking) will be associated with decreased odds of moving to a better neighborhood (as compared to not moving).*

Hypothesis 3c. *Presence of health-related indicators (heart trouble, stroke, diabetes, body mass index, and current smoking) will be associated with increased odds of moving to a worse neighborhood (as compared to not moving).*

Hypothesis 3d. *Demographic, socioeconomic, and negative life events will confound the association between the health indicators and dying, moving to a better neighborhood, and moving to a worse neighborhood.*

Background and Significance

Atherosclerosis as a major public health concern

Atherosclerosis is of paramount public health importance. It is the slowly progressive pathological condition, characterized by a thickening of the arteries, which contributes to the major forms of Cardiovascular Disease (CVD). Over 64 million Americans have CVD with resultant > 900,000 deaths a year. Many of these deaths are from heart attack and stroke and almost three-fourths of deaths from CVD are related to atherosclerosis [11].

Atherosclerosis is a life long disease process. The first stages of atherosclerosis, the development of fatty streaks, commonly appear in childhood. These lesions then develop into fibrous plaques, fat and cholesterol accumulate at the site, and smooth muscle cells migrate and multiply. The entire process translates into a narrowing of the arteries, a decrease in blood flow, and an increased risk of the plaque rupturing and creating a blood clot or emboli [9, 12].

Traditional efforts to prevent atherosclerotic development, and hence CVD occurrence, have focused on control of the modifiable risk factors for CVD: hypertension, high blood cholesterol, tobacco use, diet, physical activity, diabetes control, and weight management. It is hypothesized that modest changes in one or more of these risk factors could have a large public health impact. However, interventions at the

individual level have had limited success at reducing the occurrence of these risk factors in individuals and the population at large [1, 11-13].

Adult SEP and CVD

Numerous studies have shown that cardiovascular risk is socially patterned, with persons of low income or low education having higher prevalence, incidence and mortality from cardiovascular disease than persons of high income or education [1]. A variety of measures have been used to conceptualize adult socioeconomic position (SEP), depending on the theoretical foundation and the realities of data collection, but all try to capture some form of social stratification based on an individual's social status and accumulation of goods and resources [14].

Past work has traditionally used income, education, occupation, or indexes of varying combinations that sometimes include neighborhood quality, as indicators of SEP [15]. Each have been strongly and consistently associated with health but are also relatively limited indicators of an individual's place in society. More recent work has documented that accumulated assets, or wealth, may be an equal or stronger predictor of health status [16] and stroke [17] than income, but no study has examined a combined index of income, education, and wealth on CVD, let alone subclinical disease.

Lifecourse SEP and CVD

Atherosclerosis is known to develop over the life-course, possibly beginning as early as childhood [9-10] Thus, the investigation of measures of SEP assessed late in life could result in inaccurate estimates of the social patterning of subclinical disease,

particularly if SEP changes over a person's life-course and if childhood exposures are important to the development of atherosclerosis later in life.

The extent of atherosclerotic development is then a product of a life-time of accumulation and interaction of exposures. Logically, then, any exposure that influences progression of atherosclerosis should be measured over the life-time. The life course approach to epidemiology [18-19] uses this logic to take into account exposures being able to act at different stages of life and how these exposures can singly, or in combination, influence disease risk. There are generally three different life-course models of how exposure over the lifetime could have an effect on the outcome: the accumulation, the social mobility, and the critical period. The accumulation theory is that exposures incrementally increase over a person's life, the social mobility that it is how the exposure changes during the life course, and critical period is that certain stages or time periods increase sensitivity to the exposure, that result in health status later in life.

Neighborhoods and CVD

A number of studies have been published in the past 10 years that show a relationship between where a person lives and their risk of cardiovascular disease, independently of statistical controls for personal socioeconomic characteristics [2-8]. The biological processes responsible for this social patterning of cardiovascular events are not completely known. Neighborhood socioeconomic characteristics could be related to the development of atherosclerosis and/or to the triggering of clinical events or deaths in persons with underlying atherosclerotic disease.

Further proof for the existence of a causal relationship between neighborhood deprivation and CVD comes from associations being found between neighborhoods and factors that mediate a person's risk of developing CVD. Studies looking at different combinations of CVD risk factors - cholesterol, blood pressure, smoking, physical activity, and obesity have found consistent results of neighborhood deprivation being associated with worse cardiovascular outcomes. Most associations remaining significant after adjustment for individual SEP and generally stronger in women than men [3-4, 20-23].

The mechanism that underlies this phenomenon is unclear though. Suggested hypothesis include: certain resources and services being available in certain neighborhoods to promote healthy lifestyles, neighborhoods could contribute to development and persistence of CVD risk factors, the physical infrastructure and/or perceived safety of the neighborhood could affect physical activity patterns, social norms within the neighborhood could promote or hinder healthy behaviors, a deprived neighborhood could produce chronic stress from high levels of noise, violence, and poverty, and/or social support in a neighborhood could promote healthy lifestyle choices [24].

When trying to separate out the true contextual area effects from the effects of the individual-level attributes of the people that live there, it is important to take into account the present and past SEP of the people that live in the neighborhoods and to understand the processes which influence people to move from neighborhood to neighborhood, or not move at all. Lifecourse assessments of individual and neighborhood SEP, and the ability to explore the directionality of the association between SEP and health outcomes

will advance our understanding of the causal processes involved in neighborhoods of residence influencing CVD.

Significance

Atherosclerosis is the process underlying the number one (CHD) and number three (stroke) killers in the United States. Subclinical disease measures have been shown to be graded and show continuous associations with cardiovascular clinical events. Even small reductions in the prevalence of subclinical atherosclerosis would have a considerable impact on the prevalence and occurrence of CVD in the population [23]. This study will improve on past literature, by: (1) exploring the independent associations of individual and neighborhood measures with subclinical atherosclerosis, with adult SEP comprising a summary measure of income, education, and wealth, and neighborhood SEP capturing 20 years of exposure to local environmental conditions (**chapter 2**), (2) looking at neighborhood trajectories as opposed to single point in time measures with subclinical atherosclerosis and associated risk factors (**chapter 3**), and (3) investigating the role of disease with the self-selection of people into disadvantaged neighborhoods (**chapter 4**).

If we find that SEP at multiple points in the lifecourse and at the individual and neighborhood level contribute to the development of atherosclerosis, then CVD prevention may benefit from interventions at all time points and levels. If evidence exists that the population at large is highly mobile and that groups with increased or decreased neighborhood SEP over time have higher or lower amounts of cardiovascular outcomes expected under a purely accumulative model, than assessments of neighborhoods based on adult residence at a single point in time may be inaccurate and resultant estimates of

cardiovascular risk suspect. If people with cardiovascular disease do move to poorer neighborhoods over time, then studies of neighborhood effects on cardiovascular disease will have to take this selection bias into account.

Conceptual framework

Figure 1.1 shows the pathways by which childhood SEP, adult SEP, and neighborhood SEP may have an effect on the prevalence of subclinical atherosclerosis. All three measures may have a direct effect on the development of atherosclerosis by influencing the cardiovascular modifiable risk factors. Adult SEP is certainly influenced by childhood SEP and NSEP determined by adult SEP. Gender and race/ethnicity may be partly confounding the associations of all three SEP measures with IMT.

Figure 1.2 shows an alternative hypothesis that neighborhoods and health are associated not because neighborhoods are causing people to develop cardiovascular disease, but because the development of cardiovascular disease is causing people to move to poorer neighborhoods through the socioeconomic and life event consequences of poor health. Age and race/ethnicity are both related to health status and neighborhood of residence but are not a part of the direct causal pathway between health and neighborhood of residence. Health can determine socioeconomic status, which can have a direct effect on neighborhood of residence and inversely, socioeconomic status can also determine health. Health status can also directly, or indirectly through the socioeconomic consequences of health status, lead to negative life events such as financial problems, job loss, or separation/divorce, that would lead directly to change in residence. Negative life events can also lead to poorer health.

Figure 1.1: Conceptual framework for the possible pathways linking CSEP, ASEP, and NSEP with subclinical atherosclerosis (Aim 1& 2).

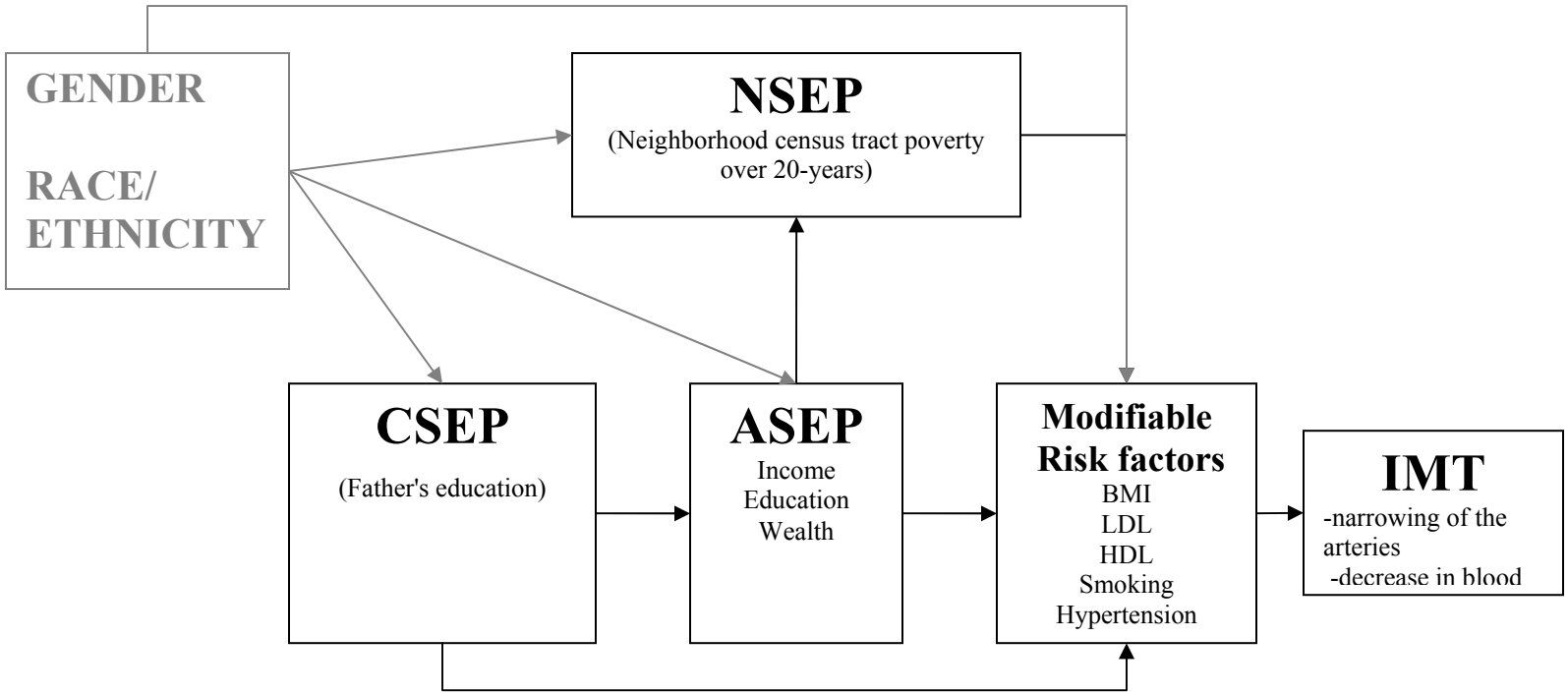
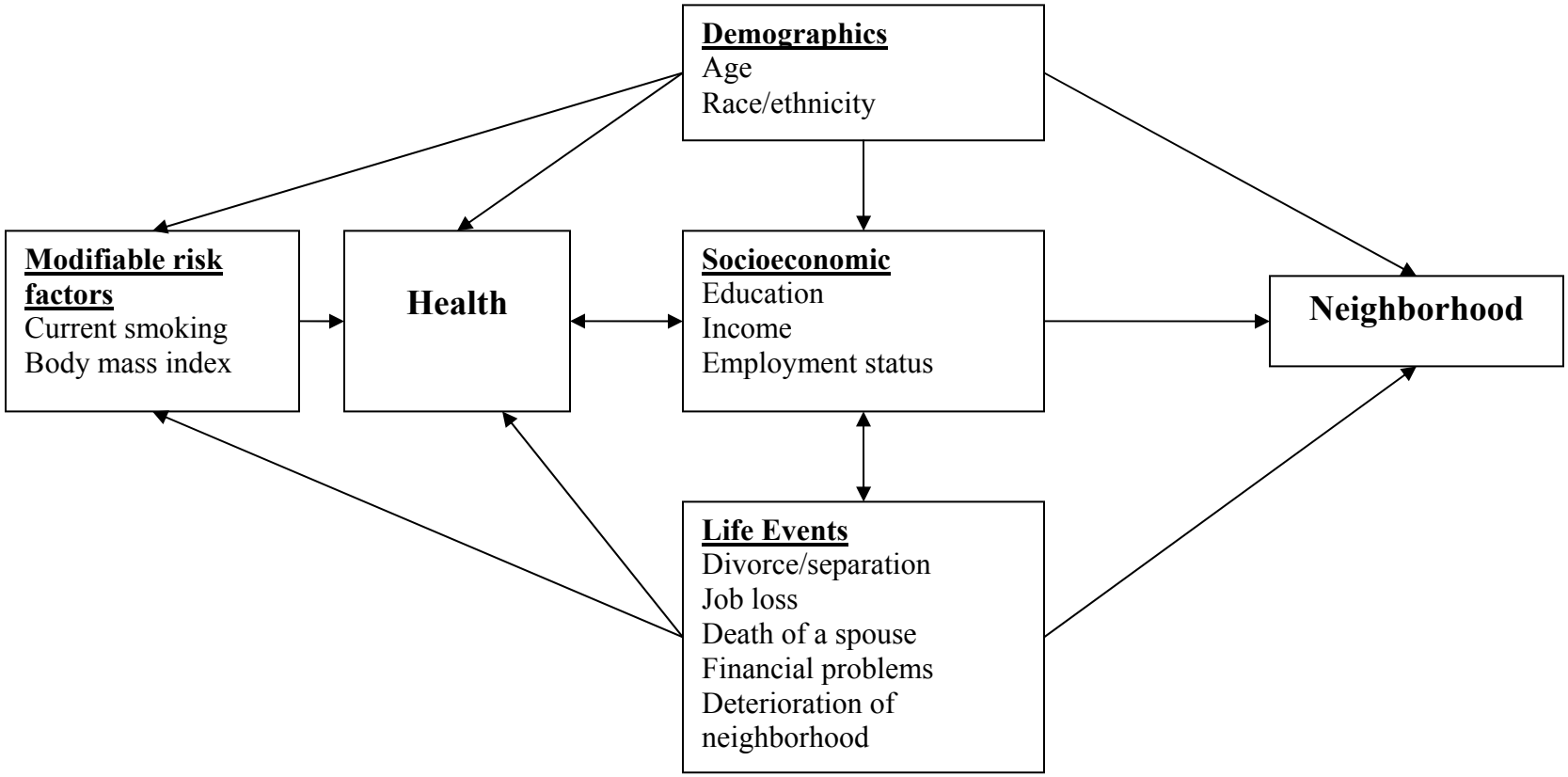


Figure 1.2: Alternative conceptual framework for the possible pathways linking health to residential mobility.



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CHAPTER 2

LIFECOURSE SOCIOECONOMIC POSITIONS AND SUBCLINICAL ATHEROSCLEROSIS IN THE MULTIETHNIC STUDY OF ATHEROSCLEROSIS

INTRODUCTION

Numerous studies have shown that cardiovascular risk is socially patterned, with persons of low income or low education having higher prevalence, incidence and mortality from cardiovascular disease than persons of high income or education [1]**.** Recent work has also shown that cardiovascular risk is patterned by neighborhood socioeconomic characteristics, with persons living in socioeconomically disadvantaged neighborhoods having higher prevalence, incidence, and mortality from cardiovascular disease than those living in more advantaged neighborhoods, even after controlling for measures of personal socioeconomic position (SEP) [2-8].

The biological processes responsible for this social patterning of cardiovascular events are not completely known. SEP could be related to the development of atherosclerosis and/or to the triggering of clinical events or deaths in persons with underlying atherosclerotic disease. A number of studies have investigated the social patterning of subclinical atherosclerosis and have consistently reported a higher prevalence of subclinical atherosclerosis in late adulthood among persons of lower SEP compared to those of higher SEP, as characterized by individual-level or neighborhood-level measures [9-14]. These findings suggest that social factors may pattern the development of atherosclerosis itself.

A major limitation of past work on the social patterning of atherosclerosis has been the reliance on measures of neighborhood or individual-level SEP assessed at a single point in time in adulthood. Atherosclerosis is known to develop over the life-course, possibly beginning as early as childhood [15-16]. Thus, the investigation of measures of SEP assessed late in life could result in inaccurate estimates of the social patterning of subclinical disease, particularly if SEP changes over a person's life-course and if childhood exposures are important to the development of atherosclerosis later in life. Although several studies have reported associations of lifecourse SEP with cardiovascular events [17], only one study of which we are aware has investigated associations of life-course measures of SEP, measured at both the individual and neighborhood-level, with the presence of subclinical atherosclerosis [18]. Identifying an association between life-course SEP and the presence of subclinical atherosclerosis in adulthood would argue for targeting preventative efforts at disadvantaged groups early in life.

Using data from the Multi-Ethnic Study of Atherosclerosis (MESA), a large, multiethnic, cohort study of the determinants of subclinical atherosclerotic disease, we examined if childhood SEP, adulthood SEP, and 20-year average exposure to neighborhood poverty are related to the amount of subclinical atherosclerosis present in mid and late adulthood. We hypothesized that all three measures would be independently related to subclinical atherosclerosis, as assessed by carotid artery intima medial thickness (IMT), a validated measure of early atherosclerosis [19-20].

METHODS

Study Population

The MESA is a population-based study of 6,814 men and women recruited from six US Communities (Baltimore, MD; Chicago, IL; Forsyth County, NC; Los Angeles County, CA; northern Manhattan, NY; and St. Paul, MN). Participants were 45-84 years old and clinically free of cardiovascular disease at baseline. The methods used for sampling and study design have been reported elsewhere [21]. Approximately 38% of the cohort is white, 28% African American, 23% Hispanic, and 11% Chinese. The baseline examination of the cohort (on which these analyses are based) took place between July 2000 and August 2002.

Definition of SEP Measures

Three dimensions of SEP were investigated in these analyses: childhood SEP (CSEP), adult SEP (ASEP), and 20-year average exposure to neighborhood poverty (NSEP). The three dimensions of SEP were investigated because they provide complementary information on exposure to social conditions over the life-course. CSEP may be related to atherosclerosis later in life through its effects on the development of behaviors that track into adulthood and/or through its effects on biological processes occurring at critical points early in life which are related to the subsequent development of atherosclerosis. ASEP may be related to the presence of atherosclerosis through its effects on the presence of behavioral and psychosocial factors known to be involved in the development of atherosclerosis. Long-term exposure to NSEP may be related to the

development of atherosclerosis through the effects of social and physical environments on behaviors and psychosocial processes related to atherosclerosis [1, 22-23].

Based on prior work [24-26] CSEP was characterized based on father's or primary caretaker's education, as reported by study participants on a self-administered questionnaire as part of one of the MESA examinations. Participants were asked to choose their father's, or primary caretaker's, level of education from six categories: no schooling, some schooling but did not complete high school, high school degree, some college but no college degree, college degree, or graduate/professional degree. The father or primary caretaker's education was selected because it was the variable most likely to reflect CSEP for the age cohorts included in MESA.

Several measures of adult SEP were available. These indicators measure different dimensions of the construct of adult SEP. Similar to prior work [17] we summarized these indicators into an ASEP score by combining information on income, education and wealth (home ownership, car ownership, investments, ownership of other land or property). As part of the baseline examination, participants were asked to select their total gross family income in the past twelve months from thirteen categories. Income was collapsed into four categories (<$25,000, $25,000-39,999, $40,000-74,999, or >=$75,000) for these analyses. Participants also reported the highest education level completed. Education was categorized into four categories (completed High School or less, some college but no degree/technical school certificate, Associate or Bachelor's degree, or graduate/professional degree) for these analyses. The four wealth variables were: (1) whether the participant, or their family, had investments such as stocks, bonds, mutual funds, retirement investments, or other investments (yes/no), (2) whether the

participant owned their home (yes/no) (3) whether the participant owned a car (yes/no) (4) whether the participant owned land or another property that was not their primary residence (yes/no). A summary ASEP score was created by summing scores for income (0-3, from lowest to highest category) and education (0-3 from lowest to highest) and adding one point for each wealth indicator present. Thus, the range of values for ASEP was 0 to 10, with higher values indicating greater ASEP.

NSEP was characterized based on a residential history questionnaire linked to US census data. Census tracts were used as proxies for neighborhoods. Neighborhood census tract poverty was chosen as the key neighborhood variable investigated because it is often used in sociological work to characterize neighborhood conditions [27], it was measured in a standardized manner for all the US Censuses relevant to the period of study, and its definition has been modified over time by the US Census to account for changes in the cost of living. The Pearson's correlation between year 2000 neighborhood census tract poverty and other frequently used neighborhood SEP measures is -0.54 for average household income, -0.42 for percent adults completed college, and -0.48 for percent adults in managerial/professional occupations.

In order to assign a measure of 20-year neighborhood poverty exposure to each participant, we obtained 20 year residential history information from a questionnaire administered by interviewers as part of one of the study clinic visits. Participants were asked for their address (street number and name, city, county, state, and zip code) in January of 1980 and all subsequent addresses. If a subject had more than one address in the time period, the month and year of departure from each address was ascertained. Those unable to provide complete addresses were asked to supply the street name and the

closest cross-street. Each address was geocoded and assigned latitude and longitude coordinates. Of the total of 14,522 addresses reported, exact coordinates were obtained for 10,257, 166 were geocoded to the block group centroid, 36 were geocoded to the census tract centroid, 3076 were geocoded to the county centroid, and 987 could not be geocoded (centroid refers to the center of the geographic location). Latitude and longitude coordinates were used to assign 1980, 1990, and 2000 census tract codes to each address. Information on census tract poverty level (specifically percent of residents living below the poverty level) for each location was obtained from the Neighborhood Change Data Base (NCDB). The NCDB allows comparison across various census years by recalculating and normalizing past census years to 2000 census tract boundaries. Smaller block groups are used to determine the population-weighted proportion of a 1970, 1980, or 1990 tract that falls within the geographic boundaries of Year 2000 tracts. Estimates for prior census years normalized to Year 2000 boundaries are then obtained by applying appropriate weights (based on these proportions) to tract data for prior years [28].

Addresses reported by each MESA participant in the Residential History questionnaire were assigned the census value for the corresponding census year (1980, 1990, or 2000). For intercensal years we interpolated the value for that year based on the two closest censuses using simple linear interpolation. Using this approach we created a database that contains a measure of census tract poverty for each month between January 1980 and the date of the MESA baseline exam for each study participant. A continuous curve was fitted to each participant's data by connecting successive non-missing monthly values of percent poverty with straight lines. The area under this continuous line then

divided by the number of months from January 1980 to the date of the baseline exam to produce a measure of average exposure to neighborhood (census tract) poverty for the 20 year period for each person (mean=0.14, range=0.01-0.71). The reliability of the measure of NSEP was examined in a subset of 505 participants who repeated the residential history questionnaire twice (approximately one year apart). The Pearson's correlation coefficient between the NSEP exposure assessed using the two repeat questionnaires was 0.95.

Measurement of Subclinical Atherosclerosis

Common carotid IMT was the measure of subclinical atherosclerosis examined. The common carotid IMT measurements were made non-invasively with a high-resolution B-mode ultrasonography (Logia 700 ultrasound machine; General Electric Medical Systems). The reported IMT reflects the mean of all available maximum wall thicknesses across all scans, across both left and right sides, and across the near and far walls. Central reading of IMT was done at the Tufts-New England Medical Center (Boston, Massachusetts). Common carotid IMT, as assessed by B-mode ultrasound, is a non-invasive, relatively simple, inexpensive, precise, reproducible, and valid measure of early atherosclerotic changes in the carotid [29]. Carotid IMT has been shown to be associated with CHD risk factors, prevalent coronary heart disease, and subsequent CHD events [19-20]. The common carotid IMT was chosen because it has been examined more frequently than the internal carotid IMT, is easier to image than the internal carotid IMT, generally produces less amounts of missing data, and has been shown in some studies to have a stronger association with myocardial infarction [30-35]. Common

carotid IMT was not highly skewed and was treated as a continuous variable in these analyses.

Covariates

Age, gender, and race/ethnicity were obtained from the baseline examination interview. Race/ethnicity was classified as one of four categories: white non Hispanic, African American non Hispanic, Chinese, and Hispanic. Established cardiovascular risk factors were also examined in some analyses. Resting blood pressure was measured three times with a Dinamap PRO 100 automated oscillometric device (Critikon Inc., Tampa, Florida). The average of the final two blood pressure readings was used for this analysis. Hypertension was defined by the JNC VI (1997) criteria as: systolic blood pressure ≥ 140 mm Hg or diastolic blood pressure ≥ 90 mm Hg or use of anti-hypertensive medication. Cigarette smoking was based on self-report and classified into one of three categories: current, former, or never. Body mass index (BMI) was calculated using measured weight (kg)/height $(m)^2$. Diabetes Mellitus was defined by the 2003 ADA criteria of fasting glucose ≥ 126 mg/dL, use of insulin or oral hypoglycemic agents (OHGAs), or self-reported physician diagnosis. Impaired fasting glucose was defined by fasting glucose = 100-125 mg/dL if the person was not using insulin OHGAs and did not report a physician diagnosis of diabetes. High density lipoprotein cholesterol (HDL) and low density lipoprotein cholesterol (LDL) were assessed using standard methods [19]. Physical activity was assessed by two variables: leisure physical activity (total walking, dancing/sport, and conditioning MET-min/wk) and a sedentariness score (total MET-min/wk spent in sedentary activities such as TV watching, reading, knitting, sitting, doing

nothing, or non-work recreational computer) using a validated questionnaire [36]. Diet was summarized using two factors (fats and processed meats and whole grains and fruit) identified through factor analyses of dietary data collected using a standardized questionnaire [37-38].

Statistical Analysis

First, we examined socio-demographic characteristics and mean common carotid IMT for categories of each socioeconomic indicator using ANOVA (continuous variables) and the chi-square statistic (categorical variables). Multiple linear regression was used to assess the association of CSEP, ASEP, and NSEP with IMT. All SEP measures were modeled as categorical variables based on 3 groups: low, medium, or high SEP (CSEP: parental education less than high school, completed high school, or more than a high school education; ASEP: 0-4, 5-7, 8-10; NSEP: >17.2% poverty , 7.6%-17.2% poverty, or <7.6% poverty). Models were fitted to estimate the effect of these exposures separately and in pairs. The final model included all three types of measures (CSEP, ASEP, and NSEP). Because race/ethnicity may be partly confounding associations of SEP with IMT, models were fitted before and after adjustment for race/ethnicity. We chose to adjust for race/ethnicity only after including the SEP indicators because SEP may be one of the reasons for race/ethnic differences, and adjusting for race/ethnicity would preclude identification of SEP effects. Because of potential differences in associations of SEP with atherosclerosis by gender, all models were run separately for men and women. Stratification by site reduced the sample size too much so results were not reported.

In order to examine the potential role of CVD risk factors as mediators of any differences observed, final models were rerun after adjustment for traditional CVD risk factors [hypertension (yes/no), cigarette use (current, former, never), BMI (continuous), diabetes status (impaired fasting glucose, diabetes, normal), HDL (continuous), LDL (continuous), physical activity (continuous), fats and processed meats diet (continuous), and whole grains and fruit diet (continuous)]. We performed tests for trend by treating each SEP measure as an ordinal variable in regressions. The interaction of each of the three SEP measures with gender and race/ethnicity was investigated by including interaction terms in the regression equations and comparing models using F tests.

RESULTS

Of 6,814 participants at baseline, 5,871 (86%) completed the residential history questionnaire. Of these, 797 were excluded because one or more addresses could not be geocoded and 132 were excluded because poverty values were not available for one or more of the tracts in which they had lived since January of 1980, leaving at total of 4,942 participants with complete neighborhood poverty information. An additional 55 were excluded because carotid IMT information was not available, and 464 were excluded due to missing adult (206) or childhood (258) SEP measures, leaving 4,423 available for analysis. Compared to persons included in the analyses, persons excluded were significantly more likely to be younger, Chinese or Hispanic, and be in the lower income or educational categories, although differences were generally not large.

The mean age was 62.0 years, 48% were male, 45% White, 27% African American, 7% Chinese, and 21% Hispanic. Fifty-five percent of the sample were in the

lowest category of CSEP (father did not complete High School), 34% of the sample were in the lowest category of education (completed HS or less), 27% of the sample were in the lowest category of income (>$25,000), 74% owned their own home, 85% owned at least one car, 68% had investments, and 33% owned land or business property that was not their primary residence. Forty-six percent of participants reported only one address for the whole period, 26% reported two addresses, 13% three addresses, 7% four addresses, and 7% five or more addresses. Participant's with a higher number of addresses were significantly more likely to be younger, White, have had a higher CSEP, a higher ASEP, and were less likely to have lived in poor neighborhoods over the past 20 years (data not shown). The mean NSEP was 15% (SD 11%), meaning that on average since 1980 participants lived in a census tract where 15% of residents were below the poverty level. Spearman's correlations between SEP measures were 0.34 for CSEP and ASEP; 0.15 for CSEP and NSEP; and 0.36 for ASEP and NSEP. The mean common carotid IMT was 896.2 micrometers (SD 194.4) in men and 846.5 micrometers (SD 180.2) in women.

Selected characteristics for categories of CSEP, ASEP, and NSEP are shown in Table 2.1. Participants in the higher CSEP tertiles were significantly more likely to be younger, White or Chinese, to have had a higher ASEP, have lived in less poor neighborhoods at the MESA baseline, and were less likely to have lived in poor neighborhoods over the past 20 years. Participants in the higher ASEP tertiles were significantly more likely to be younger, female, White, had a higher CSEP, have lived in less poor neighborhoods at the MESA baseline, and were less likely to have lived in poor neighborhoods over the past 20 years. All components of ASEP (income, education,

investments, home, car, and land ownership) followed the same pattern as the summary measure (data not shown). Participants in the highest NSEP category (i.e. who had lived in neighborhoods of low poverty for the past 20 years) were significantly more likely to be older, male, be White, had higher CSEP and ASEP and lived in less poor neighborhoods at the MESA baseline.

Tables 2.2 and 2.3 show the associations between common carotid IMT and the three measures of SEP for males and females, respectively. In men, both low CSEP and low ASEP were associated with greater carotid IMT after adjustment for age. NSEP was not consistently associated with IMT in age-adjusted models. Both CSEP and ASEP remained associated with IMT after adjustment for each other, although only CSEP was statistically significant at the 0.05 level. The further addition of NSEP and race/ethnicity did not substantially change the associations between CSEP or ASEP and IMT, although the trend test was marginally statistically significant in the full model only for CSEP. Inclusion of CVD risk factors attenuated the CSEP- IMT associations. Adjustment for site did not substantially modify results.

In women, low CSEP, low ASEP, and greater exposure to neighborhood poverty were all associated with greater IMT after adjustment for age. The association of NSEP with IMT differed significantly in men and in women (P for heterogeneity =0.0045). Associations between CSEP and ASEP were reduced, but remained significant, when both were included in the same model. Further inclusion of NSEP reduced the association between ASEP and IMT slightly, rendering it only marginally statistically significant. The association between NSEP and IMT was reduced, but remained significant, after adjustment for CSEP and ASEP. Adjustment for site did not

substantially modify results. All three SEP measures remained inversely associated with IMT after additional adjustment for race/ethnicity, although the trend test for ASEP was only marginally statistically significant. Inclusion of CVD risk factors attenuated all associations between SEP and IMT, except in the case of CSEP. Results for NSEP were similar when analysis was restricted to persons with all addresses as exact matches.

There was some evidence of heterogeneity of SEP associations with IMT by race/ethnicity. In men, the associations between ASEP and IMT differed significantly between race/ethnicity groups: among black men higher ASEP was associated with slightly greater, rather than lower, IMT (P for heterogeneity in blacks vs. whites: 0.04). No associations of ASEP with IMT were observed in Hispanics but this heterogeneity was not statistically significant. Mean differences (95% confidence limits) for highest vs. lowest adult SEP were -45.4 μm (-82.5, -8.3) in whites, -49.4 μm (-121.6, 22.8) in Chinese, 8.7 μm (-29.4, 46.7) in blacks and -3.6 μm (-64.2, 57.0) in Hispanics. Black women had a much stronger association between NSEP and IMT than white women (P for heterogeneity blacks vs. whites 0.01), while the association in Hispanic women was in the opposite direction (P for heterogeneity Hispanics vs. whites 0.01). Mean difference for highest vs. lowest NSEP were: -23.5 μm (-52.4, 5.5) in whites, 30.4 μm (-39.5, 100.2) in Chinese, -45.7 μm (-84.9, -6.4) in blacks and 25.3 μm (-21.2, 71.9) in Hispanics. Results did not differ when analyses were repeated using race-specific tertiles of neighborhood poverty. Nativity stratified results showed that the association of lower neighborhood poverty with higher IMT was observed in non- US born Hispanic women but not in US born Hispanic women (mean differences high vs. low NSEP 83.0 μm (4.8,

161.2) in non US born Hispanic women and : -6.7 μm (-70.5, 57.1) in US born Hispanic women).

DISCUSSION

We found clear evidence that carotid atherosclerosis (as assessed by IMT) is patterned by socioeconomic position over the lifecourse. Both CSEP and ASEP were inversely and independently associated with IMT in men. Higher childhood and higher adult SEP as well as higher NSEP (lower exposure to neighborhood poverty) were independently associated with greater IMT in women. Associations were somewhat reduced after adjustment for cardiovascular risk factors suggesting that these factors may play a mediating role. Differences in IMT between top and bottom categories (in approximate thirds) were in the order of 20 micrometers for males and 30 micrometers for females. Although these differences may appear small, they are equivalent to the effect of aging 3-4 years in our data. In addition, previous literature has shown differences of ~100 micrometers are associated with 2-5 fold increased risks of myocardial infarction or stroke over periods of 3-7 years [19].

In both men and women, low CSEP was related to greater IMT, even after adjustment for ASEP, NSEP, and race/ethnicity. Associations were slightly stronger in women and were only marginally statistically significant in men, but were generally of similar strength and in the expected direction in both genders. These results are consistent with the Newcastle thousand families Study [12], which also reported an association between low social class at birth (father's occupational status) and greater IMT, controlling for adult social class. One other study, the Cardiovascular Risk in

Young Finns Study [40] reported no association between CSEP (based on the parent with the higher occupation/education status) and IMT. However, the participants in that study were only 24-39 years old at the time of IMT assessment and may not have aged enough for there to be detectable differences. The link between childhood socioeconomic characteristics and IMT in adulthood, even after controlling for adult measures, suggests that the early childhood socioeconomic environment has a long-lasting effect on the development of atherosclerosis.

An important consideration in the investigation of childhood SEP effects is the presence of cohort effects. In our sample the percent of participants whose parents had not completed high school was higher for older than for younger participants. It is possible that the effects of parental education on outcomes differ from birth cohort to birth cohort due to differential meaning of a given level of parental education over time. Unfortunately because younger participants have less atherosclerosis generally we had limited power to reliably examine whether childhood SEP effects differed by birth cohort. These cohort effects need to be examined in studies with more appropriate datasets.

We also found that higher ASEP was associated with lower IMT in both men and women, even after controlling for CSEP, NSEP, and race/ethnicity. Although associations were of similar magnitude in both genders they were once again slightly stronger and only statistically significant in women. A number of prior studies combining men and women and adjusting for gender have also reported higher IMT in lower rather than in higher socioeconomic groups [9, 13]. In the only study to stratify on gender [14], higher educational status was reported to be associated with lower IMT in women, but

not men. However, at least one study restricted to men also reported inverse, graded differences between levels of education, income, and IMT [10]. Our study goes beyond prior work by including wealth in the ASEP measure and by showing that ASEP is associated with IMT independent of CSEP and NSEP.

Greater exposure to neighborhood poverty over a 20 year period was found to be associated with higher IMT in women, but not in men. This association was reduced but persisted after adjustment for CSEP, ASEP, and race/ethnicity. The only previous study to assess the relationship between cumulative neighborhood SEP and IMT [18] found that a lower cumulative neighborhood SEP was associated with a higher mean IMT in white women but not in white men, black men, or black women. The relationship in white women was not statistically significant after adjustment for individual-level SEP (a measure encompassing SEP in childhood, young adulthood, and older adulthood). The measure of neighborhood SEP used in that study was substantially different from that used in ours in that it combined a series of census variables on income, education, and occupation census variables at three specific points in the life-course (age 10, 30, and 45-65 years). In contrast, our measure of neighborhood poverty was a summary measure of exposure to neighborhood poverty over the 20 years prior to the MESA examination.

Previous literature relating socioeconomic position to CVD-related outcomes has sometimes found stronger gradients in CVD-related outcomes by SEP in women than in men [1, 41]. There is some evidence that diabetes mellitus, hypertension, smoking, hypercholesterolemia, and obesity may be more strongly associated with cardiovascular risk in women than in men [42-49]. These risk factors may all be influenced by SEP. Other explanations center around the socially constructed roles that men and women

adopt in their daily lives. Psychosocial factors like double loads of work and family, lack of social support, and being a primary care-giver are more common in women, may be cardiovascular risk factors, and are likely to be strongly patterned by SEP, thus contributing to the stronger SEP gradient in CVD risk observed in women [50-51].

Previous literature has also documented a stronger effect of neighborhood deprivation on the incidence and prevalence of CHD in women than men [3, 52-53]. It may be that exposure to neighborhood conditions is different for women than men. For instance, a study that assessed the amount of physical activity in adults in relation to the density of recreational resources within certain distances of their homes, found that women were more likely than men to exercise within one mile of their home, [54-55] suggesting that the density of recreational facilities in a neighborhood may be more important for a woman's cardiovascular health than a man's. Women may also perceive the local environment differently [56], hence influencing their utilization of health-hurting or health promoting services, facilities, and/or amenities. Men and women may also differ in their vulnerability to aspects of the local environment [56]. For example, one study found that a higher density of small grocery stores and closer proximity to chain supermarkets was only associated with higher BMI in women [57].

Our analyses also revealed interesting differences in associations by race/ethnicity. There was some suggestion that ASEP may be either not associated or even positively associated with SEP in black men. One explanation is that black men in the lowest category of ASEP may actually be at a decreased risk of developing atherosclerosis. The social patterning of CVD risk is not invariant and has changed over time [3, 58-59]. It is plausible that in US black men, higher ASEP is associated with

psychosocial or behavioral consequences that increase CVD risk. Alternatively, the absence of an ASEP gradient in black men could be due to selection bias. In the US both low income and black race are strong risk factors for death [3, 60]. Study participation rates may also be lower in black men, especially low income black men, than in other groups. Exclusion criteria such as a history of prevalent cardiovascular disease may have resulted in disproportionately more exclusions among low income black men than among low income white men. As a consequence of these differential exclusions, the sample of low income black men included in MESA may be 'healthy' survivors with consequently relatively good cardiovascular health profiles, compared to their middle or high SEP counterparts.

Neighborhood poverty also seemed to have a stronger effect in black women than white women and possibly the opposite effect in Hispanic women. Although identical categories for NSEP were used in all race/ethnic groups, the distributions of poverty within categories (especially the top and bottom categories) differed by race/ethnicity. In black women, the difference between the medians for the top and bottom categories was more extreme than in white women [0.21 vs. 0.15, respectively]. This could explain the stronger association of poverty with IMT observed in black women compared to white women. However, similar race/ethnic differences were observed when race/ethnic specific categories were used. It is plausible that these differences reflect either differential association of neighborhood poverty with cardiovascular risk –related area features by race, or greater vulnerability of Black women to neighborhood characteristics due to the absence of other buffering resources.

The unexpected association of lower neighborhood poverty with higher IMT observed in Hispanic women appeared to be restricted to non-US born women. It is plausible that the correlates of neighborhood poverty differ in immigrant compared to non-immigrant communities. For example, the high poverty neighborhoods that Hispanic (and especially non-US born Hispanic) women tend to live in may have other features that buffer or even counteract the adverse health effects of high neighborhood poverty resulting in very different associations of neighborhood poverty with health. Unfortunately the absence of information on other neighborhood features for the 20 year neighborhood measures made it impossible for us to investigate this hypothesis in our data. It is also possible that Hispanic women living in poorer neighborhoods are more likely to be recent immigrants and less acculturated than those in less poor neighborhoods, and less acculturation has been previously linked to less atherosclerosis [60]. In our data, non-US born Hispanic women who lived in less poor neighborhoods over the 20 years had greater odds of speaking English in the home, but neighborhood poverty was not associated with years in the US (data not shown).

Associations of SEP measures with IMT were reduced after adjustment for traditional CVD risk factors, suggesting that the effect of socioeconomic status is mediated at least in part through these biomedical and behavioral factors. The only exception was the effect of CSEP in women, which was virtually unchanged after risk factor adjustment. However, detailed examination of mediating mechanisms would require data and analyses different from those reported here.

A limitation of this study is that participants were selected at baseline to be clinically free of cardiovascular disease. It is plausible that people with a history of CVD

(who were excluded) have both lower SEP and higher IMT. Thus, restricting analyses to persons without CVD could have resulted in underestimates of the true association between the SEP measures and subclinical atherosclerosis. Men develop clinically apparent CHD at earlier ages than women. The differential exclusion of men could therefore have resulted in greater underestimates of the SEP-IMT association in men than in women. On the other hand, the investigation of associations of SEP with IMT in persons without clinical cardiovascular disease allows examination of whether SEP is related to the early stages of the development of atherosclerosis. Persons excluded due to missing data were more likely to be of low ASEP. If they also tended to have higher IMT, our results may underestimate associations of SEP with IMT. MESA was specifically designed to investigate the prevalence, correlates, and progression of subclinical CVD in men and women 45-84 years old, with particular emphasis on an ethnically diverse population, and hence may not be generalizable to the entire US population.

The retrospective collection of childhood and residential history information may have resulted in misclassification and could also have contributed to underestimates of SEP effects. Several of the measures we used have limitations. For example, father's education may be an imperfect proxy for childhood social environment. The neighborhood SEP measure was comprised of a single indicator and referred to census tracts which may not be the most relevant geographic units. The neighborhood SEP measure was also only able to capture 20 years of exposure to neighborhood poverty, with the 20 years capturing a different period of adulthood depending on the participant's age when they entered the study. Differing amounts of exposure time captured in the

ASEP and NSEP measures (20 years compared to a single point in time in adulthood), ASEP may have resulted in residual confounding of the NSEP-IMT associations.

An important strength our study is the use of multiple measures of SEP. We included both childhood and adult measures. We were able to capture adult SEP as a full composite measure encompassing not only traditional indicators of income and education, but also wealth. We also had information on 20 year exposure to neighborhood conditions as opposed to a measure for a single point in time. Another strength is the ability to examine variations in the effects of SEP by race/ethnicity. Our results contribute to the growing body of literature that shows that SEP at multiple points in the life-course, and at the individual and neighborhood level, contributes to the development of atherosclerosis. CVD prevention may benefit from interventions at multiple points over the lifecourse targeted at both individuals and areas.

Table 2.1. Sociodemographic Characteristics by Tertiles of Childhood, Adult, and Neighborhood SEP, the Multiethnic Study of Atherosclerosis 2000-2002.

	CHILDHOOD SEP				ADULT SEP				NEIGHBORHOOD SEP			
Variable	**LOW**	**MED**	**HIGH**	**p-value**	**LOW**	**MED**	**HIGH**	**p-value**	**LOW**	**MED**	**HIGH**	**p-value**
Mean Age (SD)	63.2 (9.8)	61.1 (9.8)	60.1 (10.2)	<0.001	64.5 (10.2)	61.1 (9.8)	60.1 (9.1)	<0.001	62.0 (10.3)	61.4 (9.9)	62.7 (9.7)	0.0014
Male, %	48	50	47	0.3073	40	50	57	<0.001	46	48	50	0.0596
Race/ethnicity, %				<0.001				<0.001				<0.001
White	33	52	64		25	52	61		17	47	68	
Black	30	12	6		28	29	25		42	28	13	
Chinese	6	6	11		7	5	9		5	7	9	
Hispanic	31	12	6		40	14	4		36	18	10	
Mean % poverty of census tract of residence at baseline (SD)	0.16 (0.12)	0.14 (0.11)	0.12 (0.09)	<0.001	0.20 (0.12)	0.13 (0.10)	0.11 (0.08)	<0.001	0.27 (0.10)	0.13 (0.06)	0.06 (0.03)	<0.001
Mean Common Carotid IMT (µm)												
Males	913.12 (195.55)	889.7 (191.05)	862.61 (191.05)	<0.001	929.30 (197.7)	887.2 (195.6)	875.8 (186.4)	<0.001	898.84 (182.9)	902.4 (191.6)	888.3 (206.3)	0.3484
Females	867.6 (185.5)	834.0 (172.4)	808.3 (165.6)	<0.001	879.6 (189.5)	837.7 (175.3)	799.5 (155.4)	<0.001	861.8 (187.5)	843.0 (174.0)	834.3 (176.8)	0.0091
Low CSEP [1], %	-	-	-		72	18	10	<0.001	64	21	15	<0.001
Low ASEP [2], %	48	34	18	<0.001	-	-	-	<0.001	59	27	14	<0.001
Low NSEP [3], %	38	30	21	<0.001	52	25	16	<0.001	-	-	-	<0.001

1 CSEP-Low: parental education less than high school, CSEP-Med: completed high school, CSEP-High: more than a high school education.

2 ASEP-Low: 0-4, ASEP-Med: 5-7, ASEP-High: 8-10

3 NSEP-Low: neighborhood poverty >17.2% poor in census tract, NSEP-Med: neighborhood poverty 8.6%-17.2% poor in census tract, NSEP-High: neighborhood poverty <8.6% poor in census tract.

† IMTs are in micrometers.

Table 2.2. Age-adjusted Mean differences (95% CIs) in Common Carotid IMT† associated with childhood SEP, adult SEP, and a 20-year cumulative exposure to neighborhood poverty, and Race/ethnicity: Males

Mean Difference:	CSEP only	ASEP only	Neighborhood poverty only	CSEP & ASEP	CSEP, ASEP, & Neighborhood poverty	CSEP, ASEP, Neighborhood poverty, & Race/ethnicity	All SEP, Race/ethnicity, & risk factors‡
CSEP[1]-Low	Reference			reference	reference	reference	reference
CSEP[1]-Med	-6.4 (-25.1, 12.2)			-3.5 (-22.4, 15.4)	-3.0 (-21.9, 15.9)	-5.0 (-24.2, 14.1)	-1.4 (-21.0, 18.3)
CSEP[1]-High	-26.0** (-44.9, -7.1)			-21.0** (-40.8, -1.3)	-20.8* (-40.6, -1.1)	-18.8* (-39.1, 1.6)	-12.0 (-32.9, 8.9)
ASEP[2]-Low		Reference		reference	reference	reference	reference
ASEP[2]-Med		-15.2 (-33.8, 3.4)		-13.1 (-31.9, 5.6)	-11.8 (-31.4, 7.9)	-17.8 (-37.8, 2.3)	-19.6 (-40.5, 1.4)
ASEP[2]-High		-24.4** (-43.6, -5.3)		-18.7* (-38.7, 1.3)	-16.3 (-37.6, 4.9)	-21.2 (-43.1, 0.7)	-15.5 (-38.5, 7.5)
NSEP[3]-Low			reference		reference	reference	Reference
NSEP[3]-Med			11.3 (-7.5, 30.1)		17.0 (-2.4, 36.3)	21.1 (1.5, 40.8)	10.8 (-9.6, 31.2)
NSEP[3]-High			-16.7* (-35.1, 1.8)		-7.2 (-27.2, 12.8)	2.0 (-19.2, 23.2)	-7.8 (-29.7, 14.1)
WHITE						reference	Reference
CHINESE						-34.4 (-63.7, -5.1)	14.9 (-17.5, 47.3)
BLACK						32.4 (12.1, 52.6)	24.0 (2.5, 45.4)
HISPANIC						-12.3 (-35.3, 10.7)	-12.6 (-35.9, 11.8)

1 CSEP-Low: parental education less than high school, CSEP-Med: completed high school, CSEP-High: more than a high school education.

2 ASEP-Low: 0-4, ASEP-Med: 5-7, ASEP-High: 8-10

3 NSEP-Low: neighborhood poverty >17.2% poor in census tract, NSEP-Med: neighborhood poverty 8.6%-17.2% poor in census tract, NSEP-High: neighborhood poverty <8.6% poor in census tract.

† IMTs are in micrometers.
‡Model 6 + CVD risk factors (hypertension, cigarette smoking, BMI, diabetes, HDL, LDL, physical activity, fats and meats diet, & fruit and whole grain diet).
* Test for trend significant at the 0.05 level ** and the 0.10 level *

Table 2.3. Age-adjusted Mean differences (95% CIs) in Common Carotid IMT† associated with childhood SEP, adult SEP, and a 20-year cumulative exposure to neighborhood poverty, and Race/ethnicity: Females

Mean Difference:	CSEP only	ASEP only	Neighborhood poverty only	CSEP & ASEP	CSEP, ASEP, & Neighborhood poverty	CSEP, ASEP, Neighborhood poverty, & Race/ethnicity	All SEP, Race/ethnicity, & risk factors‡
CSEP[1]-Low	Reference			reference	Reference	reference	Reference
CSEP[1]-Med	-15.7 (-32.3, 1.0)			-12.0 (-28.9, 4.8)	-11.7 (-28.5, 5.1)	-14.9 (-31.7, 2.0)	-12.7 (-29.8, 4.5)
CSEP[1]-High	-32.5** (-48.7, -16.3)			-25.0** (-42.0, -8.0)	-24.0** (-41.0, -7.0)	-21.1** (-38.3, -4.0)	-20.6** (-38.0, -3.2)
ASEP[2]-Low		Reference		reference	Reference	reference	Reference
ASEP[2]-Med		-13.1 (-28.3, 2.1)		-8.6 (-24.1, 6.8)	-2.9 (-18.8, 13.1)	-12.0 (-28.3, 4.2)	-3.1 (-19.9, 13.6)
ASEP[2]-High		-34.7** (-51.8, -17.5)		-26.3 ** (-44.3, -8.3)	-18.2* (-37.1, 0.6)	-29.4* (-48.8, -10.1)	-12.4 (-32.4, 7.6)
NSEP[3]-Low			reference		Reference	reference	Reference
NSEP[3]-Med			-16.3 (-32.3, -0.4)		-11.0 (-27.2, 5.4)	-7.1 (-23.8, 9.6)	0.5 (-16.5, 17.5)
NSEP[3]-High			-32.8** (-48.7, -17.0)		-24.5** (-41.4, -7.5)	-13.7** (-32.1, 4.7)	-8.0 (-26.7, 10.8)
WHITE						reference	Reference
CHINESE						-36.4 (-63.5, -9.2)	-14.9 (-44.2, 14.4)
BLACK						36.6 (19.6, 53.6)	11.7 (-6.4, 29.9)
HISPANIC						-19.1 (-39.3, 1.2)	-20.9 (-42.4, 0.5)

1 CSEP-Low: parental education less than high school, CSEP-Med: completed high school, CSEP-High: more than a high school education.

2 ASEP-Low: 0-4, ASEP-Med: 5-7, ASEP-High: 8-10

3 NSEP-Low: neighborhood poverty >17.2% poor in census tract, NSEP-Med: neighborhood poverty 8.6%-17.2% poor in census tract, NSEP-High: neighborhood poverty <8.6% poor in census tract.

† IMTs are in micrometers.
‡Model 6 + CVD risk factors (hypertension, cigarette smoking, BMI, diabetes, HDL, LDL, physical activity, fats and meats diet, & fruit and whole grain diet).
* Test for trend significant at the 0.05 level ** and the 0.10 level

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CHAPTER 3

TRAJECTORIES OF NEIGHBORHOOD POVERTY AND ASSOCIATIONS WITH SUBCLINICAL ATHEROSCLEROSIS & ASSOCIATED RISK FACTORS

INTRODUCTION

A number of studies have been published in the past 10 years that show a relationship between where a person lives and their risk of cardiovascular disease, independent of statistical controls for personal socioeconomic characteristics. Associations have been found between various neighborhood socioeconomic characteristics and coronary heart disease (CHD) prevalence, incidence, and mortality [1-6], subclinical CHD [7], and cardiovascular risk factors [3-4, 8-11].

Almost all of these studies are based on single point in time measurements of neighborhoods. Chronic diseases (including atherosclerosis and CHD) develop throughout the life course, so the use of a single point in time measure may result in misestimates of area effects. Recent work has begun to explore how exposures to neighborhood deprivation across the life-course could be related to subclinical atherosclerosis [12-13]. The authors previously researched the cumulative effects of neighborhood exposures over the life course by simply summing or averaging neighborhood exposures over a specified period of time. Other work has estimated the "independent" effects of exposures for three different time periods of the life course. These approaches do not distinguish the effects of differing patterns of neighborhood

mobility, for example stable, upward, or downward. Descriptions of what patterns of neighborhood deprivation people actually experience in the population and how these patterns translate into cardiovascular health risks are lacking in the literature.

The purpose of this study was to use latent trajectory class modeling to determine patterns of neighborhood poverty over a 20-year period in mid to late adulthood and then to examine how these patterns were related to the amount and presence of subclinical atherosclerosis, and associated risk factors, in mid to late adulthood. We hypothesized that (1) there would be significantly different latent trajectory classes of neighborhood poverty over time, (2) that groups with higher stable neighborhood census tract poverty over time would have higher amounts of subclinical atherosclerosis and poorer cardiovascular risk profiles than groups with lower stable census tract poverty over time, (3) that groups with increased neighborhood census tract poverty over time [downwardly mobile] would have higher amounts of subclinical atherosclerosis and poorer cardiovascular risk profiles than outcomes expected in persons with equivalent stable cumulative poverty, and (4) that groups with decreased neighborhood census tract poverty over time [upwardly mobile] would be associated with decreased amounts of subclinical atherosclerosis and better cardiovascular risk profiles than outcomes expected in persons with equivalent stable cumulative poverty.

METHODS

Study Population

The data consists of 4,942 men and women recruited and assessed by the Multi-Ethnic Study of Atherosclerosis (MESA) to investigate the prevalence, correlates, and

progression of subclinical Cardiovascular Disease (CVD). Participants were 45-84 years old, from six US Communities (Baltimore, MD; Chicago, IL; Forsyth County, NC; Los Angeles County, CA; northern Manhattan, NY; and St. Paul, MN), clinically free of cardiovascular disease at baseline, and sampled to be ethnically diverse (approximately 38% white, 28% African American, 23% Hispanic, and 11% Chinese). The methods used for sampling and study design have been reported elsewhere [14].

The original sample included 6,814 men and women, of which 5,871 completed a residential history questionnaire. Of these, 929 (16%) of the participants had to be excluded because (a) one or more addresses could not be geocoded (n=797) or (b) poverty values were not available for one or more of the tracts in which they had lived since January of 1980 (n=132), leaving 4,942 available for analysis.

Assessment of Neighborhoods

During one of their study clinic visits, participants were asked by trained interviewers to complete a 20 year residential history questionnaire (January 1980-data of visit). Each address was geocoded and assigned latitude and longitude coordinates, which were then used to assign 1980, 1990, and 2000 census tract codes to each address. Information on census tract poverty level (specifically percent of residents living below the poverty level) for each location was obtained from the Neighborhood Change Data Base (NCDB), a database that allows comparison across various census years by recalculating and normalizing past census years to 2000 census tract boundaries [15]. Neighborhood census tract poverty was chosen as the key neighborhood variable investigated because it is often used in sociological work to characterize neighborhood

conditions [16], it was measured in a standardized manner for all the US Censuses relevant to the period of study, and its definition is modified over time by the US Census to account for changes in the cost of living.

Addresses reported by each MESA participant in the residential history questionnaire were then assigned the census value for the corresponding census year (1980, 1990, or 2000). For intercensal years we interpolated the value for that year based on the two closest censuses using simple linear interpolation. Using this approach we created a database that contains a measure of census tract poverty for each month between January 1980 and the date of the MESA baseline exam for each study participant.

Outcomes Variables

The common carotid intima medial thickness (CIMT) was the measure of subclinical atherosclerosis examined. CIMT measurements were made non-invasively with a high-resolution B-mode ultrasonography (Logia 700 ultrasound machine; General Electric Medical Systems). The reported IMT reflects the mean of all available maximum wall thicknesses across all scans, across both left and right sides, and across the near and far walls. Central reading of IMT was done at the Tufts-New England Medical Center (Boston, Massachusetts) [14]. IMT, as assessed by B-mode ultrasound, is a non-invasive, relatively simple, inexpensive, precise, reproducible, and valid measure of early atherosclerotic changes in the carotid artery. IMT has been shown to be associated with CHD risk factors, with prevalent coronary heart disease, and with subsequent CHD events [17-18].

The cardiovascular risk factors examined were body mass index (BMI), low density lipoprotein cholesterol (LDL), high density lipoprotein cholesterol (HDL), hypertension, diabetes, and current smoking. BMI was calculated using weight (kg)/height (m) ^{2}. LDL and HDL were assessed using standard methods. Resting blood pressure was measured three times with a Dinamap PRO 100 automated oscillometric device (Critikon Inc., Tampa, Florida). The average of the final two blood pressure readings was used for this analysis. Hypertension was defined by the JNC VI (1997) criteria of self-reported treatment of hypertension or a systolic blood pressure ≥ 140 mm Hg or a diastolic blood pressure ≥ 90 mm Hg. Diabetes Mellitus was defined by the 2003 ADA criteria of fasting glucose ≥ 126 mg/dL, use of insulin or oral hypoglycemic agents (OHGAs), or self-reported physician diagnosis. Cigarette smoking was based on self-report and classified into one of three categories: current, former, or never [14].

Covariates

Age, gender, adult socioeconomic position (ASEP) and race/ethnicity were obtained from the baseline examination interview. ASEP was created by combining information on income, education and wealth. Participants were asked to select their total gross family income in the past twelve months from thirteen categories (collapsed into four categories for this analysis: <$25,000, $25,000-39,999, $40,000-74,999, or >=$75,000), report the highest education level completed (also collapsed into four categories: completed High School or less, some college but no degree/technical school certificate, Associate or Bachelor's degree, or graduate/professional degree), and a series of wealth questions: (1) whether the participant, or their family, had investments such as

stocks, bonds, mutual funds, retirement investments, or other investments (yes/no), (2) whether the participant owned their home (yes/no) (3) whether the participant owned a car (yes/no) (4) whether the participant owned land or another property that was not their primary residence (yes/no). A summary ASEP score was created by summing scores for income (0-3, from lowest to highest category) and education (0-3, from lowest to highest) and adding one point for each wealth indicator present. Thus, the range of values for ASEP was 0 to 10, with higher values indicating greater ASEP. Race/ethnicity was classified as one of four categories: white non Hispanic, African American non Hispanic, Chinese, or Hispanic.

Statistical analysis

Trajectory classes were identified by using hierarchical and latent growth curve modeling to assign individuals to trajectory classes based on their pattern of repeated measurements of annual average neighborhood poverty for each year between 1980 and 1999 [19]. A SAS procedure (SAS PROC TRAJ) [20] was used in the analysis. The data trajectory for each subject consisted of repeated measurements of neighborhood census tract poverty over T time periods, $Y_i = (Y_{i1,}\ldots,Y_{iT})$ that were independent given the group C_i. The trajectory was modeled as:

$$\mu_{ijk} = ß_{0k} + YEAR_{ij}ß_{1k} + YEAR^2_{ij}ß_{2k} + YEAR^3_{ij}ß_{3k},$$

Where μ_{ijk} represents the ith response of person j in group k, $ß_{0jk}$ is the estimated mean neighborhood poverty value for group k, where YEAR ij =0 (Jan. 1980) and $YEAR_{ij}$

denotes the number of years since January of 1980 at subject j's time i. Up to a third-order polynomial in year was allowed in order to flexibly model the effects of year. K indicates group membership. K numbers of groups were assigned, so that the likelihood of observing the data trajectory for subject j, given he belonged to group k was observed, using a censored normal model.

Model parameters are estimated through a quasi-Newton procedure that searches for the polynomial that will give the least difference in each person's expected and observed neighborhood poverty values at each time point, given group membership. Models were run for two through eight trajectory groups. The number of groups was selected based on which model as a whole had the best overall Bayesian Information Criteria (BIC) value**.** Group membership was assigned based on which K group a person had the highest likelihood of observing the data trajectory, given that they belonged to group k.

Socio-demographic characteristics and mean IMT for the chosen categories of neighborhood poverty trajectory group were compared using ANOVA (continuous variables) and the chi-square statistic (categorical variables). For hypothesis 2 (higher stable neighborhood poverty = poorer cardiovascular risk), linear regression was used to assess mean differences in CIMT, BMI, LDL, and HDL and a generalized linear model with a binomial distribution (binomial or relative risk regression) [22] was used to estimates the relative prevalence of hypertension, diabetes, and current smoking for each trajectory group versus the low stable group. Because race/ethnicity and ASEP may be partly confounding associations of neighborhood poverty with CIMT and, associated risk factors, models were fit before and after adjustment for ASEP and race/ethnicity. For

hypothesis 3 and 4 (increased or decreased census tract poverty over time = cardiovascular risk higher or lower, respectively, than outcomes expected in persons with equivalent stable cumulative poverty), the outcomes of the upwardly or downwardly mobile groups identified in hypothesis one will be compared to all other trajectory groups to see if they fall between those trajectory groups with equivalent stable cumulative poverty. Because of potential differences in associations of SEP with atherosclerosis by gender, all models for all hypotheses were run separately for men and women.

RESULTS

The mean age for the entire sample was 62.4 years, 48% were male, 43% White, 30% African American, 7% Chinese, and 20% Hispanic, the mean BMI was 28.7 (SD 5.4), mean LDL was 117.3 (31.3), mean HDL was 51.2 (14.8), 46% of the sample had hypertension, 14% had diabetes, and 13% were current smokers. The fit of the trajectory class models, as assessed by the BIC value improved, from two classes (BIC=110,809) through six classes (148,513), worsening with seven classes (143,758). The model with eight classes was a better fit (151,448), but zero individuals were assigned to two of the classes, suggesting that the six class model was the best fit for the data. Forty-four percent of the sample (2168) were in the low stable neighborhood poverty group, 23% were in the low-medium stable neighborhood poverty group (n=1116), 16% were in the medium stable neighborhood poverty group (n=806), 4% were in the Medium-high to low neighborhood poverty group (n=204), 9% were in the high stable neighborhood poverty group (n=470), and 4% were in the high-low stable neighborhood poverty group (n=178) (Figure 3.1). The average probability for group membership significantly

differed by neighborhood poverty group but the average probability only ranged from 0.977-0.996 (SD 0.032-0.081).

Selected characteristics for the six neighborhood trajectory classes are shown in Table 3.1. Participants in the low stable group were significantly more likely to be male, White, and be born in the US. For most risk factors, participants in the low stable group had the most favorable risk factor profiles of all of the groups: they had the lowest mean BMI and the lowest prevalence of hypertension, diabetes, and current smoking. Moving from the lowest stable group to the highest stable group, the groups get younger, more female, less White or Chinese, more Black and Hispanic (with a drop in Hispanics and an increase in blacks in the highest poverty group), less people are born in the US, the mean BMI is higher and the prevalence of hypertension, diabetes, and current smoking become greater. The percent of persons who moved over the 20 years decreases monotonically from a high of 56% in the low stable poverty group to a low of 42% in the high stable poverty group. Only one group with a changing (i.e. not stable) neighborhood poverty trajectory was identified. This group began the period at a medium-high neighborhood poverty level and experienced a decline in neighborhood poverty over time. The medium-high to low group is the youngest, least male, most Chinese group with the highest mean LDL, lowest mean HDL, lowest mean CIMT, and has the highest prevalence of current smokers of all of the trajectory groups. Virtually all (96%) of persons in this group moved at some point over the 20 year period with the average number of moves decreasing as poverty increases, but the highest mean number of addresses in the medium-high to low group (3.1 moves).

Figures 3.2-3.8 show the mean differences in CIMT, BMI, LDL and HDL and relative prevalences of hypertension, diabetes, and current smoking for each neighborhood trajectory group compared to the low stable group, by gender. The darker lines indicate age-adjusted estimates and the lighter lines indicate age-, race-, and –adult SEP adjusted estimates. Among women there was clear evidence that after age adjustment groups with higher stable neighborhood census tract poverty over time had higher CIMT, BMI, diabetes, hypertension, and generally lower mean HDLs than those with low stable poverty (see four estimates on the left of each figure). For men, this was only true, and with less clear patterns, for a higher prevalence of diabetes. Adjustment for race and adult SEP either reduced or completely eliminated most of these associations, but patterns were still apparent, although not always statistically significant, for the association of higher neighborhood poverty over the 20 years with higher mean BMIs, higher prevalence of diabetes, and higher prevalence of hypertension in women. Other patterns of note: (1) For men and women, stable poverty trajectory groups above the low stable group had a higher prevalence of current smoking, but a dose-response relationship was not apparent, and (2) for men, higher stable poverty was associated with lower mean LDLs, but confidence intervals were very wide. Adjustments for race and ASEP slightly reduced associations with current smoking but did not alter the LDL association.

As previously noted, only one upwardly mobile group, the medium-high to low group, and no downwardly mobile groups were identified through the trajectory analysis (Figure 4.1). In 1980, the medium-high to low group starts at the same poverty level as the stable medium-high group (~30%) but the mean neighborhood poverty exposure for

the group drops over time reaching about 10% in 2000 (see figure 3.1). If the accumulation model holds, and based on the mean exposure to neighborhood poverty over the 20 years (18.5%), the risk factor outcomes for the upwardly mobile group should fall approximately in between the low-medium and medium stable groups. Table 3.2 shows the mean differences or relative prevalences for CIMT and the CVD risk factors comparing the medium-high to low neighborhood trajectory group to each of the other groups (95% CI) , by gender. The medium-high to low group does follow this general pattern for diabetes in men and for CIMT, BMI, hypertension, and diabetes in women. For example, in women, CIMT in the medium-high to low neighborhood trajectory group is lower in a dose-response fashion than the high stable, medium-high stable, and medium stable groups but higher than the low-medium and low stable groups, although none of the differences are statistically significant. There are a few exceptions. For women, the medium-high to low group had a lower mean HDL and higher mean LDL than all of the other trajectory groups, with only three of the five HDL comparisons, and none of the LDL comparisons statistically significant. For men, CIMT's were lower and the prevalence of hypertension higher than any of the other trajectory groups, although these differences were not statistically significant.

DISCUSSION

We found clear evidence of significantly different subpopulations of neighborhood trajectory groups, with most groups having the same neighborhood poverty over the entire time period with variation in degrees of neighborhood poverty. Generally, and mostly in women, higher cumulative neighborhood poverty was associated with

worse cardiovascular outcomes with only BMI, diabetes, and hypertension associations in women apparent, but not always statistically significant, after adjustment for ASEP and race/ethnicity. The ability to look at trajectories was limited by the lack of changes in neighborhood poverty over the study period, but in general the mobility group had outcomes comparable to stable groups with similar cumulative exposures. Some evidence did exist for effects of upward mobility for CIMT and hypertension in men and for LDL and HDL in women.

There have been no previously reported findings of what trajectories of neighborhood poverty actually exist in the population. We found very little evidence of changes in exposure to neighborhood poverty over the twenty years, with most of the variability occurring in the differences in poverty between the stable trajectory groups. This does not mean that the population was non-mobile (almost 50% of the stable groups had moved at least once) or that when people moved, they necessarily moved to a neighborhood with similar neighborhood poverty (Pearson's correlations of neighborhood poverty between each pre- and post- move neighborhood, 0.36), but almost half the population (~47%) did not move over the entire twenty years, creating an average monthly stay at a residence of 240 months (which is the entire study period).

Higher cumulative neighborhood poverty was associated with worse cardiovascular outcomes mostly in women but not for every outcome. Adjustment for age, race, and adult SEP reduced all associations, but patterns were still apparent for BMI, diabetes, and hypertension in women. This is consistent with prior analyses of the same cohort and other cohorts showing that cumulative neighborhood SEP is associated with higher mean CIMT in women (Carson et al, white women only), with patterns still

apparent but non-significant after adjustment for individual SEP and race/ethnicity [12-13].

No previous findings have been reported for cumulative neighborhood poverty and cardiovascular risk outcomes. Most risk factors followed the same general pattern of the subclinical measure with, after adjustment for age, higher poverty being associated with worse BMI, diabetes, and hypertension outcomes in woman and little or no association with BMI, LDL, HDL, and hypertension in men. This supports the idea that neighborhoods are acting on known risk factors for subclinical atherosclerosis. For men, CIMT, BMI, and LDL, either had no or a slightly negative pattern with higher stable neighborhood poverty, but interpretation is limited by the width of the confidence intervals. The non dose-response association of higher stable neighborhood poverty with current smoking indicates suggesting a threshold effect.

One group, the medium-high to low group, displayed a remarkable degree of upward residential mobility, going from 30% to 10% neighborhood poverty, but it only included approximately 4% of the population. Pearson's correlates between census years for all census tracts in the Neighborhood Change Database are very high (1980-2000: 0.73, 1980-1990: 0.76, 1990-2000: 0.85), indicating that changes in poverty are probably attributable to mobility and not to changes in the neighborhood. The percentage of the US population aged one year and older that has moved in the past year averaged between 15-20% between 1980 and 2000, but also varied with age, with 20-29 year olds moving the most and mobility decreasing with age and over time in all age groups [21]. Our population is an older cohort that would have been between 25-64 years old in 1980 with

an average age of 42.4 years. It could be that a vast majority of our population had already done any moving that they were going to do in their life-times.

We also found some evidence that neighborhood poverty trajectories may have an effect on some cardiovascular outcomes in some genders. For most of the outcomes, the high-low group had results similar to what would be expected in a pure accumulation model. Membership in the high-low group produced CIMTs in men and HDLs in women that were better and hypertension in men and LDLs in women that were worse than and of the stable groups. The only relationship where this effect was statistically different was for HDLs in women. Mobility itself (whether a person had moved or not during the twenty years) was not statistically associated with any of the outcomes after adjustment for age, cumulative neighborhood poverty, and race/ethnicity (data not shown).

The potential additional effect of upward or downward neighborhood poverty on cardiovascular risk has not been discussed in the literature. Neighborhood conditions could have unequal effects on subclinical disease or a risk factor at different points in time. If the effect was most prominent for past exposures, and exposure to neighborhood deprivation changed over time, neighborhood poverty earlier in life may be more predictive of current cardiovascular health than later in life. It is also possible that where a person has lived recently is more relevant to current cardiovascular risk factors than past exposures. Or conversely, a person who grew up in a neighborhood with cardiovascular promoting qualities/amenities that moved to a more deprived area, may not be able to adapt to their new environment, or may be more vulnerable to negative cardiovascular exposures, and consequently would have worse cardiovascular health than if they had lived in the deprived area the entire time.

The ability to examine these differences was effected by small numbers in the high-low group only comprising 4% of the population (n=204) and non-mobility of the population over-all. The retrospective collection of the residential history information may have also resulted in misclassification, leading to an underestimation of effects. The selection of participants at baseline to be clinically free of cardiovascular disease could have also created underestimates if people with a history of CVD (who were excluded) had both higher levels of neighborhood poverty and higher levels of the cardiovascular outcomes. Men develop clinically apparent CVD at earlier ages than women, implying that reduced or null effects in men may be the result of differential exclusion. The exposure to neighborhood was measured with only a single indicator, neighborhood census tract poverty, and captured only 20 years of exposure in late adulthood. Census tracts may not be the most relevant geographic unit, 20 years does not capture the full extent of residential exposure or mobility, and the 20 years may be capturing a different period in adulthood depending on the participant's age when they entered the study.

An important strength of our study is the application of a novel statistical technique to identify and examine residential mobility patterns. Twenty years of residential information was synthesized in such a way to allow simultaneous examination of the magnitude and change in neighborhood poverty over time, but to also identify distinctive clusters of trajectories within the population. A broad range of outcomes that included a subclinical measure and risk factors for that measure, allowed multiple examination of the processes leading to clinically apparent CVD. The multi-ethnic and multi-geographic sampling of the cohort makes the results more generalizable. Our results add to the growing body of literature that shows that the accumulation of

neighborhood exposures over the life course contributes to the development of atherosclerosis.

Figure 3.1: Level and change of neighborhood poverty over 20 years, and percent of participants in each neighborhood poverty trajectory group: six groups

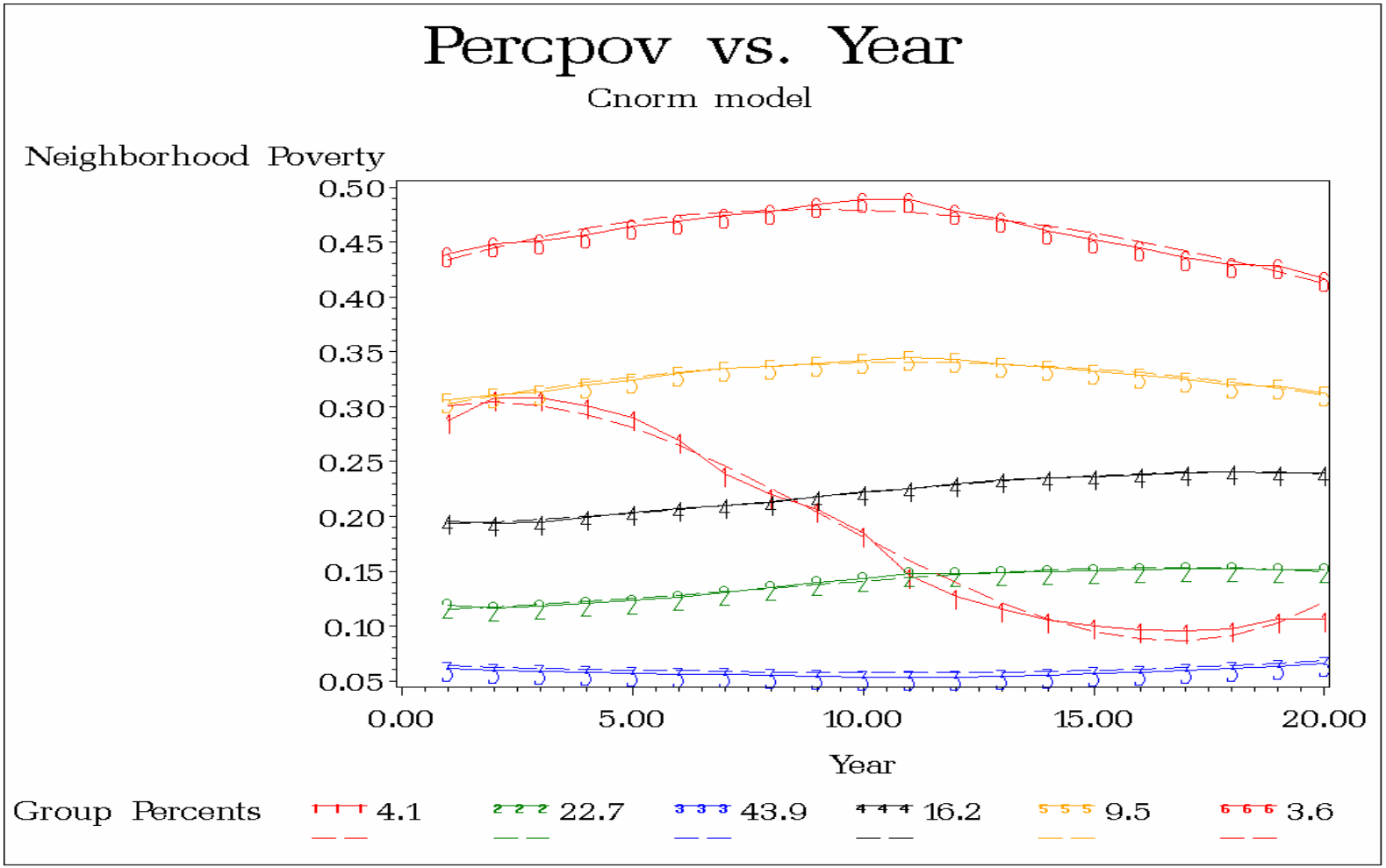


Table 3.1. Sociodemographic Characteristics by neighborhood trajectory class, the Multiethnic Study of Atherosclerosis 2000-2002.

	LOW† stable (n=2168)	L-Med† Stable (n=1116)	Med† stable (n=806)	M-High† Stable (n=470)	High† Stable (n=178)	High-Low† (n=204)
Mean age, SD**	62.8 (9.7)	61.9 (10.2)	63.5 (10.3)	62.4 (10.4)	60.1 (9.0)	59.1 (10.3)
Male, %**	50	47	46	45	42	41
Moved in 20 yrs, %**	56	52	46	40	37	96
Mean # of Moves, SD**	2.1 (1.7)	2.1 (1.5)	1.9 (1.4)	1.7 (1.2)	1.6 (1.0)	3.1 (1.5)
Race, %**						
White	63	44	21	7	5	24
Chinese	8	5	6	3	0	12
Black	19	31	43	47	62	39
Hispanic	10	19	31	43	33	25
Born in US, %**	85	82	71	55	57	69
Mean BMI (SD)**	28.1 (5.2)	28.5 (5.3)	29.4 (5.7)	29.6 (6.0)	30.0 (5.5)	29.2 (5.5)
Mean LDL (SD)	117.4 (29.9)	117.1 (32.0)	117.7 (33.5)	116.5 (31.7)	116.2 (31.0)	118.7 (33.1)
Mean HDL (SD)	51.5 (15.1)	51.6 (15.0)	50.2 (14.9)	51.1 (13.5)	51.1 (15.3)	49.4 (13.1)
Mean CIMT (SD)	872.5 (195.2)	870.9 (189.6)	893.2 (183.6)	885.7 (199.5)	879.2 (193.5)	833.2 (166.7)
Hypertension, %**	44	46	45	52	54	53
Diabetes, %**	11	15	16	18	28	22
Current Cigarette smoker, %**	10	14	15	16	19	24

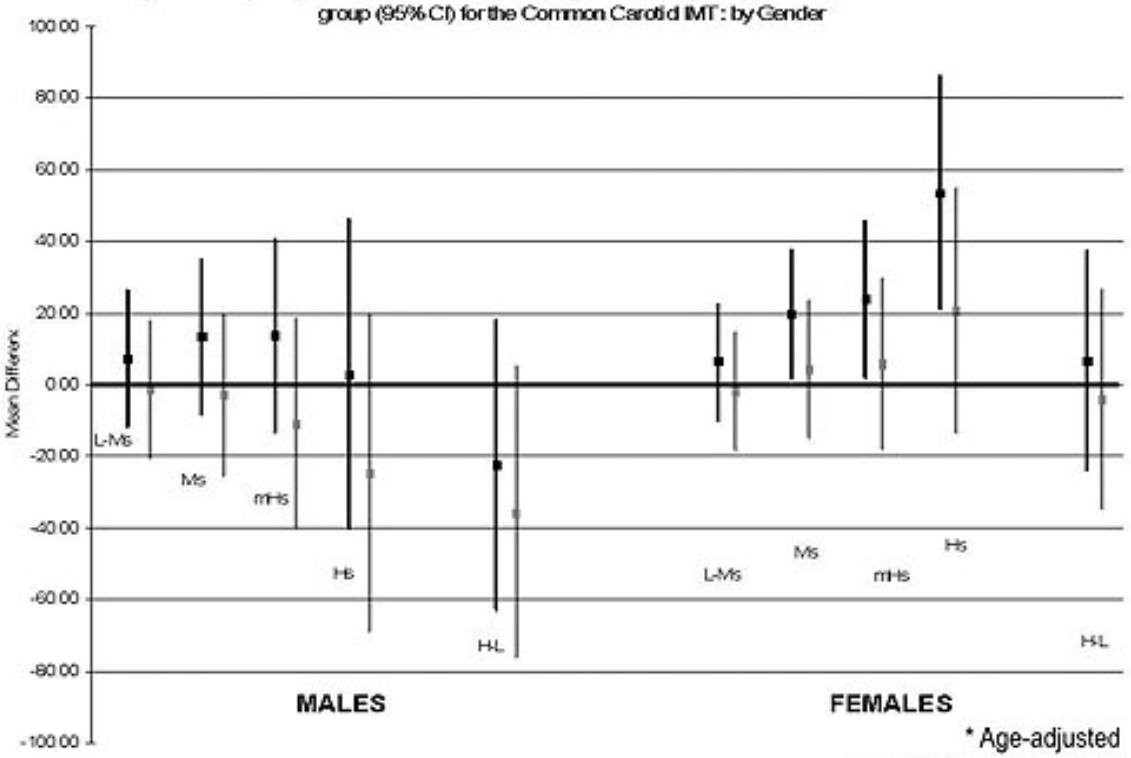
† Low stable = ~ 6% neighborhood poverty, L-med stable ~ 12%, Med stable ~ 20%, M-High stable ~ 31%, High stable ~ 45%, High-Low ~30-10%

Table 3.2: Mean differences in the CIMT, BMI, LDL, & HDL and relative prevalences of Hypertension, Diabetes, and Current Smoking for the medium-high to low neighborhood trajectory group compared to each separate trajectory group individually (95% CI), by gender.

	CIMT	BMI	LDL	HDL	HYP	DIAB	CURSMK
Males							
H-H	-25.4 (-82.2, 31.5)	-0.9 (-2.2, 0.5)	3.0 (-6.7, 12.8)	1.6 (-2.0, 5.1)	1.5 (0.8, 2.9)	0.5 (0.2, 1.0)	0.7 (0.3, 1.5)
Mh-mH	-36.1 (-82.2, 9.9)	0.2 (-0.9, 1.3)	0.5 (-7.4, 8.4)	-1.6 (-4.5, 1.3)	1.2 (0.7, 2.0)	1.1 (0.5, 2.1)	0.5 (0.2, 1.0)
Med stable	-35.6 (-78.7, 7.4)	-0.6 (-1.6, 0.4)	-2.0 (-9.4, 5.3)	0.5 (-2.2, 3.2)	1.3 (0.8, 2.1)	0.8 (0.4, 1.5)	0.8 (0.4, 1.5)
L-M stable	-29.7 (-71.5, 12.1)	-0.4 (-1.4, 0.6)	-2.1 (-9.3, 6.1)	-0.9 (-3.5, 1.7)	1.7 (1.1, 2.8) *	1.0 (0.5, 1.9)	0.7 (0.4, 1.4)
L-L	-22.3 (-62.7, 18.0)	-0.4 (-1.4, 0.5)	-1.4 (-8.3, 5.5)	0.0 (-2.6, 2.5)	1.5 (1.0, 2.4) *	1.1 (0.6, 2.1)	1.2 (0.6, 2.3)
Females							
H-H	-46.7 (-89.0, -4.4) *	-0.8 (-2.4, 0.8)	1.6 (-6.7, 9.9)	-3.8 (-7.8, 0.1)	0.4 (0.3, 0.8) *	0.4 (0.2, 0.8) *	0.9 (0.5, 1.8)
mH-mH	-17.1 (-52.1, 17.8)	-1.2 (-2.5, 0.1)	2.2 (-4.6, 9.1)	-1.8 (-5.0, 1.5)	0.8 (0.5, 1.3)	0.6 (0.3, 1.2)	1.5 (0.8, 2.8)
Med stable	-12.7 (-45.5, 20.0)	-0.4 (-1.6, 0.9)	2.1 (-4.3, 8.6)	-1.8 (-4.9, 1.3)	0.9 (0.6, 1.4)	1.0 (0.5, 1.8)	1.1 (0.6, 1.9)
L-M stable	0.8 (-31.0, 32.5)	1.2 (-0.1, 2.4)	3.7 (-2.6, 9.9)	-3.7 (-6.7, -0.7) *	1.1 (0.7, 1.6)	1.2 (0.7, 2.2)	1.4 (0.8, 2.4)
L-L	6.9 (-23.6, 37.5)	2.0 (0.8, 3.1) *	2.1 (-3.9, 8.1)	-5.0 (-7.8, -2.1) *	1.3 (0.8, 1.9)	2.3 (1.3, 4.1) *	2.0 (1.2, 3.4) *

* Significant at the 0.05 level *

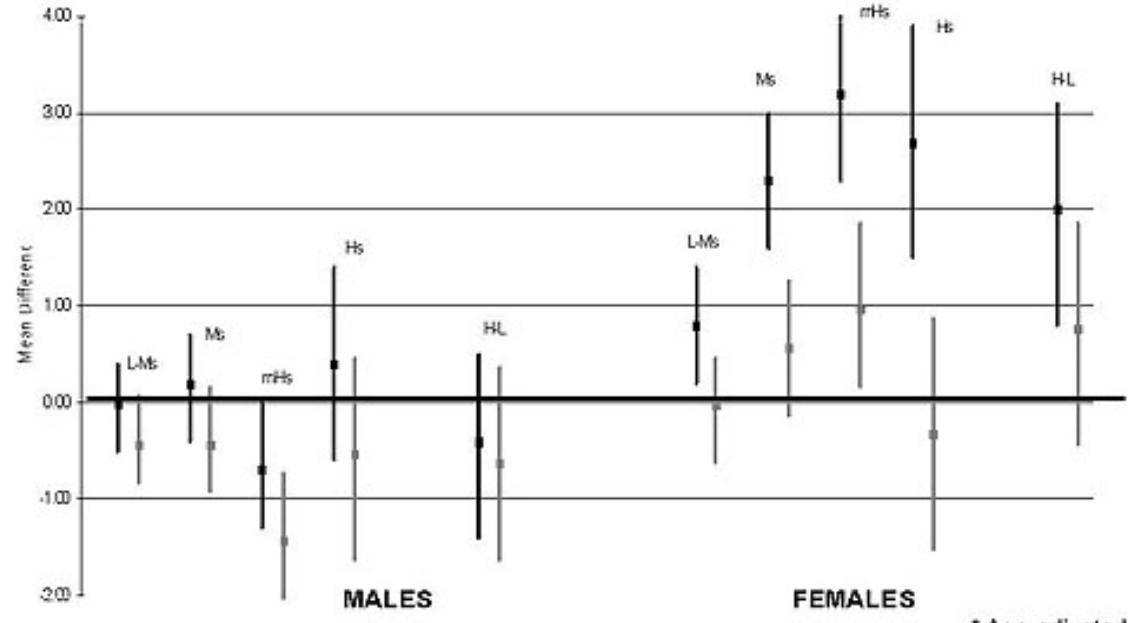
Figure 3.2. Age-adjusted mean differences of neighborhood trajectory classes versus the low stable group (95% CI) for the Common Carotid IMT: by Gender



* Age-adjusted

* Age- ASEP- and Race-adjusted

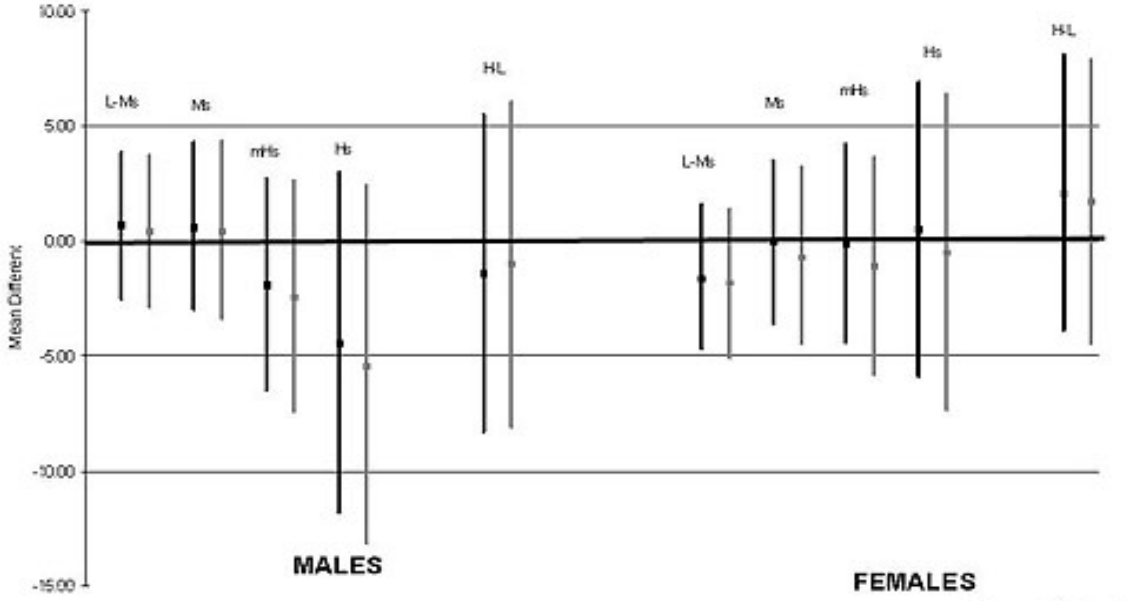
Figure 3.3. Mean differences of neighborhood trajectory classes versus the low stable group (95% CI) for Body Mass Index: by Gender



* Age-adjusted

* Age- ASEP- and Race-adjusted

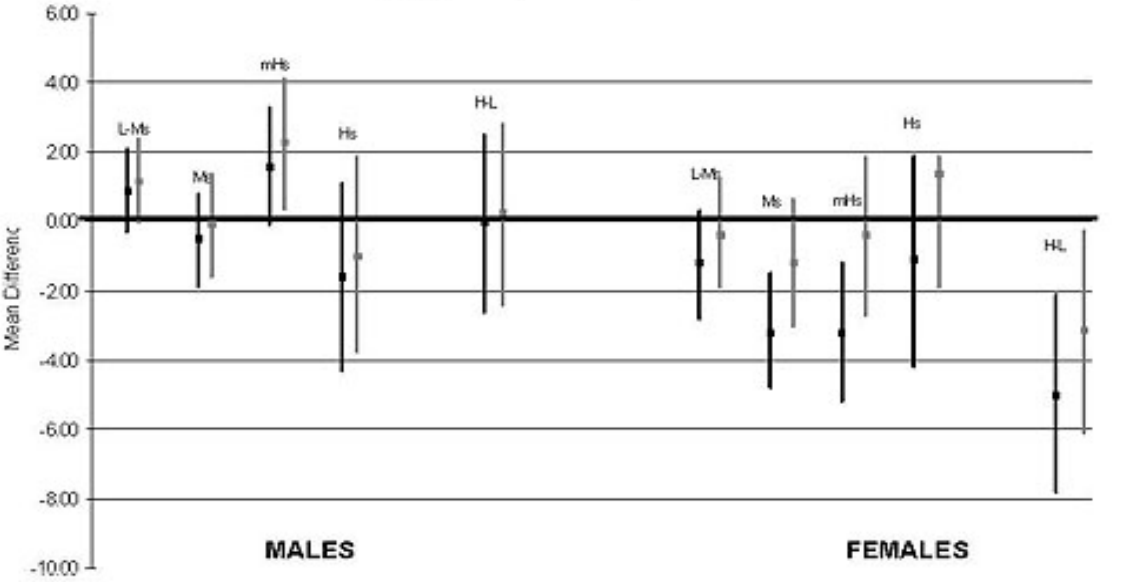
Figure 3.4: Age-adjusted mean differences of neighborhood trajectory classes versus the low stable group (95% CI) for LDL : by Gender



* Age-adjusted

* Age- ASEP- and Race-adjusted

Figure 3.5: Age-adjusted mean differences of neighborhood trajectory classes versus the low stable group (95% CI) for HDL : by Gender



* Age-adjusted

* Age- ASEP- and Race-adjusted

Figure 6: Relative Prevalence of neighborhood trajectory classes versus the low stable group (95% CI) for Hypertension : by Gender

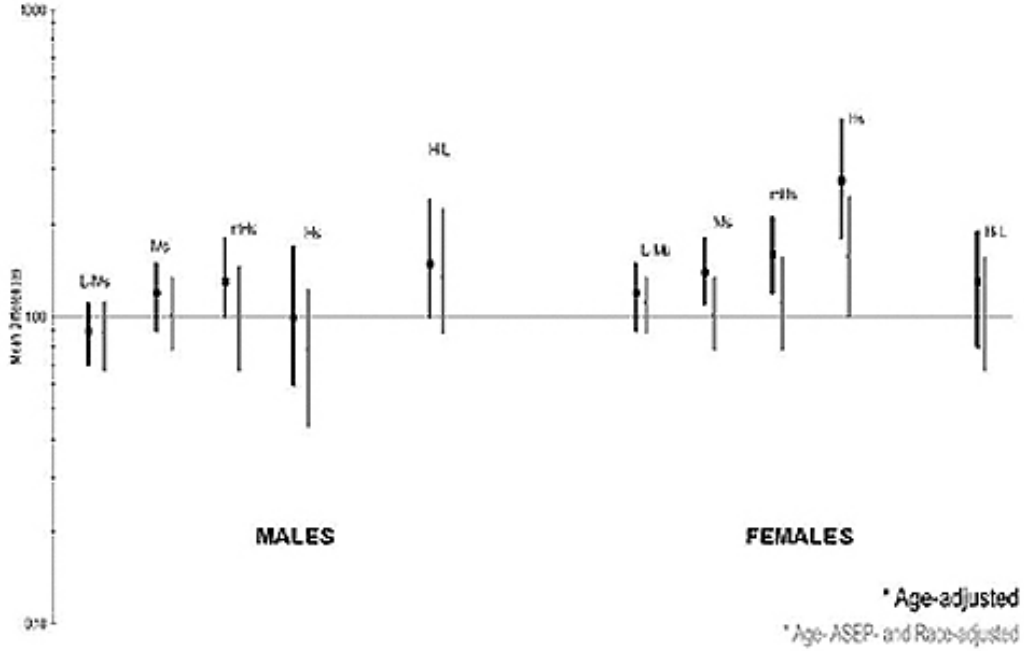


Figure 7. Relative Prevalence of neighborhood trajectory classes versus the low stable group (95% CI) for Diabetes : by Gender

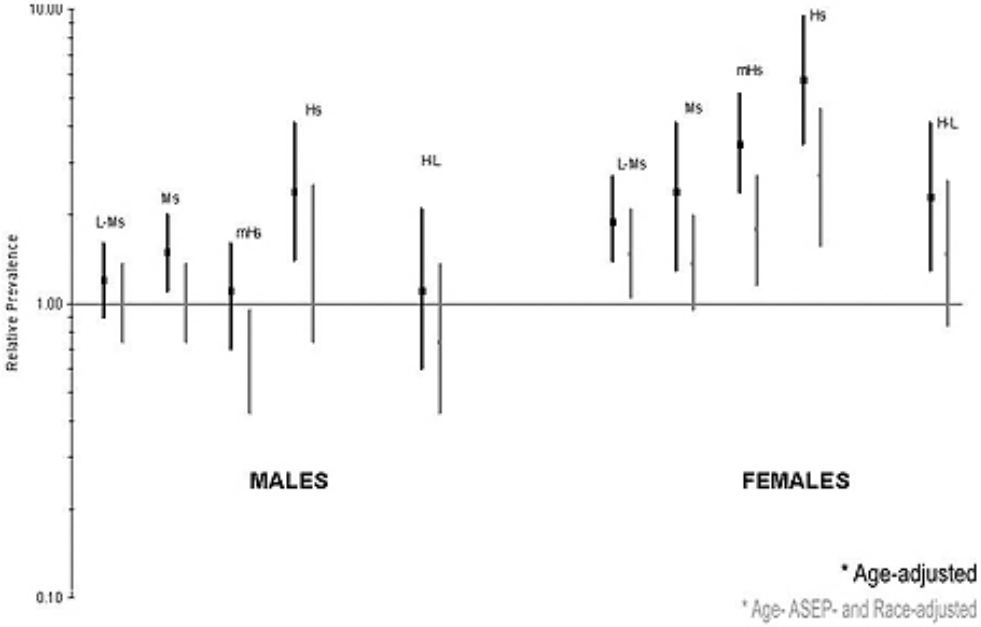
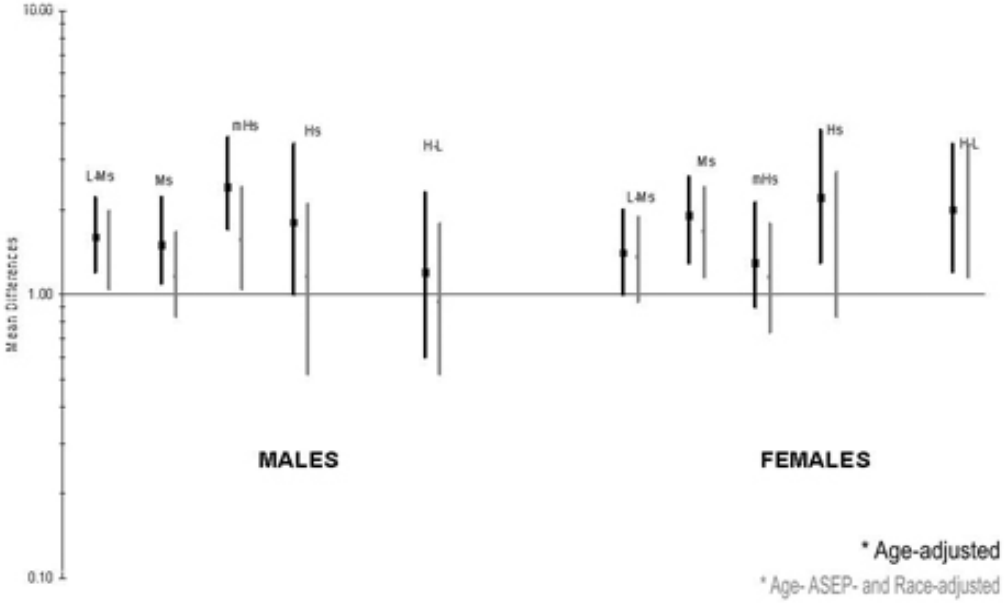


Figure 8: Age-adjusted Relative Prevalence of neighborhood trajectory classes versus the low stable group (95% CI) for Current Smoking : by Gender



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CHAPTER 4

HEALTH INDICATORS AS PREDICTORS OF RESIDENTIAL SOCIOECONOMIC MOBILITY IN THE ALAMEDA COUNTY STUDY

INTRODUCTION

In recent years a considerable number of publications have documented an association between properties of the area in which people live and their health. Different levels of aggregation have been used – regions, states, counties, census tracts, etc – with almost all finding associations between higher levels of socioeconomic deprivation of the areas in which people live and increased risk of mortality and morbidity, particularly cardiovascular-related outcomes [1-7]. By adjusting for individual-level characteristics of residents (for example with socioeconomic characteristics), researchers have attempted to separate the health effects of area deprivation from the effects of the individual-level attributes of the people who live there.

An important challenge in these analyses is separating true contextual area effects from selection of unhealthy people into disadvantaged neighborhoods. For example, if poor health predisposes people to move to a poorer area then selection of people into deprived areas as a function of their health may be what is responsible for the apparent contextual effects. The statistical adjustment strategies commonly used in neighborhood effects research may not fully account for these health selection effects. Despite the fact that health selection effects are often raised in critiques on neighborhood health effects

research, there are few empirical investigations of the impact of health on residential mobility.

Four main processes have been posited to explain residential mobility [8-11] and health could play a role in each of these. The first process involves changes in the housing market or other forces external to the household (such as changes in the job market, zoning changes, etc) causing dissatisfaction with housing and leading people to move to alleviate this dissatisfaction. Health could be involved in this process if, for instance, those in ill health move away from an area that they perceive as harmful to their health or move to areas which they perceive have better health related resources such as health services. The second process is that people adjust to changes in the life cycle by moving. For example, people may move to different areas as they age, and age is often correlated with health. The third process involves moves resulting from household formation/dissolution or job changes. For example, marriage and job changes may be linked to both good health and residential mobility. In contrast, job loss and divorce may also be linked to mobility and poor health. The fourth process involves moves as a result of financial hardship or gain. For example, the financial consequences of health conditions could result in housing loss or the need to move to less expensive housing.

There has been some evidence in the literature to support the theory that it is healthy people who are more likely to move. The 1991 England and Wales census showed that persons who had moved more than 50 km during the prior year were less likely than those who had not moved more than 50 km to report a limiting long-term illness [12]. Elderly African Americans who had moved to northern states at some point over their lifetime reported better health status than those who remained in the South

[13]. People in England and Wales who had moved regional residence from 1939 to 1971 had lower cardiovascular mortality ratios than those who had not moved. Alternatively though, two papers in the late 1980's, often cited in the literature, showed that among the elderly, movers were more likely to be in poor health than non movers, whereas no association between health and mobility was observed in younger people [10, 14]. Two more recent papers (2000s) have documented that movers may at first appear to be healthier, but when socioeconomic and demographic variables are controlled for, they are less healthy than non movers [15-16].

A limitation of past work on health and residential mobility has been that it has focused on dichotomous measures of mobility as the main endpoint, rather than on effects of health on the types of neighborhoods that people move to. Selection biases are not necessarily created by movers being different than non-movers, they are created when people move to socioeconomically advantaged or disadvantaged areas differentially based on their individual attributes; in this case, health.

Using 34 years of longitudinal data from the Alameda County Study (ACS), we examined if health-related indicators were associated with residential mobility. No literature exists that documents the influence of health on the socioeconomic environments in which people live. Based on the possibility that cross-sectional associations of neighborhood deprivation and health might be a consequence of chronically ill individuals moving to poorer neighborhoods [1, 7, 17], we hypothesized that the presence of health conditions would make a person less likely to move to a more socioeconomically advantaged area and more likely to move to a more socioeconomically disadvantaged area. The long follow-up period and repeat measures

of health-related indicators and residential socioeconomic characteristics of participants makes this sample particularly well suited to examine this research question.

METHODS

Study Population

The ACS is a prospective study of a stratified random sample of residents of Alameda County, California households. The study was initiated in 1965 on a sample of 8,023 non institutionalized adults from 4,453 housing units with age ranges from 16-94 years. The methods used for sampling and study design have been reported elsewhere [18]. Five subsequent waves of data collection were carried out in 1974, 1983, 1994, 1995, and 1999. In 1974, all respondents from the 1965 survey were re-contacted. In 1983, a 50% sample of all 1974 respondents not known to be dead were contacted. In 1994, all respondents from the 1983 sample and all those in 1974 that were not a part of the 1983 sample were re-contacted. In 1995 a shortened questionnaire was mailed to all 1994 respondents. The 1995 survey was not included in these analyses because of its close proximity to the 1994 survey. The 1999 survey included all respondents from the 1995 survey who could be re-contacted. For those eligible (not dead for all study waves and a part of the 50% sampling frame in 1983) and able to be located, completed questionnaires were received for 6,928 (86%) in 1965, 4,864 (85.1%) in 1974, 1,798 (87.3%) in 1983, 2569 (93%) in 1994, and 2,123 (96%) in 1999. Additional details on the size of the original sample, those located, and those with complete data are shown in Table 1.

Health Indicators

Six dimensions of health were investigated in these analyses: three medical conditions (heart trouble, stroke, & diabetes) and two risk factors (current smoking and body mass index (BMI). The medical conditions were investigated because they have the potential to result in mobility through a perceived need for better health services or financial hardships created through the onset of ill health. Risk factors were also examined because indirect selection could also occur if people with certain health risk factors move to or from specific areas [16]. Current smoking and BMI have been shown to be related to the medical conditions investigated [19-20] and may also have their own influence on mobility through financial costs associated with cigarette consumption [21-22] or obesity [23].

At each survey participants were given a list of medical conditions and asked if they had ever had any of the conditions. If they said yes, they were asked to indicate what year they first developed the condition. Respondents were considered to have the condition from that year onward. Risk factors were assessed at each study wave. Participants were considered current smokers if they answered yes to smoking any cigarettes at the present study wave. BMI was calculated as self-reported weight (lbs)/height^2*703. Participants were asked to report their weight without clothes on and their height without shoes.

Outcome Variables

There were four possible outcomes that participants could have experienced at each wave: not moved over the interval, moved to a socioeconomically better

neighborhood, moved to a socioeconomically worse neighborhood, or died. The classification of better or worse neighborhood was based on the median family income of the census tract of the residence of the participant. Median family income was chosen as the key neighborhood variable because it was the only census variable available in all census years between 1960 and 2000 that measured the socioeconomic condition of the area. Participant's addresses were based on reported addresses at each wave. Each address was Geocoded and assigned a census tract for the two closest census years.

Information on census tract median family income was obtained from various sources. Median family income for the 1960 census was listed directly for each census tract in the San Francisco-Oakland, California Standard Metropolitan Statistical Area [24]. For census years 1970 and 1980, median family income was calculated from grouped frequency distributions [25-26]. For years 1990 and 2000, data was directly extracted from The Census of Population and Housing Summary Tape File 3 [27-28]. All values were then converted to 2000 dollars to allow comparisons across various census years [29]. Addresses reported by each ACS participant at each study wave were assigned the census value for an interpolated value for that year based on the distance between the two closest census years [for example, median family income (MFI) 1965 = 0.5 (MFI 1960) + 0.5 (MFI 1970)]. Using this approach we created a database that contains a measure of census tract median family income for each wave for each study participant, given that they participated in that wave. Deaths were ascertained by active tracing and by linkage to the National Death Index. All matches were confirmed by detailed comparisons between death certificate information and previous information gathered from the participants.

If a participant died between study waves, their outcome for that interval was 'died'. If a participant did not die, change of residence since the last wave of data collection was assessed by comparing the participant's address at the preceding study wave to the current study wave. If the addresses were the same, then they were considered not to have moved during the prior interval. If the addresses were different, then their change in neighborhood median family income from the preceding interval to the current interval was assessed. If the change in median family income was equal to, or greater than $5000, then they were considered to have moved to a 'better' neighborhood. If the change in median family income was equal to, or greater than -$5000 than they were considered to have moved to a 'worse' neighborhood. The percentage of persons who had moved but whose change in median family income was between -$5000 to $5000 was only 9% of the sample during the 1965-74 interval and only 2-3% in subsequent intervals, so the categories of not moving or moving to a neighborhood of similar income were collapsed into one category of no neighborhood socioeconomic change. For those who moved, change in median family income varied across study intervals with a mean (SD) of ~ $3,500 (13,000) from 1965-74, $13,900 (15,800) from 1974-83, $5,400 (21,800) from 1983-94, and $-1,200 (18,900) from 1994-99, so cut-off values of $0, $2500, $5000 and $7500 models were also examined in sensitivity analyses. Due to small numbers of persons with medical conditions with changes in median family income above $5000, the $7500 cutoff model did not converge. Results for cut-offs $0, $2500, and $5000 were not substantially different, so a cut-off value of $5000 was chosen.

Covariates

Demographic and life event characteristics previously linked to residential mobility [30] were investigated as confounders. Demographic (age, gender, race/ethnicity, marital status) and socioeconomic (education, income, and employment status) data was collected at all waves. Participants were asked to choose their race/ethnicity from nine categories: white, black, American Indian, Asian, Chinese, Japanese, Filipino, Hispanic, or other. Marital status was selected from five categories: never married, married, separated, divorced, or widowed. Participants were asked to circle how many grades they had finished in school: none, grade school 1-8, High School 9-12, or college 1-5+ or advanced degree. Income was collected by the participants choosing an income category from a list of categories that varied from wave to wave. Income data was then subsequently combined with the Current Population Survey (CPS) of the same year in order to impute continuous data at each wave [31-32]. Imputed incomes for the participants were conditional on the distributions observed in the CPS data for persons similar in age, education, gender, race, marital status, occupation, and number of household members. At each wave, the same procedure was run five times, with different random starting seeds. The resulting five imputed values averaged out to represent the final imputed income for that wave. All values were then adjusted for inflation to year 2000 dollars to make values equivalent across time. Employment status was collected in three slightly different ways depending on the survey year. Categories were only slightly variant and collected the same information, so were collapsed to make them equivalent across study waves.

Life event (separated or divorced, job loss, death of spouse, financial problems) data was collected at all waves after the baseline wave (1974, 1983, 1994, and 1999). For a given survey year, participants were asked to indicate if selected life events had happened to them during the years between the last survey and the current survey. If they said that a life event had happened to them in the preceding interval, they were considered to have that exposure at the current study wave. The life events included in these analyses were selected based on prior work indicating that they may be related to residential mobility [30]**.** They included: separated or divorced ('Your own marital separation or divorce), lost job ('Loss of your job or other job problems or difficulties'), death of a spouse ('Death of your husband or wife'), and financial problems ('Serious financial difficulties or problems').

Statistical analysis

All demographic, life event, health indicators, and outcome categories were compared across study waves using ANOVA (continuous variables) and the chi-square statistic (categorical variables). Multinomial logistic regression, interpreted as a multivariate binary model [33], was used to assess the association between each of the health indicators at the beginning of the interval and the odds of moving to a better neighborhood, moving to a worse neighborhood, or dying (as compared to not moving) during the subsequent interval. Models were fitted to estimate the effect of each health indicator separately. All health indicator, demographic, socioeconomic, and life event measures were modeled as dichotomous or categorical variables. Body mass index was collapsed into three categories: <25, 25-29.9, and 30+.

Generalized estimating equations (GEE) were used to account for within person correlations over time. The model was defined as:

$$\text{Log}\ (r_i/r\text{-}1) = ß_{0r} + X_{ij}\ ß_{1r},\ r=2,...,\ R$$

Where $ß_{0r}$ is the log odds of the binary response category [2 = died, 3 = moved to a better neighborhood, and 4 = moved to a worse neighborhood] versus the reference [not moved] for person i during interval j when $X_{ij}=0$, and $ß_{1r}$ the change in the logs odds of the binary response category with a one-unit change in $X_{ij.}$ X_{ij} is characteristic for person i at the beginning of the interval j. PROC GENMOD with a 'repeated' statement was used to model the correlations within subjects; the subject effect was specified as an interaction of the intercept with the original subject variable, and an independent working correlation matrix (the only correlation accepted with interacting subject effects) was used.

In order to examine the potential role of demographics, socioeconomics, and life events as confounders or mediators of any differences observed, associations of these factors with mobility were also examined. Age was collapsed into five categories: <30, 30-39, 40-49, 50-59, and 60+ years. Race/ethnicity was collapsed into three categories: white, black, and other. Marital status was collapsed into three categories: never married, married, and separated/divorced/widowed. Education was collapsed into three categories: less than high school, complete high school, and more than high school. Income was collapsed into approximate tertile categories: <$40,000, $40-79,999, and >$80,000. Employment status was collapsed into four categories: employed full time,

unemployed, retired, and other. Life events were dichotomous (Y/N). The final model included adjustment for age, income, education, and all life events.

RESULTS

Of those who completed a questionnaire or were known to have died during the subsequent study interval, 4769 (85.4%) could be categorized into one of the four outcomes [died, not moved, moved to a better neighborhood, moved to a worse neighborhood] at the end of the interval for the 1965 study wave, 2265 (86.4%) for the 1974 study wave, 2250 (57.4%) for the 1983 study wave, and 2482 (94.3%) for the 1994 study wave. 2,259 participants had complete data for one interval, 1849 for two intervals, 752 for three intervals, and 619 for all four intervals. Reasons for exclusion included non-response, lost to follow-up, missing address at one or both study waves, one or both of the addresses could not be geocoded, or median family income values were not available for one or both of the census tracts in which they had lived at either study wave (see table 4.1). Compared to persons with data on fewer intervals (range of one to four intervals possible), persons with information from more intervals were more like to be older, White, married, educated, have higher incomes, be employed, and be of normal weight.

Selected demographic, socioeconomic, life event, health indicator, and outcome variables by study interval are shown in table 4.2. At baseline, the mean age for the sample was 46 years, 45% male, 80% White, 12% Black, and 9% other race/ethnicity, 76% married, 37% had less than a High School education, 38% had 2000 income less than or equal to $40,000 a year, 58% were employed, 43% were current smokers, 7%

were obese, 3% had heart trouble, <1% had a stroke, 2% diabetes, and the mean neighborhood median family income in 2000 dollars was ~$46,000 a year. Across study waves the sample is more White, more educated, the middle income group decreased while the lower and upper income groups increase, less people were employed or otherwise employed (mostly homemakers) while more were retired, less were never married but more become separated, divorced, or widowed, the prevalence of current smoking generally went down with an upward bump in 1983, and the prevalence of heart trouble, high blood pressure, stroke, diabetes, and overweight and obesity all increased. Between the 1965 and 1974 wave, 52% did not move (or moved to a similar neighborhood), 23% moved to a better neighborhood, 11% to a worse neighborhood, and 13% died. The number of years between each study wave differs, but in general participants are the most likely to have died during the 1983-1994 wave, the percentage moving to a better neighborhood decreases across study interval, and participant's are the least likely to move to a worse neighborhood during the 1974-83 interval and least likely to not move during the 1983-94 interval.

Table 4.3 shows associations of health indicators at the beginning of the interval with the odds of dying, moving to a better neighborhood, or moving to a worse neighborhood (as compare to not moving) during the subsequent interval. After adjustment for age, a BMI less than 25, current smoking, and stroke were associated with increased odds of dying versus not moving. The presence of heart trouble, stroke and diabetes were each associated with lower odds of moving to a better neighborhood versus not moving, although associations were only statistically significant for diabetes. High BMI was associated with increased odds of moving to a worse neighborhood versus not

moving (but no dose response by BMI level was observed). The presence of a stroke was associated with lower odds of moving to a worse neighborhood versus not moving but confidence intervals were very wide.

To simplify presentation of results, table's 4a-4b show separate results for moving to a better neighborhood and moving to a worse neighborhood, versus not moving, respectively. Separate adjustment for demographics, socioeconomics, and life events did not substantially change the associations between the health indicators and moving to a better neighborhood versus not moving, although confidence intervals widened. The association of diabetes with lower odds of moving to a better neighborhood versus not moving was no longer statistically significant after adjustment. Higher BMI remained associated with increased odds of moving to a worse neighborhood versus not moving after adjustment for demographic, socioeconomic and life events indicators. Adjustment for life events strengthened the association between all medical conditions and decreased odds of moving to a worse neighborhood versus not moving, although only heart trouble and diabetes were statistically significant.

DISCUSSION

The study found some evidence for an effect of health on residential mobility migration over a 34 year period in participant's who had been residents of Alameda, CA in 1965. However, the direction of the effect was inconsistent and not always in the hypothesized direction. The medical conditions we examined were associated with lower odds of moving to both better and worse neighborhoods (versus not moving). The

clearest pattern was observed for BMI>30 which was consistently associated with increased odds of moving to a worse neighborhood versus not moving.

In age-adjusted models, diabetes was the only health indicator significantly associated with lower odds of moving to a better neighborhood versus not moving. Similar albeit not statistically significant associations were observed for heart trouble and stroke. Point estimates did not change after adjustment for socioeconomic and life events characteristics but all confidence intervals included the null value. Counter to previous literature, our results indicate that stroke, diabetes and heart trouble reduced the odds of moving to a worse neighborhood (versus not moving) after adjustment for socioeconomic characteristics and life events but only associations with stroke and diabetes were statistically significant.

These two findings taken together suggest that health is acting as a barrier to movement in general. In our data, when moving to a better neighborhood and moving to a worse neighborhood categories were collapsed into one category of 'moving', all medical conditions were shown to be associated with lower odds of moving versus not moving (data not shown). This is in keeping with literature that shows that migration is the purview of the healthy [12-13]. When asked by the 2000 Current Population Survey what their main reason was for moving, only 1% of people indicated a health reason. More than half designated housing related reasons, such as wanting to own a home, new/better housing, etc as their main reason for moving with family-related and work-related reasons making up most of the other reasons [34]. Migration research has also consistently shown that movers are younger, White, more educated, have higher status

occupations, and have had recent changes in career patterns and life cycle changes than people who do not move [30].

We also found that adjusting for life events attenuated the inverse relationship between diabetes and moving to a better neighborhood versus not moving and strengthened the inverse association between heart trouble and moving to a better neighborhood versus not moving. All associations with medical conditions and moving to a worse neighborhood versus not moving were also strengthened by adjustment for life events. Negative life events such as separation/divorce [35], financial problems [36-37], job loss [38], and death of a spouse [39-40] have been documented in the literature as affecting health and also to influence residential mobility [37, 41-42]. There is little evidence that health influences negative life events, so it is likely that they are confounders rather than mediators of the relationship between medical conditions and movement to a better or worse neighborhood (versus not moving).

Stroke was more strongly associated with reduced odds of moving to a worse or better neighborhood (versus not moving) than diabetes or heart trouble. This suggests that some medical conditions may pose a greater barrier to mobility than others. Stroke is the leading cause of serious, long-term disability in the U.S. [43] and people with stroke are most likely to report difficulty with basic activities of daily living with potentially greater effect on financial and work-related problems [44-45]. As opposed to an induced move produced from loss of a housing unit, unhealthy individuals may be staying in their residences because they either do not have the financial resources to move or do not having the physical or mental capability for doing so.

Obesity was the only health indicator to be associated with increased odds of moving to a worse neighborhood versus not moving. Adjustment for socioeconomics slightly strengthened this association. Individual adjustment showed that employment status was the only socioeconomic variable to strengthen the association between obesity and movement to a worse neighborhood. In our data, retirement was associated with decreased odds of moving to a worse neighborhood versus not moving and unemployment with increase odds of moving to a worse neighborhood versus not moving. The Survey on Health and Aging in Europe (SHARE) [46] has recently shown that those retired or unemployed are more likely to be obese than those employed, suggesting that retirement may be negatively confounding the association between obesity and moving to a worse neighborhood.

There are several limitations of the analyses. Data on participant's residential mobility were collected at intervals of varying length. In addition, we have no information on additional moves occurring within the intervals. In two of the waves (1974 and 1983) participants were asked how many times they had moved since the prior wave. During the 1965-74 wave and 1974-83 intervals the mean numbers of moves reported by participants during the interval was 2.4 (SD 2.8) and 1.9 (SD 1.4), respectively. This suggests that our analyses may have missed a substantial number of moves within intervals. We used a single measure of neighborhood socioeconomic position to characterize neighborhoods and proxied neighborhoods by census tracts, both of which may have resulted in misspecification of neighborhood conditions. Geographic boundaries of census tracts could also change over time and median family income values are more likely to be missing in earlier census years. The associations between

these indicators and residential mobility could also be changing over time, something not captured in a pooled analysis. The self-reported collection of all demographic, life event, and health indicators may have also resulted in misclassification and could have contributed to underestimates of effects. As mentioned previously, persons with more intervals of complete data were different than persons with less complete data. It is plausible that that these differences may be related to both the presence of health indicators and the odds of moving to a better or worse neighborhood, potentially resulting in misestimates of effects. The study population was also sampled from one county on the east side of the San Francisco Bay area at one point in time and may not be representative of the greater US population or even that county at this point in time.

An important strength of our study is the use of multiple repeated measures of demographic, life events, health-related indicators, and socioeconomic status of participant's residences. Data was collected prospectively and was able to capture exposure to these individual attributes and changes in outcomes for the same people over a 34 year period. At baseline, there were no upper restrictions on age and sampling done on household, so that the study sample captured people at all ages and stages of the life-course. Our results show that contrary to hypothesis in the literature, medical conditions are more likely to restrict mobility than to lead to upward or downward residential mobility. If health selection is occurring, it is most likely to be an indirect result of the socioeconomic consequences of extreme weight or general increased mobility of healthy individuals. Suggesting that the associations between area deprivation and health are more likely to be a result of the areas in which people live affecting health rather than persons of ill health moving to socioeconomically deprived areas. And buoying the

argument that the physical environment in which we inhabit may be just as, or more, important to public health as the individual health choices that we make.

Table 4.1. Number (%) of completed questionnaires, addresses, census tract assignment, and median family income of the Alameda County Study population for each interval (1965-99).

Study Wave	Alive	Sample	Located	Completed questionnaire	Address obtained	Census Tract assigned for both census years	Median family income available for both census tracts
	N	N (%)	N (%)[a]	N (%)[b]	N (%)[c]	N (%)[d]	N (%)[e]
1965	6928	6928 (100.0)	6928 (100.0)	6928 (100.0)	6908 (99.7)	6828 (98.8)	6631 (97.1)
1974	6209	6209 (100.0)	5907 (95.1)	4864 (85.1)	4466 (91.8)	4159 (93.1)	4149 (99.8)
1983	5385	3105 (50%)	2060 (66.3)	1798 (87.3)	1796 (99.9)	1564 (87.1)	1563 (99.9)
1994	4193	4193 (100.0)	2934 (70.0)	2729 (93.0)	2729 (100.0)	2556 (93.7)	2547 (99.6)
1999	3685	3685 (100.0)	2211 (60.0)	2123 (96.0)	2089 (98.4)	1951 (93.4)	1944 (99.6)
Total	26,400	24,142 (91.4)	20,040 (83.0)	18,444 (92.0)	17,988 (97.5)	17,058 (94.8)	16,834 (98.7)

a Percent located out of those sampled.

b Percent who completed questionnaire out of those that were located.

c Percent of addresses obtained out of those who completed a questionnaire

d Percent of census tracts assigned for both census years out of those with addresses obtained.

e Percent of neighborhood median family income values available for both census tracts for those with census tracts assigned.

Table 4.2. Demographic, Life event, and Health Indicators of the Alameda County Study population for each interval (1965-99)

Demographics (beginning of interval)	1965-74 N=4769	1974-83 N=2265	1983-94 N=2250	1994-99 N=2482
Male, %	2147 (45.0)	949 (45.8)	653 (44.6)	997 (43.5)
Mean Age (SD) **	46.1 (16.2)	54.1 (15.3)	58.2 (13.1)	64.7 (10.4)
Race/ethnicity, % **				
White	3798 (79.6)	1697 (81.8)	1198 (81.8)	1912 (83.5)
Black	546 (11.5)	221 (10.7)	145 (9.9)	167 (7.3)
Other	425 (8.9)	156 (7.5)	122 (8.3)	211 (9.2)
Marital Status, % **				
Never married	399 (8.3)	94 (4.5)	41 (2.9)	42 (1.9)
Married	3602 (75.5)	1539 (74.3)	1068 (75.9)	1627 (73.2)
Sep/Div/Widowed	768 (16.0)	438 (21.2)	299 (21.2)	553 (24.9)
Mean Neighborhood Median Family Income (in 2000 dollars) **	45,764.0 (12,257.5)	49,749.8 (16,478.9)	65,189.5 (24,520.9)	72,092.6 (30,668.8)
Socioeconomics (beginning of interval)				
Education, % **				
<12 yrs	1757 (37.0)	661 (32.1)	335 (23.0)	344 (15.2)
12 yrs	1472 (31.0)	606 (29.4)	429 (29.6)	611 (27.0)
>12 yrs	1515 (31.9)	795 (38.6)	688 (47.4)	1312 (57.8)
Income† (2000 dollars), % **				
<=40,000	1818 (38.1)	846 (40.8)	640 (43.7)	974 (42.5)
40,000-79,999	2412 (50.6)	847 (40.8)	541 (36.9)	779 (34.0)
=>80,000	539 (11.3)	381 (18.4)	284 (19.4)	537 (23.5)
Employment Status, % **				
Employed	2752 (57.7)	1098 (52.9)	767 (52.4)	927 (40.5)
Unemployed	100 (2.1)	62 (3.0)	49 (3.3)	44 (1.9)
Retired	451 (9.5)	461 (22.2)	437 (29.8)	1065 (46.5)
Other	1466 (30.7)	453 (21.8)	212 (14.5)	254 (11.1)
Life Events (during prior interval)				
Separated/Divorced, % **	-	197 (9.7)	160 (11.0)	171 (7.5)
Lost job, % **	-	165 (8.1)	169 (11.7)	310 (13.5)
Death of spouse, % **	-	135 (6.6)	80 (5.5)	201 (8.8)
Financial problems, %	-	285 (14.0)	227 (15.7)	311 (13.6)
Health Indicators (beginning of interval)				
Current smoker (yes) **	2006 (42.5)	678 (32.8)	388 (45.2)	311 (24.5)
BMI **				
<25	1322 (27.9)	622 (30.3)	465 (32.0)	807 (35.7)
25-29.9	3098 (65.5)	1276 (62.2)	831 (57.2)	1087 (48.1)
30+	312 (6.6)	152 (7.4)	156 (10.7)	366 (16.2)
Heart trouble **	122 (2.6)	209 (10.1)	182 (12.4)	489 (21.4)
Stroke **	11 (0.2)	53 (2.6)	31 (2.1)	89 (3.9)
Diabetes **	74 (1.6)	119 (5.7)	92 (6.3)	216 (9.4)
Outcomes (During the Interval)				
Not Moved**	2685 (56.3)	1148 (50.7)	800 (35.6)	1664 (67.0)
Moved Better Neighborhood **	985 (20.7)	312 (13.8)	167 (7.4)	143 (5.8)
Moved Worse Neighborhood **	406 (8.5)	73 (3.2)	178 (7.9)	186 (7.5)
Died **	693 (14.5)	732 (32.3)	1105 (49.1)	489 (19.7)

** Test for trend significant at 0.05 level.

† Average of five imputed values, adjusted to 2000 dollars.

Table 4.3. Age-adjusted odds ratios (95% CIs) of dying, moving to a 'better' neighborhood, and moving to a 'worse' neighborhood compared to not moving: Health Indicators of the Alameda County Study Population (1965-1999)

	Died	Moved to a 'better' neighborhood	Moved to a 'worse' neighborhood
Body Mass Index			
<25	1.30 (1.12, 1.50)	1.05 (0.96, 1.15)	1.13 (0.92, 1.40)
25-29 (ref)	-	-	-
30+	0.80 (0.56, 1.11)	0.85 (0.64, 1.12)	1.26 (1.10, 1.45)
Current Smoker	1.69 (1.55, 1.85)	1.04 (0.96, 1.12)	1.09 (0.97, 1.22)
Heart trouble	1.00 (0.58, 1.76)	0.90 (0.81, 1.00)	1.03 (0.82, 1.30)
Stroke	1.85 (1.10, 3.13)	0.70 (0.41, 1.17)	0.64 (0.23, 1.74)
Diabetes	1.29 (0.70, 2.35)	0.81 (0.69, 0.96)	0.96 (0.67, 1.37)

Table 4.4a. Adjusted odds ratios (95% CIs) of moving to a 'better' neighborhood compared to not moving: Health Indicators of the Alameda County Study Population (1965-1999)

Moved Better Neighborhood vs. Not moved	Age-adjusted	Age & Demographic Adjusted	Age & Socioeconomic adjusted	Age & Life Event Adjusted	Age, Income, Education, & Life Event Adjusted
Body Mass Index					
<25	1.05 (0.96, 1.15)	1.02 (0.91, 1.13)	1.16 (1.09, 1.23)	1.10 (0.95, 1.26)	1.09 (0.96, 1.25)
25-29 (ref)	-	-	-	-	-
30+	0.85 (0.64, 1.12)	0.87 (0.67, 1.13)	0.83 (0.62, 1.12)	0.91 (0.59, 1.42)	0.89 (0.59, 1.34)
Current Smoker	1.04 (0.96, 1.12)	1.01 (0.94, 1.10)	1.01 (0.90, 1.14)	1.00 (0.78, 1.28)	0.98 (0.75, 1.29)
Heart trouble	0.90 (0.81, 1.00)	0.90 (0.80, 1.01)	0.92 (0.88, 0.97)	0.86 (0.82, 0.91)	0.85 (0.81, 0.90)
Stroke	0.70 (0.41, 1.17)	0.71 (0.45, 1.12)	0.86 (0.54, 1.35)	0.75 (0.39, 1.45)	0.75 (0.39, 1.44)
Diabetes	0.81 (0.69, 0.96)	0.85 (0.73, 1.00)	0.81 (0.62, 1.07)	0.89 (0.69, 1.13)	0.89 (0.71, 1.11)

Table 4.4b. Adjusted odds ratios (95% CIs) of moving to a 'worse' neighborhood compared to not moving: Health Indicators of the Alameda County Study Population (1965-1999)

Moved Worse Neighborhood vs. Not moved	Age-adjusted	Age & Demographic Adjusted	Age & Socioeconomic adjusted	Age & Life Event Adjusted	Age, Income, Education, & Life Event Adjusted
Body Mass Index					
<25	1.13 (0.92, 1.40)	1.12 (0.89, 1.41)	1.17 (0.92, 1.48)	1.05 (0.73, 1.50)	1.01 (0.71, 1.46)
25-29 (ref)	-	-	-	-	-
30+	1.26 (1.10, 1.45)	1.23 (1.10, 1.39)	1.34 (1.17, 1.54)	1.16 (0.95, 1.42)	1.18 (1.00, 1.39)
Current Smoker	1.09 (0.97, 1.22)	1.08 (0.96, 1.21)	1.09 (0.95, 1.25)	0.99 (0.82, 1.19)	1.03 (0.85, 1.25)
Heart trouble	1.03 (0.82, 1.30)	1.03 (0.81, 1.29)	1.05 (0.90, 1.21)	0.90 (0.72, 1.11)	0.91 (0.73, 1.14)
Stroke	0.64 (0.23, 1.74)	0.61 (0.25, 1.52)	0.78 (0.28, 2.13)	0.38 (0.19, 0.75)	0.38 (0.18, 0.82)
Diabetes	0.96 (0.67, 1.37)	0.97 (0.71, 1.32)	0.92 (0.66, 1.28)	0.78 (0.62, 0.98)	0.83 (0.68, 1.02)

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CHAPTER 5

CONCLUSIONS

Summary of findings

The purpose of this dissertation was to move beyond single point in time assessments of neighborhood socioeconomic position (SEP) and to explore how individuals, neighborhoods of residence, and health interact over time. When trying to separate out the effects of neighborhoods on CVD, it is important to take into the account the present and past SEP of the people that live in the neighborhood and to understand the processes which influence people to move from neighborhood to neighborhood, or not move at all. Lifecourse assessments of individual and neighborhood SEP, and the ability to explore the directionality of the association between SEP and health outcomes will advance our understanding of the causal processes involved in neighborhood of residence influencing CVD. My overall objective was to: examine associations between lifecourse individual and neighborhood measures of SEP and subclinical atherosclerosis, examine neighborhood trajectories of SEP and investigate how they are related to subclinical atherosclerosis, and investigate the role of selection of people with disease into disadvantaged neighborhoods.

In answer to the primary hypothesis in **Chapter 2,** after adjustment for age, childhood SEP (CSEP) and adult SEP (ASEP) were both inversely and independently associated with common carotid intimal-medial thickness (IMT) in men [mean difference

for highest vs. lowest tertile (95% CI): -20.8 (-40.6, -1.1) for CSEP; -16.3 (-37.6, 4.9) for ASEP]. All three indicators: CSEP, ASEP, and 20-year average exposure to neighborhood poverty (NSEP) were inversely and independently associated with IMT in women [-24.0 (-41.0, -7.0) for CSEP; -18.2 (-37.1, 0.6) for ASEP; -24.5 (-41.5, -7.5) for NSEP). Associations were somewhat reduced after adjustment for cardiovascular risk factors, suggesting that these factors may play a mediating role. For CSEP and ASEP, associations were slightly stronger in women but were generally of the same strength and in the expected direction for both genders. Evidence existed for heterogeneity of effects by gender for the NSEP and IMT association and by race/ethnicity for the ASEP- and NSEP- IMT associations.

In the second paper, **chapter 3,** I examined the effects of trajectories of neighborhood poverty over time. Six distinctive clusters of neighborhood census tract poverty were found over a 20-year period; five stable trajectory groups with variation in level of neighborhood poverty and one trajectory group with significant upward residential mobility (change of 30-10% neighborhood poverty over the 20 years). Generally, and mostly in women, higher stable neighborhood poverty was associated with worse cardiovascular outcomes than lower stable neighborhood poverty over the 20 years. The ability to look at trajectories was limited by the lack of changes in neighborhood poverty over the study period, but in general the mobility group had outcomes comparable to stable groups with similar cumulative exposures. Some evidence did exist for effects of upward mobility for CIMT and hypertension in men and for LDL and HDL in women. For women, the medium-high to low group had a lower mean HDL and higher mean LDL than all of the other trajectory groups, with only three

of the five HDL comparisons, and none of the LDL comparisons statistically significant. For men, CIMT's were lower and the prevalence of hypertension higher than any of the other trajectory groups, although these differences were not statistically significant.

In the third paper, **chapter 4**, I found some evidence for an effect of health on residential mobility migration over a 34 year period in participant's who had been residents of Alameda, CA in 1965. However, the direction of the effect was inconsistent and not always in the hypothesized direction. All medical conditions showed a (non-significant) decreased odds of moving to a better neighborhood and a decreased odd of moving to a worse neighborhood, after adjustment for life events, with stroke and diabetes showing stronger associations than heart trouble. Being underweight was slightly associated with movement to a better and worse neighborhood with the strongest association after adjustment for socioeconomic variables. Being obese significantly increased the odds of moving to a worse neighborhood but did not decrease the risk of moving to a better neighborhood.

Strengths and limitations

Generalizability

A strength of the work presented here is that both data sets utilized for this project are large population-based samples. However, both have separate selection and recruitment designs that may limit the generalizability of the research findings presented. MESA was specifically designed to investigate the prevalence, correlates, and progression of subclinical CVD in men and women 45-84 years old, with particular emphasis on an ethnically diverse study population. Investigations of SEP with IMT in

persons without clinical CVD allow examination of whether SEP is related to early stages of the development of atherosclerosis. Exclusions based on a history of CVD could have resulted in underestimates of the true association between SEP and IMT if excluded persons had both lower SEP and higher IMT. Men also develop clinically apparent CVD earlier than women, with a differential exclusion of men resulting in greater underestimates of the SEP-IMT association in men than women. Persons excluded due to missing data were also more likely to be of low ASEP, with potential underestimates of ASEP with IMT if people with low ASEP also had higher IMT.

Younger age groups, particularly the 20-29 year age group are the most mobile and mobility decreases with age [1], so age restrictions could have resulted in a non-mobile study population and contributed to the inability to examine differences in upward or downward residential mobility with stable neighborhood socioeconomic conditions. The over-sampling of minority race/ethnic groups also allows investigation of homogeneity of SEP/IMT effects across race/ethnic groups. However, MESA was not designed to be representative of the four race/ethnic groups included, although general patterns observed in terms of risk factors suggest that associations may be generalizable.

The Alameda County Study also utilized a population-based sampling frame with an added stratified random sampling to make it representative of all households living in Alameda County, CA in 1965. The study had no upper restrictions on age, so consequently was able to cover the full adult age spectrum of 16-94 years old. According to the 1960 census [2], the study racial distribution of ~ 80% white, 10% Black, and 10% other is comparative to the population of Alameda County at that time. The population was only sampled from one county on the East Bay of the San Francisco bay area and is

probably not representative of the greater US population. The County itself has even undergone population and demographic changes, making this sample representative of one geographic area at one point in time.

Longitudinal assessments of individual and neighborhood SEP

An important strength of this work was the ability to assess individual and neighborhood SEP at multiple points in the life-course. In paper 1 (chapter 2), we were able to include measures of both childhood and adult SEP, with the measure of adult SEP encompassing not only traditional indicators of income and education, but also wealth. In papers 1 and 2 (chapters 2 and 3) neighborhood exposure was assessed over a 20 year period as opposed to a measure for a single point in time. Paper 3 (chapter 4) captured 34 years of prospectively collected individual SEP measures and residential information collected at six intervals. Limitations are that these assessments do not cover the entire life span, and in the case of the ACS, movements within intervals were missed. Further work is needed that captures individual and residential information over the entire Lifecourse to further elucidate the processes linking SEP to poor health.

Measurement Error

Individual-level measures. There were many potential sources of measurement error for the individual-level measures used in these analyses; that shall be addressed separately for each study. For paper 1 (chapter 2), childhood SEP was based on a self-reported retrospectively collected father or care-taker's education. Father's education may also be an imperfect proxy for childhood social environment. All of these factors could have

potentially resulted in misclassification and contributed to underestimates of SEP effects. Despite its limitations however, the measure we used are commonly used in the literature and are most likely to reflect CSEP for the age cohorts included in MESA.

ASEP, characterized by combining information on income, education, and wealth, was used as a main exposure for paper 1 (chapter 2) and as an adjustment variable in paper 2 (chapter 3). Using not only traditional indicators of income and education, but also wealth, allowed us to develop a better composite of an individual's SEP. It is still an imperfect measure, and only reflects SEP at one time point in mid to late adulthood, but is an improvement over prior methods [3]. The indicators used to measure wealth only assessed ownership of housing units, automobiles, investments, and land. Future research with actual monetary values of said property and/or net worth/retirement/savings account assessments may allow more accurate assessments of an individual's wealth status and reduce potential measurement error of ASEP.

For the Alameda County Study (chapter 4), the main exposure variables of health indicators and adjustment variables of demographic, socioeconomic, and life events were collected prospectively, but similar to MESA were self-reported. While definitions of demographic and socioeconomic variables may have been clear and consistent, life event and health indicators were not based on rigid medical definitions, could have been interpreted differently by different people across time, and were collected at cross-sectional slices. All these factors which could lead to misclassification and contribute to underestimation of effects. The same persons and the same questions were asked at each wave though, resulting in consistent multiple repeated measures, and changes in these measures, over a 34 year period.

Neighborhood-level measures. A major strength of this work was the ability to capture information on exposure to neighborhood conditions for long periods of time in adulthood (20 continuous years in MESA and 6 waves in ACS encompassing 34 years) as opposed to a measures for a single point in time. It is plausible that neighborhoods could have different effects at different points in time. Neighborhoods are not static; what a certain level of poverty in a neighborhood is at a certain time may equate to something different depending on what time epoch is investigated. Neighborhoods may also affect residents differently depending on their age group. Depending on a person's stage in the life-course, they may or may not utilize different features of the neighborhood. And if certain features are predominant in poor or rich neighborhoods, then the effect may be stronger or only in certain age groups. Participants in both studies utilized, especially the ACS, covered a wide variety of age groups in adulthood. Some age stratified and time period stratified analyses were conducted, but this was not the main focus of this research topic and is a topic for future research. Ideally, to capture the full effect of neighborhood SEP on adult health, exposures should be collected over the entire life-course but that is outside the scope of this project.

The neighborhood SEP measure in both projects was comprised of only a single indicator and census tracts were used as proxies for neighborhoods. For papers 1 and 2 (chapters 2 and 3), neighborhood census tract poverty was chosen as the key variable investigated because it is often used in sociological work to characterize neighborhood conditions [4]. Median family income was chosen as the key neighborhood variable for paper 3 (chapter 4) because it was the only census variable utilized in all census years

between 1960 and 2000 that measured the socioeconomic condition of the area (census tract poverty was first used in the 1970 census). There is no consensus in the literature on what measure of neighborhood SEP is the most accurate measure of neighborhood socioeconomic conditions, with most measures and combinations of census tract aggregate measures resulting in significant associations with health [5-14]. It is important when conducting longitudinal assessments of neighborhoods to have the indicators be available and mean the same thing over the entire time period and both neighborhood census tract poverty and median family income (adjusted for inflation) fulfill this condition. Future work has begun (15-17), and is needed, that moves beyond aggregate measures of neighborhoods SEP to directly measure what specific features of the neighborhood environment are contributing to poor health.

There is also no consensus in the literature on what measure or geographic boundary of neighborhoods is the most accurate or appropriate to examine neighborhoods effect on cardiovascular health. Census tracts are administratively drawn for the purpose of taking a census, designed to have between 2,500 and 8,000 persons that are socioeconomically homogenous. They do not cross county boundaries; their size can vary widely depending on population density, and may change over time as populations grow or decline [18]. For papers 1 and 2, a specific database was utilized that allowed comparison across census years by recalculating and normalizing past census years to 2000 census tract boundaries. The earliest census year this method was applied to was the 1970 census, so this database could not be used for paper 3. Areas which experienced large population changes, increases or decreases, were more likely to experience boundary changes. Future work has also begun that explores different designations of

neighborhoods [19], subjective versus objective measures [20], etc to begin elucidating the causal processes of neighborhoods effects health.

Major Contributions and Conclusion

This body of work will serve as a significant contribution to our understanding of how people, places, and cardiovascular disease interact over time. The existing literature examining associations between neighborhoods and cardiovascular outcomes has been criticized for assessing individual and neighborhood SEP at single points in time, usually at the same time as the outcome. I was able to provide evidence that SEP at multiple points in the life-course, and at the individual and neighborhood level, contributes to the development of atherosclerosis. Additionally, I was able to document that a novel statistical technique such as latent trajectory class modeling could be utilized to identify distinctive clusters of the magnitude and changes in neighborhood poverty of the population over a twenty year period and then take those patterns to examine if they were related to cardiovascular risk.

Given the limitations of data collection and the complexities inherent in investigating the processes of places affecting health over the life course, and vice versa, the findings from these studies are remarkable. The independent contributions of childhood SEP, adult SEP, and neighborhood SEP to cardiovascular risk indicate that future analysis and interventions will need to take into account these life course and contextual nature of these exposures. Effects were not always equal by gender and race/ethnicity with more work needed to discover if these are true biological or behavioral differences or if it is the unequal distribution of SEP for these groups that is

explaining these differences. The findings from the second paper suggest that exposures to areas accumulate over time with higher levels of neighborhood deprivation equaling higher amounts of cardiovascular risk. The high degree of non-mobility in the study population limited our ability to look at the effects of upward and downward mobility. Future studies are needed that capture the residential mobility of younger age groups or with information over longer periods of time over the life course.

A large strength of this dissertation was the ability to provide evidence that contrary to conjectures in the literature, medical conditions are more likely to restrict mobility than lead to upward or downward mobility. Previous associations found between living in a disadvantaged neighborhood and poor health has been limited by the inability to separate the true contextual effects of an area from the selection of unhealthy people into disadvantaged neighborhoods. Results from this dissertation project indicate that if health selection is occurring; it is most likely to be an indirect result of the socioeconomic consequences of extreme weight or a general increased mobility of healthy individuals and not sick individuals moving to poorer neighborhoods. These results suggest that the associations between area deprivation and health are more likely to be a result of the areas in which people live affecting health rather than persons of ill health moving to socioeconomically deprived areas. Studies are needed to investigate if these findings are true across all age groups and time periods and to elucidate the processes linking health to residential, in this case, non-mobility.

Ideally, the investigation of life course effects will require longitudinal studies that include detailed measures of areas and individual-level characteristics over time, including sophisticated and detailed measures of socioeconomic position and

neighborhood environments. Because many of these processes begin in infancy, this will require the study of birth cohorts that track people as well as the environments they live in over time. Our results point to the need for such studies.

Cardiovascular disease is the number one killer in the US. It has been argued that modest changes in the one or more of the modifiable CVD risk factors could have a very large impact at reducing the occurrence of CVD in the population. Unfortunately, interventions aimed at changing the behavior of individuals and treatments focused on controlling risk factors after they have developed may have limited success without attention to social context broadly understood. My work shows that social context over the lifecourse both at the individual and neighborhood level is relevant to cardiovascular risk. I also demonstrate that selective migration of persons with cardiovascular risk factors into disadvantaged neighborhoods is unlikely to fully explain the associations of neighborhood factors with cardiovascular outcomes that we see. I am confident that this work will contribute to the growing body of literature that shows that SEP at multiple points in the life-course, and at the individual and neighborhood level, contributes to the development of atherosclerosis. But I also see it as influencing scientists and the public at large that prevention of CVD, and all diseases, is something that should occur at all points in the lifecourse and that the physical environment in which we inhabit may be just as, or more, important to public health as the individual health choices that we make.

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