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The Role of Social Structural and Social Contextual Factors in Shaping Chronic
Disease and Chronic Disease Risk Behavior: A Multilevel Study of Hypertension,
General Health Status, and Mental Distress

by

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A dissertation submitted in partial fulfillment
of the requirements for the degree of
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capital, health behavior, cardiovascular disease, self-reported health, mental
health, multilevel models

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Dedication

This work is dedicated to two very important people in my life, my husband, Beau McKay, and my Mom, Shirley Frishman. Without them, this work may never have been completed. To Beau, your faith in me carried me along when I could not carry the load alone, your irreverence helped me to laugh at myself and keep needed perspective, your constant curiosity reminded me there was a whole world outside my desk just waiting to be discovered and studied, and most of all, your love constantly inspires me to be the person I see when I look in your eyes. To Mom, your never-wavering belief that I could accomplish this has helped me through this process. I want you to know that I envision this work and what it represents as a part of a long line of women who believed in tomorrow – Ida to Clara to you to me.

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ABSTRACT

At present there is a reliance on behavioral interventions that have been limited in their effectiveness to reduce the public health burden of chronic disease, partly because the effects of social context on the initiation and maintenance of health behaviors is not incorporated into public health policy and practice. Yet current research indicates that there are macro-level structural and contextual influences on population health that *cannot* be reduced to individual or compositional effects. This study investigated the associations between social structural factors, community social context, individual characteristics, and self-reported correlates of disease. Distal influences included social structural inequalities such as income inequality and absolute deprivation or poverty. Pertinent mechanisms through which these influences might have operated on

disease included social contextual factors, such as social capital. Both political economy and the ecosocial perspective were selected to inform this study and to provide the theoretical framework from which hypotheses were derived.

The design was a multilevel, retrospective, nonexperimental study using secondary data. The study linked three data sources (2001 Behavioral Risk Factor Surveillance System, Social Capital Community Benchmark Study, and U.S. Census) by Federal Information Processing Standards codes in order for individuals to be placed in their community or state contexts. Results provided mixed evidence of the direct role of structural and contextual inequalities on self-rated health. Any direct effects of social structural inequalities on the health outcomes disappeared once individual factors were included in the models. Findings demonstrated that one dimension of social capital, organizational activism, retained its significant direct effect on general health status, once individual characteristics were considered. Conclusions suggested indirect associations whereby the negative influence of social structural inequalities on health was mediated by the erosion of social trust, which in turn was associated with engaging in risk behavior, thus increasing the odds of reporting hypertension, fair/poor general health, and mental distress. Although results were inconsistent, this study contributed to advancing Healthy People 2010 goals of increasing quality of life and reducing health disparities by advancing understanding of the multilevel nature of perceived health and the chronic diseases they predict.

CHAPTER 1: BACKGROUND AND SIGNIFICANCE

Introduction

Cardiovascular diseases (CVD) are the leading cause of death for most population groups in the United States. CVD comprise a cluster of diseases, including coronary heart disease and stroke, which together explain over 40% of all deaths annually; almost one million individuals die from CVD each year, with greater than half among women (American Heart Association, 2001). The burden that CVD place on the population is not just in terms of loss of life. For 2005, the costs of CVD are estimated to be \$393 billion (American Heart Association, 2004). Approximately one-quarter of the population lives with CVD. Although the human and economic costs of CVD are widespread, the diseases are not equally distributed throughout the population. There are well-documented disparities related to age, gender, race/ethnicity, SES and geographical location. The prevalence for the entire population was 354.1/100,000 in 1999, and subgroup-specific rates illustrate the disproportionate burden of CVD (American Heart Association, 2001). For example, rates among white males are 411.5 as compared to 526.0 among black males and 402.1 among black females, with the smallest rates for white women at 295.0 (American Heart Association, 2001).

Approaches to the prevention of CVD address the health problem at the individual level. The purpose of these strategies is to focus resources on those who suffer or are most likely to suffer from CVD – that is, persons who have a history of coronary heart disease, hypertension, smoking, inactivity, obesity or elevated cholesterol. Generally, individuals at greatest risk are targeted with resources to reduce the rates of disease in the small portion of the population who currently suffer the greatest burden of disease - the tail of the distribution (Rose, 1992). For example, the current National Heart, Lung, and Blood Institute (NHLBI) strategic plan is a high-risk prevention strategy in which the focus is on individual-level risk factors (behavioral and genetic) and a suggested intervention is medical management through better pharmaceuticals (National Heart, Lung, and Blood Institute, 2002). The primary limitation of this approach is that it does not reduce the overall burden of disease. This position is consistent with a biomedical perspective: it views risk in isolation from other factors; it focuses on behavioral or genetic contributors; it relegates responsibility for disease (here – CVD) squarely on the shoulders of the individual; and by labeling and focusing on volition to the exclusion of more fundamental factors, it implicitly reflects a “blame the victim” perspective.

Another implication of the biomedical approach is that it perpetuates a false Cartesian dualism, whereby the physical dimension of disease and the mental aspect are envisioned as representing two separate and distinct systems, only marginally related. Prevention of disease, therefore, is approached as

prevention of either physical or mental symptomatology and morbidity, not reflecting one individual's complete experience of pathology or illness. This bifurcated view has influenced primary, secondary, and tertiary prevention efforts. The preponderance of current prevention as well as treatment approaches are focused on the intra- and/or inter-individual factors, to the exclusion of contextual or structural considerations as fundamental causes. This perspective has served not to reduce the burden of disease, but rather to worsen it.

For example, mentally and physically healthy days have decreased from 53% in 1997 to 48% in 2001, with those reporting greater than half of each month unhealthy increasing from 15% in 1997 to 18% in 2001 (Zahran et al., 2005). The burden is economic as well, with costs of poor mental health reaching \$150 billion annually (Williams, Chapman, & Lando, 2005). In addition, rates reflect widening disparities in perceived poor health status by ethnicity, gender, socioeconomic status, and geographical location (Jia, Muennig, Lubetkin, & Gold, 2004; Sehili, Elbasha, Moriarty, & Zack, 2005; Zahran et al., 2005). The increase in prevalence of reporting poor general and mental health, along with the chronic diseases they often precede, reflects the ineffectiveness of these risk-factor prevention strategies that focus on proximal causes of disease, rather than directing attention to the persistent inequalities in resources that are the result of broader determinants of health (Link & Phelan, 1995; Link & Phelan, 1996).

Purpose of Study and Study Significance

The purpose of this study was to investigate the associations between social structural factors (e.g., income inequality), community social context (e.g., social capital), and individual characteristics (e.g., risk behavioral factors) and self-reported correlates of disease (hypertension, general health status, and mental distress). This study examined the extent to which upstream structural and contextual factors indirectly affect disease through their influence on risk behavior and the degree to which social structure and context independently influence self-reported disease.

The possibility that behavior only partially mediates relationship between context and disease has profound implications. Currently there is a reliance on behavioral interventions that have been limited in their effectiveness to reduce the public health burden of chronic disease, partly because the effects of social context on the initiation and maintenance of health behaviors is not incorporated into public health policy and practice. By adopting a society-and-health lens (Walsh, Sorensen, & Leonard, 1995), this study had the potential to extend our understanding of the multilevel nature of health disparities and the need for multilevel interventions to reduce them.

Study Rationale

Rose (1992) suggests that population-level prevention strategies that acknowledge the social determinants of health are much more effective in eradicating disease than high-risk, individually-based, acontextualized approaches. However, primary prevention is not just a question of selecting a level of analysis to target resources. Population-based strategies may be limited if they rely on high-risk population *behavioral* change, to the exclusion of contextual influence. There are macro-level structural and contextual influences on population health that cannot be reduced to individual or compositional effects – in essence, that which places people “at risk of risks” (Link & Phelan, 1995). Overall, macro-level features of society are posited to shape health through meso, or intermediary, factors which then differentially expose individuals to physical, social, and psychological contexts in which health promoting or health damaging behavior occurs (Berkman & Glass, 2000). Distal influences include social structural inequalities, such as income inequality, discrimination, and absolute deprivation or poverty. Pertinent mechanisms through which these influences may operate on disease include social contextual factors, such as social capital. The relative effects of these inequalities on risk behaviors and rates of self-reported health are not known. The extent to which these broader

social determinants shape disparate rates of multiple indicators of health and well-being, such as hypertension, general health status, or mental distress, is also unclear. As self-rated health is a strong predictor of future morbidity and mortality, it is critical to consider perceived health and rates of chronic disease as reflecting assessments of both physical and mental states of well-being (Williams et al., 2005).

When studying the social determinants of health, examining not just the differences between individuals within a population, but also differences between populations themselves, is critical. In essence, this view posits that health disparities reflect differential exposure and differential resources shaped by society at multiple levels. There is no single theory that encompasses all the underlying assumptions in this area of study. As no one theory satisfactorily explains the relationships that will be investigated, a hybrid of two approaches is selected to provide the theoretical framework from which hypotheses are derived. Both the political economy and the ecosocial perspective have informed this study.

Selected Explanations for Inequalities

Disparities in health may result from many mechanisms. Socially related origins of these differences include social structural and contextual influences that differentially affect certain groups. There are a variety of current

justifications for the disparate burden of disease. Theoretical and empirical evidence indicates that there is a range of explanations for social inequalities in self-reported health. Three of the most prominent rationales inform this study – those that attribute inequalities to material conditions, psychosocial factors, and health-related behaviors that confer biological risk (Marmot, Bobak, & Smith, 1995).

Material conditions refer to both relative and absolute deprivation. Income inequality can be considered a fundamental cause of the disparate burden of disease in that it reflects the economic and political institutions that generate and perpetuate inequality (Krieger, 2001). In essence, the effects of income inequality on health may be viewed as the physical consequences of structural power differentials and the resultant unequal distribution of resources (Doyal, 1995), as health and disease are socially produced (Turshen, 1989). Studies have shown that there are both direct and indirect effects of income inequality on health (Wilkinson, 1992; Kaplan, Pamuk, Lynch, Cohen, & Balfour, 1996; Kawachi, Kennedy, Lochner, & Prothrow-Stith, 1997; Kennedy, Kawachi, Glass, & Prothrow-Stith, 1998; Lynch et al., 1998)

An established pattern in public health evidence is the association between area-level poverty and health. In regards to material deprivation, one of the most consistent associations is that between health outcomes and growing up and/or living in poverty (Lynch & Kaplan, 2000). Although evidence supports a gradient effect (Adler et al., 1994), there is little debate that those living under

an absolute level of poverty have poorer health in general. The explanation for this disparity has both material and psychosocial aspects. Certain groups are usually dominant in the allocation of scarce resources and this structured inequality has a major impact on the health of less powerful groups (Doyal, 1995). Findings show that some effects of deprivation are contextual in nature above and beyond composition of the area (Macintyre, Maciver, & Sooman, 1993; Jones & Duncan, 1995).

Poor health results from subordination through social, political, and economic space to which some populations are relegated (Doyal, 1995). Evidence of this form of discrimination is seen in the health effects of residential segregation. Studies have found that residential segregation influences health through multiple pathways, including concentrated deprivation and the physical/social quality of the community (LaVeist, 1993; Williams, 1997). There have been few studies that have examined the role of physical and social isolation of a group with specific CVD-related health outcomes. However, there is substantial evidence that residential segregation negatively impacts mortality rates for some groups (Acevedo-Garcia, Lochner, Osypuk, & Subramanian, 2003).

There are collective community characteristics that may partly or fully mediate the relationship between social structural variables and risk behaviors and disease partly through their effects on psychosocial processes. Studies indicate that one mechanism through which social structural variables affect

health is social capital (Kawachi et al., 1997; Kawachi & Kennedy, 1999). Social capital refers to features of social organization, such as participation in associations and civic engagement, interpersonal trust, and norms of reciprocity, which act as resources and facilitate collective action (Putnam, 2000). Another potential influence of social context is racial trust. One health impact of the social experience of racism may be eroded trust in others. The experience of institutionalized discrimination may erode one's sense of connection and place in the community. Health effects of this may be engaging in risk behaviors related to stress, such as smoking and overeating.

Ultimately, the social patterning of health disparities reflects in part the social patterning of health behaviors as well as the social patterning of emotions (Emmons, 2000; Kubzansky & Kawachi, 2000). Evidence suggests that risk behaviors cluster (e.g., those who smoke often drink, those who have healthy dietary practices also tend to exercise). One can see how contextual forces differentially place certain groups "at risks of risks" (Link & Phelan, 1995). For example, there is evidence that lower socioeconomic status is associated with negative emotions and distress (Kubzansky & Kawachi, 2000) and there are substantial findings that those who are distressed tend to engage in risk behavior (e.g., smoke, overeat, do not exercise) that negatively impacts heart health. Because of the complex contribution of social structure and context to the ecology of most health outcomes, including CVD, there is some evidence that programs do not completely eliminate behavioral risk when it is focused upon in

isolation; there is a commensurate need to concentrate on contextual effects in addition to compositional effects.

Limitations of Existing Knowledge Base

At this time, there are three major gaps in the existing knowledge related to the role of the social environment in health. One major limitation in current empirical data is a lack of studies on the influence of social structure on CVD-specific outcomes. Most of what we know comes from studies on morbidity/mortality rates, life expectancy, and general health. Another deficiency is insufficient research on hypertension, general health status, or mental distress outcomes using data that is expressly collected to study the effects of social capital. Finally, there is a dearth of knowledge on the structural and contextual influences on risk behavior.

Preliminary Hypotheses

1. *Behavior only partially mediates associations between social structure and hypertension, general health status, or mental distress.*
2. *Social context partially mediates associations between social structure and hypertension, general health status, or mental distress.*

3. *Behavior only partially mediates associations between social context and hypertension, general health status, or mental distress.*

Overview of Design

The design is a multilevel, retrospective, nonexperimental study using secondary data. Until recently there has been a reliance on ecological studies in the study of macro-level social determinants of health. Primarily, cross-sectional ecological designs have been used. Multilevel modeling is utilized in this study for two reasons. First, a multilevel design is selected because of the nature of questions that will be investigated and the data that will be utilized. Different data sources representing differing levels of analysis are critical to this study as no one data set has specific multilevel data related to any outcomes in this study. This design is chosen also because it has multiple benefits over ecological approaches, including limits problems related to fallacies (ecologic, atomistic) and allows for the unique variance of contextual and compositional levels (e.g., to test whether income inequality and social capital effects on hypertension are significant while adjusting for individual-level factors, such as SES and individual health behaviors) (Subramanian, Jones, & Duncan, 2003).

In addition to having unique methodological features, this study advances our substantive understanding of inequalities and health. Few studies exist that explicitly address the effects of social capital and related variables on specific

health outcomes. At present, there are no studies that link individual-level health outcomes to a rich source of contextual data. The study links the BRFSS with the Social Capital Benchmark Study by Federal Information Processing Standards (FIPS) codes that are present in both – in this way, individuals can be placed in their respective community or state contexts.

Data Sources

The study employs three data sources: the 2001 Behavioral Risk Factor Surveillance System (BRFSS), the Social Capital Benchmark Study (SCCBS), and the U.S. Census. The BRFSS is an annual telephone survey administered by the CDC. The study's purpose is to collect information on the lifestyle and health behaviors of adults in the U.S that can be used to inform prevention policy and public health practice. Other measures include: sociodemographic variables, risk behaviors (BMI/diet, physical activity, smoking), and self-reported outcome variables (hypertension, general health status, mental distress).

Data on social contextual factors (i.e., social capital) have been obtained from the Social Capital Community Benchmark Study (SCCBS). The SCCBS is the first nationwide effort to measure social capital and its correlates (i.e., social trust and civic participation). The SCCBS was conducted in 2000 using both a nationwide and community-specific sample of adults. Variables of interest are social trust, formal and informal social engagement, and mutual aid, each of

which is measured through a structured survey administered by telephone via random digit dialing. Only 27 out of the 40 communities in the original study are used. Thirteen are omitted because the geography of the community could not be matched with Census data, did not have FIPS codes assigned, and therefore could not be linked to the BRFSS data or did not have BRFSS data for year 2001. Geographical units that are utilized include both counties and lightly populated states. The number of subjects in each community range from 89 to 4068.

Finally, data also are extracted from the 2000 Census. Specifically, measures of social structural inequalities (i.e., absolute and relative deprivation) are employed. Income inequality is calculated using the Gini Coefficient (Rogers, 1979; Kennedy, Kawachi, & Prothrow-Stith, 1996), which estimates the proportion of income above the mean that needs to be redistributed to approximate an equal distribution of incomes (Kawachi & Kennedy, 1997a). Area measures of poverty include the percent of families living at or below 200% of the Federal Poverty Level (FPL).

Implications for Public Health

Future implications of this study for public health include contributions to both research and practice. In a more general sense, evidence may demonstrate that interventions focusing on disparities in multiple health

outcomes should simultaneously address social and behavioral factors to inform service delivery and health policy. In regards to its influence on future studies, evidence from this work may promote the needed restructuring of large national surveillance systems to include contextual data. By broadening influential public health surveillance systems, the knowledge base from which interventions are developed and conducted for diverse populations regarding common behavioral risk factors for chronic disease (e.g., smoking, physical activity, dietary practices, substance use) will be expanded.

This study will provide a significant contribution to understanding the relationships between social structural, contextual, and behavioral aspects of self-reported health. If the social context within which behavior occurs is not considered, interventions targeting behavior change as a prevention strategy will have limited effectiveness. For example, future efforts would not be exclusively expended on changing proximal factors (i.e., individual health behaviors such as inactivity or smoking), but rather attention would be given to implementing social and structural changes. To reduce the disparate burden of CVD, intervention targets would include: instituting regulatory changes in political and economic policy which currently shape market influences which produce and perpetuate social inequalities (Kaplan & Lynch, 1999; Terris, 1999); strengthening social capital within communities (Kawachi, 1999) or perhaps directing prevention efforts towards developing community capacity (Elliott, Taylor, Cameron, & Schabas, 1998); developing models that are aimed at shaping local public

agendas to include community-level CVD prevention (Schmid, Pratt, & Howze, 1995; Finnegan, Viswanath, & Hertog, 1999).

In addition, findings from this study may inform policies focusing on improving individual *and* community-level general and mental health. Policy initiatives shaped by these results would target multiple levels of the social world, in order to reduce the disparate burden of poor mental and physical health status and its impact on perpetuating disparities in chronic disease. This study advances practice by informing surveillance and interventions focusing on the interrelated physical and mental dimensions of chronic disease prevention and health promotion. By advancing knowledge regarding perceived health and the chronic diseases they predict and by adding to the growing evidence base indicating health disparities reflect differential exposure and resources shaped by society, this study contributes to advancing Healthy People 2010 goals of increasing quality of life and reducing health disparities.

Delimitations

The following are delimitations of this study imposed by the researcher:

1. The study is limited to counties and lightly populated states – no locations are included where FIPS codes could not be assigned (e.g. cities, suburban or rural areas not identified by their county-specific locations).

2. The study includes only those counties that are represented in the 2001 BRFSS.
3. The study is limited to individuals from the 2001 BRFSS sample who reside in communities represented in the SCCBS.
4. The operationalization of CVD is restricted to self-reports of one critical form of cardiovascular disease, hypertension.
5. The operationalization of general health status and mental health are constrained to self-report measures.
6. Health risk includes a small selection of discrete behaviors and is restricted to poor physical activity, being overweight or obese, and engaging in smoking behavior.
7. The operationalization of health risk behavior is restricted to self-report measures.

Limitations

1. Individuals residing in communities in 1999 may be different than individuals residing in those communities in 2001.
2. The transience of residents between 1999 and 2001 may influence the social structure and social context of the community.
3. The investigation is restricted to the questionnaires, items, and survey techniques utilized in the BRFSS, SCCBS, and the Census.

4. The three levels of sampling used may result in somewhat different comparable sociodemographic characteristics of each community sample.
5. All prominent CVD risk behavior not included.
6. Although referred to as mental distress, no specific psychopathology is indicated.

Definitions

Absolute Deprivation – an area characteristic indicating the quantitative level of poverty; area-level SES.

Cardiovascular Disease (CVD) – a cluster of diseases, including coronary heart disease, hypertension, myocardial infarction, stroke, peripheral arterial disease, aortic aneurysm, and deep vein thrombosis.

Ecological – collective or group-level variables.

Ecosocial Perspective – an inherently multilevel theoretical framework in which the social production of disease view and biological and ecological perspectives are integrated; core constructs include embodiment and pathways of embodiment (Krieger, 1994, 2001).

Federal Information Processing Standards (FIPS) codes – federally designated unique numbers assigned to each county in each state within the United States.

Fundamental causes – persistent pathogenic social conditions that place individuals “at risk of risks,” are linked to various diseases through multiple

mechanisms, and are responsible for maintaining and perpetuating inequalities in health (Link & Phelan, 1995).

General Health Status – a self-reported assessment of one’s perceived qualitative level of overall health, with a range from excellent to poor.

Health-Related Behaviors – actions and activities of the individual that have health-associated consequences.

Income Inequality – qualitative and quantitative description of the dispersion or distribution or range of income in a population.

Informal Social Engagement - a dimension of social capital referring to a *collective* level of participation in familiar or casual relationships (e.g., with neighbors, co-workers, friends).

Macro-Level Factor – characteristics of the distal or broad social and/or physical environment.

Material Conditions – physical features of the environment, such as housing, assets, available services.

Mental Distress – a global estimate of perceived mental status (e.g., feeling depressed, anxious or stressed); not associated with Mental Status Exam.

Meso-Level Factor (aka Mezzo) – characteristics of the intermediary social and/or physical environment through which macro factors may influence individual health.

Mutual Aid - a dimension of social capital referring to a *collective* sense of shared or common assistance (e.g., volunteering, charitable contributions).

Obesity – excess body weight meeting U. S. Department of Health and Human Services (USDHHS) cutoffs (BMI \geq 30).

Organizational Activism - a dimension of social capital referring to a *collective* level of engagement in formal social relationships (e.g., participation in community groups, associations, or organizations).

Overweight – excess body weight meeting USDHHS cutoffs ($25 < \text{BMI} \leq 29.9$).

Psychosocial Factors or Processes – inter- and intrapersonal mechanisms through which the social environment “gets under one’s skin” (Taylor, Repetti, & Seeman, 1997).

Physical Activity – any non-work related activity that can be considered exercise.

Political Economy – a theory that posits that health and disease are socially produced; economic and political institutions and decisions that create, enforce, and perpetuate economic and social privilege and inequality are root or fundamental causes (Turshen, 1989; Doyal, 1995); the political economy (the national economy in interaction with governmental policies) influences a nation’s health through the mechanisms of production, distribution, and consumption (Brenner, 1995).

Poverty – absolute standard of area-level deprivation.

Relative Deprivation – an area-level characteristic indicating qualitative (comparative) differences in income within a population.

Residential Segregation – a multidimensional construct representing the differentiation and spatial distribution of two or more groups within a population of

an area (Massey & Denton, 1988; Massey, White, & Phua, 1996; Acevedo-Garcia & Lochner, 2002).

Risk Behavior – individual behaviors known to influence the pathogenesis of chronic disease, such as eating patterns, physical inactivity, and smoking.

Smoking – active tobacco smoking.

Social Contextual Factors – a characteristic of the collective; features of the social environment, such as neighborhood or community factors, which may link distal influences to individual health outcomes.

Social Capital – features of social organization, such as participation in associations and civic engagement, interpersonal trust, and norms of reciprocity, which act as resources and facilitate collective action (Putnam, 2000).

Social Determinant – elements of the social environment that influence (e.g., health).

Social Patterning – the way in which factors are distributed and arranged by sociodemographic groups.

Social Structural Factors – broad conditions of a society, such as inequality, discrimination, and poverty that shape the nature of intermediate and proximal factors.

Social Trust – a dimension of social capital referring to a *collective* sense of faith or confidence in bonds with others (e.g., with neighbors, coworkers, police).

CHAPTER 2: CONCEPTUAL FRAMEWORK AND REVIEW OF LITERATURE

Review of Literature

Theoretical Framework

The two orientations that form the broad theoretical framework of this study are the social structural and ecological perspectives. Specifically, political economy and the ecosocial approach are selected. Each provides a unique view as well as forms a complementary perspective informing this study, which reflects the author's epistemological stance of the multilevel nature of the social world.

In general, sociological theories in health focus on the social rates of disease, which reflect a group or population-based perspective. Social structural perspectives consider the structural barriers that restrict people from living healthy lives. Political economy theory examines the physical consequences of structural power differentials and the resultant unequal distribution of resources (Doyal, 1995). Health and disease are socially produced; disease/health is a function of the relative power of different groups (Turshen, 1989). Fundamental causes of the disparate burden of disease stem from economic and political institutions that generate and perpetuate social and economic inequality (Krieger, 1994, 2001). The political economy (the national economy in interaction with governmental policies) influences a nation's health through the mechanisms of

production, distribution, and consumption (Brenner, 1995). The focus of disease shifts from the host or individual to social classes defined in relation to production and the way production is organized; stress is largely a response to capitalist social relations (Turshen, 1989). Certain groups are usually dominant in the allocation of scarce resources and this structured inequality has a major impact on the health of less powerful groups; poor health results from subordination through social, political, and economic space to which some populations are relegated (Doyal, 1995). The applicability and appropriateness of using this theory for this work is that it focuses on the social production of disease, an implicit assumption in studies of social inequalities in health. However, inherent weaknesses in using this type of perspective are that it does not provide for the role of agency (e.g., the role of individual behavior) and that it may focus more on materialist explanations for social inequalities in health rather than the effects of relative deprivation.

Some of the limitations of political economy theory are reduced by including an ecological orientation, which does not restrict its view to broad features and is more inclusive of individual-level factors. Specifically, the ecosocial approach (Krieger, 1994), although not a theory per se, is considered an ecological theory-in-development as it does have a coherent set of (complex) propositions linking the social production of disease with biology in a dynamic ecological analysis. The ecosocial approach is appropriate for this study in that it is explicitly multilevel in its focus on “current and changing patterns of [social

inequalities in] health ...in relation to each level of biological, ecological and social organization (cell, organ, organism/individual, family, community, population, society, ecosystem)” (Krieger, 2001). Also, the ecosocial approach incorporates the notion of embodiment (how the social world gets under our skin to influence our biology) with the pathways of that process (similar to the political economy perspective).

There are several strengths and weaknesses of this perspective. For example, its comprehensiveness is a limitation and an advantage – on one hand, one may ask what does this theoretical position *not* explain, but future efforts at improving the formalization will increase its specificity. On the other hand, the comprehensiveness of the ecosocial approach is a plus, as it more adequately recognizes and seeks to explain the nested or multidimensional order of social reality, which due to methodological advances (e.g., multilevel modeling), can be rigorously tested. A considerable advantage of the approach is its applicability to a wide range of data from public health to education to environmental and political science. Currently, this theoretical approach is being applied in social science and epidemiology and is in a period of reflection, revision, and refinement (i.e., scientific self-regulation). Support for utilizing this view, specifically in regards to this study, includes that the perspective comprehensively explains the multileveled nature of the world. In addition, the approach incorporates social production of disease with biological expressions of inequality (i.e., there is a place for individual agency in this perspective).

Constraints of this view include that it is not a coherent theory. Moreover, the ecosocial approach has been limited in its use in empirical studies, primarily due to lacking in precision and insufficient evidence of its predictive power.

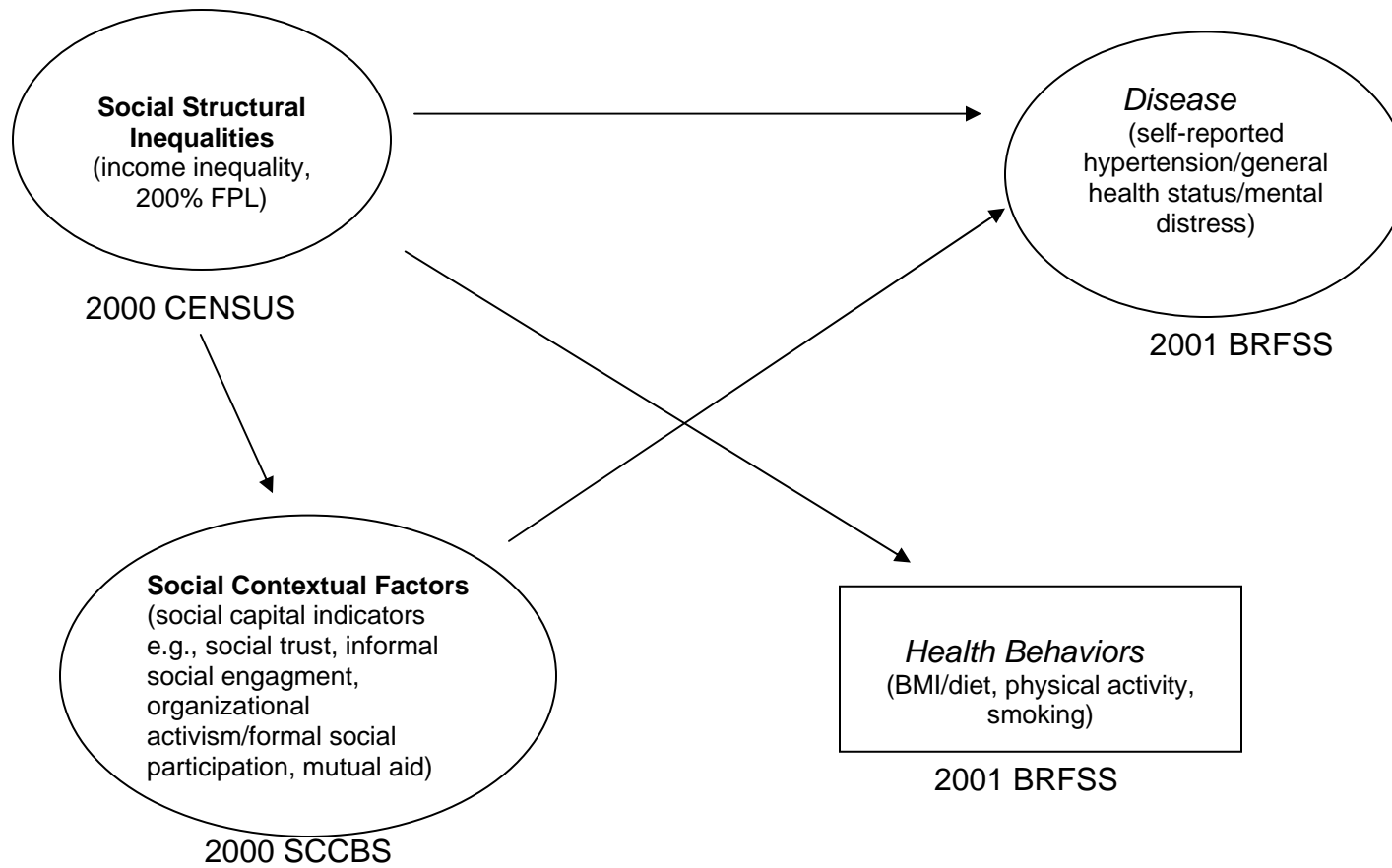
Although either the ecosocial perspective or political economy might individually be suitable for this study, the most appropriate use is an integration of the two, which utilizes the strengths of each while reducing the limitations of both. These two theoretical frameworks differ in their emphasis on features of society and biology and how they incorporate the two (Krieger, 2001).

Essentially, utilizing both fits best and is most consistent with the questions asked in this study.

Literature Review

This research is framed by the theoretically-driven conceptual model (Figure 2.1), which is based in ecosocial and political economy perspectives. In essence, this model proposes that disease and risk behaviors are not shaped solely by proximal individual-level factors. Rather, individual agency exists in a social context, which itself is driven by broader structural determinants of health. In following this model, the literature review presents evidence linking social structural inequalities, such as income inequality and poverty, specifically to CVD, general health status, mental distress, risk behavior, and the social context

Figure 2.1 Conceptual Model



within which it occurs. Evidence is presented that social context is directly associated with disease as well as indirectly through its shaping of behaviors (such as physical activity, obesity, and smoking), which have been causally linked to CVD.

On the whole, very little of the current literature establishes causal relationships between the variables. Some of this may be due to the multilevel nature of the relationships and the methodological challenges that imposes. Ecological studies demonstrate the associations between broader factors and health, but only in the past few years have methodological advances enabled researchers to examine these relationships with causality in mind. Multilevel modeling has permitted ecological and atomistic fallacy to be less of a threat to validity and therefore has allowed inferences to be made and alternative explanations to be ruled out in working with individual and contextual levels of analysis simultaneously. Nevertheless, these advances have not allowed this literature to approximate causality, due to the dearth of longitudinal designs and studies of the effect of time.

Temporal ordering of effects has not been systematically studied in most of the literature pertinent to this study. Timing has been considered only in regards to the relationship between health behavior and CVD and it is in that literature only that causality has been established. To remedy this limitation, a new stream of work encompassing lifestage and timing of transitions has begun. In sum, the association between macro factors and health is established and competing explanations for results found have been ruled out due to

methodological advances; however, few, if any, studies have looked at the effects of temporal ordering.

Social Structural Factors and Health

Relative Deprivation: Income Inequality

There is a vast and growing literature on the influences and consequences of the positive association between income inequality and health inequalities, although very little of it has been able to definitively establish temporal precedence of relative deprivation, as many designs have been cross-sectional in nature. Even so, most of the current literature implicates the role of social structural factors in creating environments that are pathogenic. Although not conclusive, there is substantial evidence from both ecological and multilevel studies of the relationship between income inequality and health.

Some studies have found mixed evidence for the relative deprivation hypothesis that suggests it is the distribution of income across a population, not just the absolute level of deprivation, which accounts for individual health and health inequalities (Blakely, Atkinson, & O'Dea, 2003; Hou & Chen, 2003; Osler et al., 2003). However, many studies have addressed the issue of whether relative deprivation impacts health over and above absolute or individual deprivation by demonstrating associations even after accounting for area-level poverty as well as SES in their models (Kennedy, Kawachi, Glass, & Prothrow-

Stith, 1998). In doing so, these investigations have served to rule out possible alternative explanations.

Consistent results demonstrate that this social structural factor has both direct and indirect effects for a host of health-related issues, such as CVD (Kennedy et al., 1996; Waitzman & Smith, 1998b; Diez-Roux, Link, & Northridge, 2000; Cooper, 2001; Mellor & Milyo, 2003), all-cause and premature mortality (Ben-Shlomo, White, & Marmot, 1996; Kaplan, Pamuk, et al., 1996; Kennedy et al., 1996; Kawachi & Kennedy, 1997a; Lynch et al., 1998; Cooper et al., 2001; Lochner, Pamuk, Makuc, Kennedy, & Kawachi, 2001; Lobmayer & Wilkinson, 2002; McLaughlin & Stokes, 2002; Sanmartin et al., 2003), self-rated health (Kennedy, Kawachi, Glass et al., 1998; Soobader & LeClere, 1999; Fiscella & Franks, 2000; Kahn, Wise, Kennedy, & Kawachi, 2000; Subramanian, Kawachi, & Kennedy, 2001; Blakely, Lochner, & Kawachi, 2002; Weich, Lewis, & Jenkins, 2002; Subramanian & Kawachi, 2003; Lopez, 2004), mental distress (Fiscella & Franks, 2000; Kahn et al., 2000; Weich, Lewis, & Jenkins, 2001; Shi, Starfield, Politzer, & Regan, 2002; Muramatsu, 2003), life expectancy (Wilkinson, 1992), STI/AIDS (Holtgrave & Crosby, 2003), crime (Kawachi, Kennedy, & Wilkinson, 1999), and teen birth rates (Gold, Kennedy, Connell, & Kawachi, 2002).

Although both individual-level and aggregated-level outcomes have been examined, the majority of outcomes have been group-level due to the methods with which these studies were conducted. The reliance on findings from ecological designs has resulted in debates regarding both conceptual as well as empirical limitations in the literature.

Current reviews of literature identify several possible issues that may influence the detection of an association between income inequality and health, including concerns for lag effects, scale, design, and confounding (Lynch et al., 2004; Subramanian & Kawachi, 2004). In regards to matters of scale, strength of associations between income inequality and various outcomes may be dependent upon the geographic level in which income inequality is assessed. Overall, in regards to U.S. studies, there is stronger evidence of an association when income inequality is measured at the state level and more debate regarding the relationship at a smaller scale (i.e., counties, tracts, block groups) (Lynch et al., 2004; Subramanian & Kawachi, 2004).

Issues related to design and the possible role of confounders are connected. There is a serious gap in the literature related to confounding. Several individual and ecologic confounders may influence the detection of the association between income inequality and health (Subramanian & Kawachi, 2004). Pertaining to design issues, as the majority of studies are ecological, cross-sectional investigations, there have been concerns regarding the validity of interpreting associations between broader, structural factors as reflecting real influences for individual health. This issue has resulted in much discussion over whether the effects of income inequality are a statistical artifact - representing results that are ecologically fallacious by ascribing the effects found to alternative sources of variance (Gravelle, 1998).

Some have countered this argument with evidence that the relationship between income inequality and rates of mortality do not reflect a fundamental

relationship between individual-level characteristics and mortality and therefore are not explained by artifact (Wolfson, Kaplan, Lynch, Ross, & Backlund, 1999). In point of fact, the current use of multilevel modeling has added to the empirical evidence supporting the independent contextual effects of income inequality on health by adjusting for cross-level confounding.

In essence, the emergence of multilevel studies has served to expand the previous methodological restrictions in making conclusions regarding income inequality. Results from these works have allowed the contextual influence of income inequality, as compared to the compositional influence of SES, to be firmly established as a critical consideration of social structural influences on health.¹ Although findings have been consistent from these types of studies, there is still a gap in the literature related to multilevel studies of specific diseases. One example is the dearth of multilevel investigations of CVD. Only one study has investigated specific CVD risk and its relationship with income inequality in a multilevel study (Diez-Roux, Link, & Northridge, 2000).

Although there have been investigations into the association between income inequality and CVD-related mortality, there are far fewer studies of CVD-related health behavior (Kaplan et al, 1996). At this time, no studies have examined the effects of income inequality on both CVD risk behavior and rates of CVD simultaneously. Generally, most multilevel studies of income inequality examine its association with mortality or self-reported health.

¹ Subramanian, Kawachi, & Kennedy (2001) provide the most accessible explanation of these terms: “contextual (the difference a place makes) and the compositional (what’s in a place)” p.10

Although there are studies which have found no association between income inequality and health status (Mellor & Milyo, 2002, 2003), there is by far more literature from multilevel studies supporting this relationship. Findings suggest that those individuals residing in areas with highest inequality are up to 30% more likely to state they have fair/poor health (Kennedy, Kawachi, Glass et al., 1998; Blakely, Lochner, & Kawachi, 2002) with a growing income inequality increasing those reporting fair/poor health by up to 39% (Subramanian & Kawachi, 2003). More recent research indicates that this association may be stronger, with evidence that a one point increase in the Gini Coefficient (on a 100 percent scale) corresponds to a 4% (1.6% – 6.5%) increase in reporting fair or poor health (Lopez, 2004), with income inequality becoming a progressively more important influence as self-rated health deteriorates (Shi & Starfield, 2000; Lopez, 2004). Moreover, the influence may be differential based on gender (Kahn, Wise, Kennedy, & Kawachi, 2000), individual SES (Subramanian, Kawachi, & Kennedy, 2001; Weich, Lewis, & Jenkins, 2002), and geographic scale at which inequality is measured. Multilevel studies have demonstrated a strong association between self-rated health and income inequality at various levels of aggregation, including state (Kawachi, Kennedy, Lochner, & Prothrow-Stith, 1997; Kennedy, Kawachi, Glass et al., 1998; Subramanian et al., 2001), region (Weich et al., 2002), metropolitan area (Blakely, Lochner, & Kawachi, 2002; Lopez, 2004) and county (Soobader & LeClere, 1999).

Income inequality has been shown to have a detrimental impact on other forms of self-reported health. However, the body of literature as a whole is far

smaller for mental health/distress and demonstrates more mixed evidence than studies examining general health alone. For example, while some findings support a harmful influence of income inequality on mental distress (Kahn et al, 2000; Weich, Lewis, & Jenkins, 2001; Muramatsu, 2003), others either find no such association, or the association disappears after other contextual or individual-level factors are added to the model (Weich, Twigg, Holt, Lewis, & Jones, 2003; Muntaner et al, 2004). Few studies find no evidence of a significant independent association between income inequality and mental health outcomes (Sturm & Gresenz, 2002; Henderson, Liu, Diez Roux, Link, & Hasin, 2004).

In spite of these results, slightly more studies report a positive relationship, with those living in higher income inequality areas having up to a 70% excess risk of suffering with depressive symptomatology (Fiscella & Franks, 2000; Kahn et al., 2000; Weich, Lewis, & Jenkins, 2001). Amongst studies which find a health damaging impact of living in high income inequality region, there is evidence of an additional moderating influence of absolute poverty in shaping the strength and directionality of the relationship. Although results are significant, they have substantively contrasting interpretations. For example, some suggest that the negative influence of living in a state with high levels of income inequality on mental distress is more profound if one has higher income (Weich et al., 2001), while other studies have indicated that it is lower incomes which confer a worse impact on mental distress (Kahn et al., 2000).

In addition to the evidence base regarding the direct, independent association between income inequality and health outcomes, several studies

have found the relationship to be mediated by social factors, such as the social context (Kawachi et al, 1997; Kennedy, Kawachi, Prothrow-Stith, Lochner, & Gupta, 1998; Fiscella & Franks, 2000; Gold et al., 2002; Veenstra, 2002) and neighborhood processes (Soobader & LeClere, 1999) as well as access to health resources, such as primary care (Shi, Starfield, Politzer, & Regan, 2002). In addition, there is limited evidence of interactional association between income inequality and minority concentration (McLaughlin & Stokes, 2002) as well as residential segregation (Cooper et al., 2001). However, substantial evidence finds that income inequality exerts significant independent effects on health (Kaplan, Pamuk, Lynch, Cohen, & Balfour, 1996; Kennedy, Kawachi, & Prothrow-Stith, 1996; Soobader & LeClere, 1999; Gold, Kawachi, Kennedy, Lynch, & Connell, 2001; Lochner, Pamuk, Makuc, Kennedy, & Kawachi, 2001).

Absolute Deprivation: Poverty

The association between area-level material or absolute deprivation and morbidity and mortality is well established, with evidence of a growing gradient effect (Singh & Siahpush, 2002). As with relative deprivation, causality has not been established as few, if any, studies have been able to suggest temporal ordering. Both qualitative (Cattell, 2001) and quantitative studies have found significant positive associations between area-level poverty and a variety of health outcomes, such as CVD (Diez-Roux et al., 1997; Davey Smith, Hart, Watt, Hole, & Hawthorne, 1998; LeClere, Rogers, & Peters, 1998; Cubbin, Hadden, &

Winkleby, 2001; Huff & Gray, 2001; Davey Smith & Hart, 2002; Singh & Siahpush, 2002; Cohen, Farley, & Mason, 2003), mortality (Ben-Shlomo, White & Marmot, 1996; Waitzman & Smith, 1998a, 1998b; Yen & Kaplan, 1999a; Singh & Siahpush, 2002; Cohen et al., 2003), self-rated health or quality of life (Robert, 1998; Malmstrom, Sundquist, & Johansson, 1999; Cattell, 2001; Ross & Mirowsky, 2001; Steptoe & Feldman, 2001; Drukker & van Os, 2003), mental distress (Aneshensel & Sucoff, 1996; Yen & Kaplan, 1999b; Elliott, 2000; Schulz, Williams et al., 2000; Belle & Doucet, 2003; Leventhal & Brooks-Gunn, 2003; Ferrer & Palmer, 2004), teen birth rate (Gold et al., 2002), residential instability and neighborhood violence (Sampson, Raudenbush, & Earls, 1997) and STI/AIDS (Holtgrave & Crosby, 2003). Although all these associational investigations provide empirical support for the contextual influence of poverty, there are two pressing issues currently debated, both of which impact future studies of possible causality. The first one involves methodological approaches (i.e., use of multilevel methods). The second relates to conceptual issues (e.g., differential influence of poverty as contextual rather than reflecting individual-level SES effects and the differential influence of poverty and income inequality).

A primary issue of both methodological and conceptual concern has been how and to what extent poverty at the contextual level is important above and beyond the effects of individual SES alone. Multilevel methods have allowed this issue to be investigated empirically. Overall, results demonstrate that the negative effects of deprivation are not a proxy for individual characteristics and, in fact, reflect a true contextual feature of the social structural environment (Diex-

Roux et al., 1997; Robert, 1998; Waitzman & Smith, 1998a; Ross & Mirowsky, 2001; Sundquist, Lindstrom, Malmstrom, Johansson, & Sundquist, 2004).

In regards to study designs, although many studies have not used multilevel designs explicitly, they have statistically adjusted for area-level measures in an attempt to predict individual risk through proportional hazards models and therefore reduce the risk of alternative explanations. In doing so, these studies come closest to suggesting that there may be a causal relationship. Results from these studies are consistent and indicate a contextual pathogenic effect of living in poverty on CVD (Davey Smith, Hart, Watt, Hole, & Hawthorne, 1998; Waitzman & Smith, 1998a, 1998b; Huff & Gray, 2001; Davey Smith & Hart, 2002). However, the adoption of multilevel studies in this area of research has produced a growing literature to support the already established finding that the socioeconomic environment one lives in has both direct and indirect effects on mortality. Specifically in regards to CVD risk, some have found an independent influence of absolute deprivation of an area (Davey Smith et al., 1998; Robert, 1999; Cubbin, Hadden, & Winkleby, 2001; Sundquist et al., 2004). Additionally, research specifically examining the area effects of poverty provide evidence that risk of MI is partly explained by context and therefore only somewhat accounted for by composition of an area (Stjarne et al, 2002). However, it must be noted that as timing of effect has not been thoroughly investigated, causality has not been established.

Findings suggest both independent and mixed effects. For instance, a direct association is found for CHD (Diez-Roux et al., 1997) as well as mortality

Waitzman & Smith, 1998a, 1998b; Yen & Kaplan, 1999a, Bosma, van de Mheen, Borsboom, & Mackenbach, 2001). Indirect associations exist as well, but there is a debate in the literature as to what extent individual factors, such as SES, moderate (Jones & Duncan, 1995; O'Campo, Xue, Wang, & Caughy, 1997; Yen & Kaplan, 1999a) or mediate (Drukker & van Os, 2003) the relationship. As of yet, there is insufficient evidence to confirm either type of association over the other. Overall, however, research indicates that there would be a 20% reduction in mortality rates in the U.S. if all groups had the same rates as those living in the highest SES areas, controlling for a host of individual factors (Winkleby & Cubbin, 2003).

Evidence supports a direct association between poverty and self-reported health status. International as well as national multilevel findings provide support for the adverse health effects of deprivation on self-rated health. There are both direct and indirect effects of living in a deprived area on self-rated health (Ross & Mirowsky, 2001), with some findings indicating a moderating relationship between contextual and individual levels of deprivation (Ferrer & Palmer, 2004). Moreover, in regards to mediating effects, both qualitative and quantitative evidence suggests that the influence of poverty on health status may be partially through its impact on social context (Cattell, 2001; Ross & Mirowsky, 2001; Drukker & van Os, 2003).

Overall, evidence of the negative impact of living in poverty on health status is strong. Findings come from a variety of studies, with the majority of recent evidence from multilevel investigations. For example, from a longitudinal

cohort study, there is evidence of a higher risk of reporting fair/poor health (OR 3.30, 95% CI 2.32 – 4.71) up to nine years later associated with living in poverty (Yen & Kaplan, 1999a). In addition, results indicate that living in absolute deprivation increases the odds by a range of 70% – 200% of reporting fair/poor health (Malmstrom, Sundquist, & Johansson, 1999; Steptoe & Feldman, 2001).

A significant positive association exists between residing in an impoverished area and general mental distress (Schulz, Williams, et al., 2000; Leventhal & Brooks-Gunn, 2003) as well as specific disorders, such as depression and anxiety, in a variety of demographic subgroups. There is some evidence that women (Belle & Doucet, 2003) and adolescents (Aneshensel & Sucoff, 1996) appear to be at greater risk. Studies have found that poverty increases the risk of experiencing mental distress, with residing in deprivation conferring over two-times (OR 2.14, 95% CI 1.49-3.06) the risk of developing poor mental health nine years later (Yen & Kaplan, 1999b). Some suggest it is the exposure to stressful, disadvantaged conditions (Schulz et al., 2000; Steptoe & Feldman, 2001), restricted protective resources (Elliott, 2000) and neurochemical responses which result in excess morbidity for those who live in poverty (Pearlin, 1989; McEwen, 1998; Ferrer & Palmer, 2004). Although this literature is limited, it is developing a strong evidence base, as provided by both multilevel (Steptoe & Feldman, 2001) and RCT (Leventhal & Brooks-Gunn, 2003) studies.

The other conceptual issue considers the differential influence of poverty compared to the impact of other structural factors. One study found that poverty

acts as a mediator through which residential segregation influences health (LeClere, Rogers, & Peters, 1998). One review suggests that there are independent effects of community deprivation on other structural or contextual factors such as the social, service, and physical environment (Robert, 1999). In regards to income inequality, some studies that have explicitly examined the combined effects of absolute and relative income on mortality (Ben-Shlomo, White, & Marmot, 1996; Kaplan, Pamuk, Lynch, Cohen, & Balfour, 1996; Kennedy, Kawachi, & Prothrow-Stith, 1996). Nevertheless, few have studied the impact on specific health outcomes (Gold et al., 2002; Holtgrave & Crosby, 2003). At this time, simultaneous examinations of the relative effects of poverty and income inequality on the social context of risk behavior and CVD, general health status, or mental distress have not been conducted. This gap in the literature results in competing explanations between contextual influences, thus inhibiting causality to be confirmed or denied.

Social Structural Factors and Health Behaviors

Currently, the evidence base to support direct associations between various structural factors and health behaviors is sparse, with no studies explicitly examining the temporal relationships between macro factors and individual behavior. However, the small literature that exists indicates that the relationship between social structural inequalities and disease may be partly mediated by health behavior. As contrasted with income inequality, the vast majority of

studies have examined the role of poverty. To date, no studies have investigated both relative and absolute deprivation and health behavior, and therefore alternative explanations cannot be ruled out. This gap may be due, in part, to little evidence on the impact of income inequality on behavior, as some studies have controlled for behavioral and sociodemographic factors (Kennedy et al, 1998).

However, recently, stronger evidence has accumulated. For example, in a multilevel study, Diez-Roux et al (2000) have found significant positive associations between income inequality and three prominent CVD risk factors - BMI, hypertension, and sedentarism, adjusting for individual SES. In addition, these authors found moderating effects of gender and absolute income levels. To specify, there was stronger evidence of the relationships for all risk behaviors for women as well as a more robust positive association between income inequality and risk behavior for those living at lower income levels. Other studies have not examined a cluster of risk behavior, but rather only one or two behaviors at a time. Findings illustrate that increases in weight are positively associated with income inequality at the state level, especially for men, (OR 1.12, 95% CI 1.03 - 1.22) (Kahn, Tatham, Pamuk, & Heath, 1998). In addition, smoking and physical activity (Kaplan, Strawbridge, Cohen, & Hungerford, 1996) as well as sexual risk activity (Thomas & Thomas, 1999) are positively related to relative deprivation.

Compared to the limited literature on relative deprivation and risk behavior, there are, on the whole, many more studies on the effects of poverty on

health behavior. There is a large scientific base on the effects of individual SES on risk behavior, but very little on area-level economy or poverty and behavior. In general, there are a small number of studies of the association between poverty and singular risk behavior. Most look at a cluster of risk, unlike the income inequality literature, and virtually none explore causal relationships. Both ecological and multilevel studies have found strong evidence supporting the negative impact of area-level poverty on behavior, while adjusting for individual sociodemographic factors. In controlling for extraneous factors, these findings support the structural influence of material deprivation above and beyond individual status on such CVD risk factors as physical activity, BMI/obesity, and smoking (Cubbin, Hadden, & Winkleby, 2001).

Results indicate that not only is there a significant negative association between poverty and physical activity, but also that there may be a dose-response effect between area factors and activity (Parks, Housemann, & Brownson, 2003). Although this specific finding has not been replicated, the inverse relationship between absolute deprivation and physical activity cannot be questioned. The majority of support has come from cross-sectional studies (Wister, 1996; Lantz et al., 1998; Ross, 2000b; Cubbin et al., 2001; Lee & Cubbin, 2002), although there have been multilevel investigations as well (Ross & Mirowsky, 2001). To date, the only multilevel studies examining the relationship of poverty on combined effects of physical activity, BMI/obesity, smoking, or excessive alcohol use have been international. Results from these studies demonstrate an increased risk of engaging in smoking, being sedentary, and

obese for those who reside in impoverished areas (Sundquist, Malmstrom, & Johansson, 1999; Bosma, van de Mheen, Borsboom, & Mackenbach, 2001).

The increased risk of obesity for those who live in poverty has been well-established (Davey Smith, Hart, Watt, Hole, & Hawthorne, 1998; Cubbin et al., 2001). In most cross-sectional studies, the associations found are both for independent (Lee & Cubbin, 2002) as well as mediated effects (Lantz et al., 1998). There are two similarities between the physical activity and obesity literature regarding the impact of poverty. Firstly, just as with physical activity, a dose-response relationship has been found between poverty and obesity, with the former measured by environmental proxy. Specifically, (Reidpath, Burns, Garrard, Mahoney, & Townsend, 2002) find that those who reside in impoverished areas are exposed to 2.5 times the density of fast food outlets than those who live in more affluent areas. The second commonality is that the only multilevel studies of obesity as one of a cluster of risk factors are from other nations (Reijneveld, 1998; Sundquist, Malmstrom, & Johansson, 1999).

By and large, the majority of studies examining the associations between poverty and risk behaviors pertain to the negative effects of smoking, where evidence has come from qualitative (Bancroft, Wiltshire, Parry, & Amos, 2003), cross-sectional (Krieger, 1992), and multilevel findings (Sundquist et al., 1999; Bosma et al., 2001). Risk related to area deprivation and smoking has been found in both the U.S. (Lantz et al., 1998; Cubbin et al., 2001) and Europe (Davey Smith & Hart, 2002). On the whole, results reveal a strong relationship between poverty and smoking, with living in disadvantaged areas conferring a

50% to 110% increase in odds of smoking, controlling for individual SES (Diez-Roux et al, 2003). An essential aspect of this literature is separating out area versus individual influences that hinder or facilitate smoking. In regards to this health-damaging behavior, evidence exists for the detrimental effects of neighborhood deprivation accounting for residential segregation, individual status (Kleinschmidt, Hills, & Elliott, 1995) as well the moderating effect of race/ethnicity (Diez-Roux et al., 1997; Cubbin et al., 2001).

Overall, as one can see from the above discussion, the role of social structural inequalities on CVD risk behavior is strong, even though the study of area-level absolute and relative income on risk behaviors is quite new. However, one caveat is in order in interpreting the findings as the majority of empirical evidence is from studies from other nations. Even so, one issue cannot be questioned – the distribution of income is reflected in the distribution of cardiovascular risk (Diez-Roux, 2003).

Social Structural Factors and Social Context

While there is support of a direct association between social structural inequalities and health, no studies examine a possible causal relationship. Nonetheless, evidence exists that some of the effect occurs through the social context. There have been several key pathways elucidated in current literature that make the conceptual and methodological connection between social structure and social context in regards to public health. Generally, two schools of

thought have emerged in the literature regarding the mechanisms through which income affects health.

One group proposes that the pathway is essentially materialist in nature, primarily reflecting political changes, class or economic relations, and material conditions or resources that often accompany poverty (Kaplan, Pamuk et al., 1996; Lynch et al., 1998; Lynch, Smith, Kaplan, & House, 2000). The other view posits that although social structure's relationship with health is related to material deprivation, these conditions are not sufficient; the association is also due to the deleterious effects on social context through relative deprivation (Wilkinson, 1992; Wilkinson, 1996) and social organizational processes (Marmot & Wilkinson, 2001). Fundamentally, the expansive effect of income inequality serves to corrode the social fabric of communities through such factors as increased violence, reduced civic participation, and reduced productivity (Kawachi & Kennedy, 1997). It is this perspective that informs this study.

Generally, social structure is posited to affect health through social contextual pathways such as disinvestments in forms of human and social capital as well as psychosocial mechanisms (Kawachi, 2000). Other mechanisms linking macro to meso ecological factors include physical and social characteristics of residence (MacIntyre & Ellaway, 2000). The specific aspects of the social context that have been addressed in the relative and absolute deprivation literature are, by and large, social capital, collective efficacy, and the social environment overall.

One potential pathway by which social structural variables, such as income inequality, shapes health is through its effects on social capital (Kawachi et al., 1997; Kawachi & Berkman, 2000; McCulloch, 2003). To note, this pathway does not reflect the health benefits of individual social networks. Rather social capital is a contextual construct, impacting health through characteristics of the collective, such as levels of trust, reciprocity, civic participation (Putnam, 2000). A seminal work by Kawachi et al (1997) has found that relative deprivation at the state-level may lead to disinvestments in many forms of capital, including social, with associations as strong 0.46 to 0 .76. Some studies demonstrate that the group experience of income inequality serves to erode relational resources critical to health, such as mutual trust and civic participation (Daly, Duncan, Kaplan, & Lynch, 1998).

Most studies have been ecological, cross-sectional investigations of associations, although there have been a few multilevel investigations, primarily in other countries, which have adjusted for some alternative explanations. Currently, the majority of the public health literature identifies social capital as a mediator in the relationship between broader, structural factors and health. For example, there are cross-sectional studies of the mediating effects of this variable on a variety of health outcomes, including mortality (Kawachi et al, 1997), violent crime (Kennedy, Kawachi, Prothrow-Stith, Lochner, & Gupta, 1998), and teen birth rate (Gold et al., 2002).

There have been far fewer studies on the associations between constructs related to social capital and macro factors affecting public health. To date,

income inequality and social context have not been rigorously studied with respect to chronic disease. Just as with income inequality, one consistent observation in the social structural literature is that there is little evidence due to few empirical studies explicitly examining the associations between poverty and contextual factors such as collective efficacy, social cohesion, social environment and chronic disease and risk behavior in adults, even though the association between residing in poverty and poor rates of social cohesion has been established (Coleman, 1988).

There is more evidence on the direct effects of poverty on health as compared to income inequality effects (Gold et al., 2002), where much of the literature has found indirect effects mediated through social context. Although there is more evidence regarding poverty's association mediated through collective efficacy, social cohesion, or social environment, recently some support has been found specifically on the mediating effects of social capital on the relationship between absolute deprivation and health (Cattell, 2001; Steptoe & Feldman, 2001; Gold et al., 2002; Holtgrave & Crosby, 2003). There is, however, a substantial literature on indirect effects from both multilevel and ecological studies on poverty and health, mediated by collective efficacy (Sampson et al., 1997; Cohen, Farley, & Mason, 2003), social cohesion (Drukker et al, 2003), and social environment (Ross & Mirowsky, 2001). In all these studies, attempts have been made to control for competing explanations for findings.

Overall, living in poverty results in non-random exposure to pathogenic environments and restriction of salubrious resources (Lynch & Kaplan, 2000).

Consensus from multilevel studies has demonstrated a mediating effect whereby exposure to physically and socially deprived areas influence health above and beyond individual sociodemographic factors. Even as both income inequality and poverty reflect structural factors, the relationship between each and the social context as a mediating force is quite different. In regards to absolute deprivation, the mechanism of influence may be more related to the experience of poverty in regards to social processes, such as stress and health behaviors (Elliott, 2000; Bosman et al., 2001; Pickett & Pearl, 2001; Ross & Mirowsky, 2001; Steptoe & Feldman, 2001) or material deprivation (Kaplan, Pamuk et al., 1996; Lynch et al., 1998). At this time, more evidence of the relative effects of structural and contextual characteristics on disease and its processes is needed. Despite these advances in studying how the broader social structure shapes the social environmental context in which health or disease occurs, there is a gap in the literature investigating associations between these characteristics and specifically CVD (Diez Roux, 2003), especially in relation to the temporal ordering of effects.

Social Context and Health

As most of what is known regarding social capital and its correlates comes predominantly from cross-sectional studies, there is no evidence supporting causality as yet. There are, nonetheless, a number of investigations pertaining to the association of health and other contextual aspects of the social world, such

as social cohesion, social stress and the social environment (Turner, Wheaton, & Lloyd, 1995; Schulz, Israel et al., 2000; Ellaway, Macintyre, & Kearns, 2001; Steptoe & Feldman, 2001; Drukker & van Os, 2003). Although the number of studies investigating the health effects of social capital is relatively small, evidence is mounting of the impact of this aspect of the social environment. Consistent with its contextual nature, regional differences in indicators of social capital have been found (Cattell, 2001; Steptoe & Feldman, 2001). Associational relationships have been observed, with several studies controlling for extraneous factors. For example, social capital has been associated with a wide variety of outcomes in public health, including CVD mortality (Kawachi et al., 1997; Franzini & Spears, 2003; Lochner, Kawachi, Brennan, & Buka, 2003), overall mortality (Kawachi et al., 1997; Franzini & Spears, 2003; Lochner et al., 2003; Skrabski, Kopp, & Kawachi, 2003), self-rated health (Macintyre, Maciver, & Sooman, 1993; Kawachi, Kennedy, & Glass, 1999; Blakely, Kennedy, & Kawachi, 2001; Cattell, 2001; Subramanian et al., 2001; Subramanian, Kim, & Kawachi, 2002; Greiner, Li, Kawachi, Hunt, & Ahluwalia, 2004), mental distress (McCulloch, 2001; Mitchell & LaGory, 2002; Campbell, Cornish, & McLean, 2004; Greiner et al., 2004; Ziersch, Baum, Macdougall, & Putland, 2005), violence (Kennedy, Kawachi, Prothrow-Stith et al., 1998; Hemenway, Kennedy, Kawachi, & Putnam, 2001; Galea, Karpati, & Kennedy, 2002), STI/AIDS (Holtgrave & Crosby, 2003), quality of life (Raphael et al., 2001), and teen birth rates (Gold et al., 2002). Investigations have shown that variation in area social capital do not solely reflect

differences in individual factors, but rather support social capital as a contextual, rather than individual-level, construct (Franzini & Spears, 2003).

Currently, there are few studies that have examined the relationship between social capital and cardiovascular disease (CVD). Seminal studies of social capital have been cross-sectional and ecological in nature investigating the mediating influence of social capital on all-cause and coronary heart disease (CHD) mortality at the state-level (Kawachi et al., 1997). Indicators of social capital, such as group membership and social trust, have independent contextual associations with CVD mortality, adjusting for other contextual factors, such as income inequality and poverty or material deprivation (Kawachi et al., 1997; Lochner, Kawachi, Brennan, & Buka, 2003).

Recently, multilevel studies have provided evidence of the unique contextual influence of social capital on CVD mortality while controlling for extraneous factors at differing levels of analysis. Lochner, Kawachi, Brennan, & Buka (2003) replicated earlier findings of the significant association between social capital and CVD mortality, and extended our understanding of indicators of social capital by measuring aspects of social cohesion at smaller, substantively meaningful levels of analysis (i.e., neighborhoods). Additionally, research indicates that the association between social capital and CVD mortality occur at multiple levels of the social environment, such as neighborhood and county levels, after the effects of individual characteristics are taken into account (Franzini & Spears, 2003).

In regards to the health effects of indicators of social capital on individual measures of health, such as self-rated health (SRH), evidence suggests that the influence is contextual rather than compositional in nature (Macintyre et al., 1993; Kawachi, Kennedy, & Glass, 1999; Blakely et al., 2001; Subramanian et al., 2001). Results supporting these conclusions have been found in both ecological, or macro-level, as well as multilevel studies. Therefore, criticisms regarding the validity of these findings (e.g., ecological fallacy) for individual-level outcomes such as SRH are rare. The two most pressing issues at this time are in what manner should social capital be conceptualized and measured and to what extent the influence of social capital on health is moderated by individual level factors.

Although social capital has been envisioned as an aspect of the individual (Portes, 1998), the majority of social, political, and health scientists concur with its inherent contextual essence. What is less clear and less consistent is the way in which social capital is viewed as a characteristic of the collective having multiple dimensions (e.g., structural vs. cognitive aspects, bridging vs. bonding, participatory vs. perception). At this time, there are few studies of self-rated health in which multiple aspects of this construct (participation in voluntary organizations, social trust, mutual aid or reciprocity) are examined simultaneously (Kawachi, Kennedy, & Glass, 1999; Greiner et al., 2004). Typically, studies address only one dimension, usually social trust, as a single proxy for this multidimensional construct. Given this limitation in the literature, there is a significant association between social capital and general health status,

with up to 40% greater odds of reporting fair/poor health if one resides in a community with low forms of social capital after adjusting for a variety of possible confounders (Kawachi, Kennedy, & Glass, 1999; Blakely et al., 2001; Jun, Subramanian, Gortmaker, & Kawachi, 2004; Rohrer, Arif, Pierce, & Blackburn, 2004).

The other issue concerns the possible moderators of the social capital and self-rated health association. Parallel to the level of analysis debate in the poverty-health literature, a debate has evolved regarding if and to what extent contextual-level effects of social capital are moderated by individual-level correlates (e.g., social networks, social support). Because of the abovementioned restriction in the current evidence base, few multilevel studies have examined the probable role of cross-level interactions between individual and contextual characteristics. For example, in examining the possible moderating influence of individual-level correlates of social capital on the contextual level influence of social capital, one study found that social capital may be associated with good self-reported health only for those individuals who are trusting themselves (Subramanian, Kim, & Kawachi, 2002). In general, more studies are needed on conceptual clarity and multilevel interactions to reach a consensus regarding the relationship between social capital and general health status.

As with other specific health outcomes, there have been few empirical studies on the association between social capital and mental distress. Less specifically, there is evidence that aspects of the social environment, such as

inadequate social cohesion (Aneshensel & Sucoff, 1996; Ellaway, Macintyre, & Kearns, 2001) and related social processes (Dressler & Badger, 1985; Ross, 2000a; Schulz, Williams et al., 2000; Leventhal & Brooks-Gunn, 2003), are deleterious contextual influences on mental health outcomes. By and large the literature on this area as well as, more expressly, social capital is conceptual by nature (Kawachi & Berkman, 2001; McKenzie, Whitley, & Weich, 2002; Sartorius, 2003). Many debates abound regarding social capital and mental distress, some of which center around validity issues. Just as in the self-rated health literature, the most pressing matters at this time include conceptualization, definition, and measurement concerns. In this area, social capital has been characterized as having multiple dimensions: structural and cognitive, bridging and bonding. Although social capital is more frequently envisioned as a characteristic of the collective, there are some who maintain that this construct reflects both individual and community-level dimensions of social relatedness.

There is evidence to support both, although some question whether using individual reports of social networks and social support really is social capital at all, but rather testing individual resources only. Given these concerns, both qualitative (Campbell, Cornish, & McLean, 2004; Ziersch, Baum, Macdougall, & Putland, 2005) and quantitative evidence still exists that living in areas with little social connectedness, trust and engagement confers excess risk of mental distress (Mitchell & LaGory, 2002; Greiner et al., 2004; Ziersch et al., 2005), with almost a doubling of the odds of suffering from psychiatric morbidity (OR 1.96,

95% CI 1.39-2.75) for those living in areas with low social capital (McCulloch, 2001).

As discussed above, there is a limited, yet strong body of evidence regarding the health effects of social capital. However, as social capital can be seen as an aspect of social cohesion (Kawachi & Berkman, 2000), the findings regarding the association between correlates of the social environment and health will be reviewed to lend additional support for the relationship. In current literature, conceptually related constructs are collective efficacy, social cohesion, and the social environment.

Research on the health effects of collective efficacy includes substantial evidence demonstrating the mediating and moderating effects of this social construct on a range of health outcomes, such as CVD mortality (Cohen, Farley, & Mason, 2003), SRH (Browning & Cagney, 2002), neighborhood violence and residential instability (Sampson et al., 1997). Studies of area differences in collective efficacy report improved indicators of health in communities or neighborhoods where this aspect of social cohesion is strongest. Consistent with evidence from other studies, in a recent review, Sampson (Sampson, 2003) encourages using social context as a critical unit of analysis in studies of the health effects of the social environment, such as collective efficacy.

In contrast to the preponderance of quantitative studies, Fullilove (1998) used data from a qualitative study to examine the relationship between social cohesion and health in four communities. Findings were consistent with other studies, which indicated that promotion of social cohesion improves community

health. Evidence pertaining to general health suggests, just as with most social capital studies, that social cohesion *mediates* the relationship between broader structural environment (e.g., area deprivation) and individual perceptions of health (Macintyre et al, 2000; Drukker et al, 2003).

Most studies investigating the consequences of social location on health have studied a wide variety of aspects of the social environment. For this study, pertinent literature includes those studies that have focused on CVD-related outcomes. Area influences include such factors as urban/rural setting (Barnett & Halverson, 2000; Ewing, Schmid, Killingsworth, Zlot, & Raudenbush, 2003) racial/ethnic influence or composition (Barnett & Halverson, 2000; Franzini & Spears, 2003; Reidpath, 2003), and female concentration (LeClere et al., 1998). The majority of these studies were multilevel, where individual level sociodemographic influences have been controlled. This provides additional support for area or contextual nature of the social world and its effects on CVD morbidity and mortality. What is less known is the direct and indirect effects of a particular aspect of the social world, social capital, on specific CVD-related health and behavior. From the above review, one can see that there is a need for additional evidence of the health effects of social capital from multilevel studies, investigating its potential independent and cross-level associations, from data that is expressly intended to measure and investigate social capital.

Social Context and Health Behaviors

Overall, there is a very small literature on the influences of social context on health behavior, with most focusing on school environment in childhood and adolescence. By and large, this early phase in the literature of this area examines the relationship between social context and health behavior, without systematic study of alternative explanations or temporal ordering as yet. This status is due, in part, to the decontextualized manner in which most health promotion/behavior change studies and interventions are created, with little concern of the features and influence of place (MacIntyre & Ellaway, 2000). The reliance on intraindividual determinants of behavior to the exclusion of broader societal influences, which construct or shape risk behavior (Backett & Davison, 1995), is seen in most studies of chronic disease including CVD. Just as the physical environment either inhibits or facilitates health behavior, so does the social or collective characteristics of the community affect individual action (Sorensen et al., 2003). The environment provides opportunities or barriers for agency. A primary way in which the environment “gets under one’s skin” and influences the physiology and pathology of CVD is through its effect on risk behaviors, such as physical activity, diet/obesity, and smoking (Taylor et al., 1997; Kawachi & Berkman, 2000).

In regards to exercise, social contexts characterized by weak social cohesion are associated with reduced physical activity. Support for this

relationship comes from literature examining both the social environment in general and social capital in particular. The vast majority of research of environmental influences on physical activity focuses upon the physical context in which activity occurs and in this way does address timing of effect. Overall, these studies find that the most pressing factors include accessibility, opportunity, weather, safety, and aesthetics (Humpel, Owen, & Leslie, 2002). Comparatively, the influence of the social environment on physical activity is less well understood, primarily because behavior has been viewed in a myopic manner – completely under volitional control of the individual – without concern for the way in which the social context impacts opportunity, agency, and choice. The limitations of this literature demonstrate the critical role of the social environment on physical activity. Although most studies are quantitative, there has been one qualitative study that explored the differential effects of the social environment on activity (Burton, Turrell, & Oldenburg, 2003).

Generally, the majority of studies examine both physical and social aspects simultaneously (Brownson, Baker, Housemann, Brennanm & Bacak, 2001; Giles-Corti & Donovan, 2002; Lee & Cubbin, 2002; Ewing et al., 2003). By doing this, these studies were able to control for some alternative explanations from both environmental and individual influences. Findings suggest that there is a positive association between socially cohesive environments and physical activity with the physical locale necessary but not sufficient in shaping physical activity (Giles-Corti & Donovan, 2002). Other studies found that it is not just the personal factors such as enjoyment and preferences that shape activity levels,

but the attributes of the collective, such as social disengagement, which negatively influence health behavior (Brownson, Baker, Housemann, Brennan, & Bacak, 2001; Salmon, Owen, Crawford, Bauman, & Sallis, 2003). Although there is limited knowledge regarding the effect of social context on behavior, the emerging consensus is that there are multiple dimensions of the environment that exert influence on physical activity (Macintyre, 2000; Sallis, Kraft, & Linton, 2002).

At this time, there is no clear agreement or debate regarding the effects of social capital on physical activity, primarily because there are so few studies that have examined these relationships. In a Swedish prospective cohort study, Lindstrom, Hanson, & Ostergren (2001) examine the psychosocial conditions that may help to explain group differences in physical activity. The authors found that social capital, defined as social participation or engagement in social, civic, or political activities including formal and informal associations, predict behavior. Specifically, those living in socially disengaged areas had over twice the odds (OR 2.2, 95% CI 1.8 – 2.7) of low physical activity, with little differential effect by individual factors. Although in a follow-up study the authors find some contrary evidence of a contextual effect (Lindstrom et al, 2003), these conclusions may be called into question because the way in which social capital has been measured is not consistent with current definition and usage. Therefore, the results of the original study that inadequate social capital (possibly through provision of social norms) is a mechanism explaining group differences in physical activity are a more robust finding.

The only other study investigating social capital and activity was also from a European nation. An Irish study examines the associations between neighborhood design and social capital (Leyden, 2003). Positive associations are found between social capital and physical activity. However, the author conceptualizes the relationship differently in terms of directionality. He concludes that there are higher levels of social capital in walkable neighborhoods – essentially that physical activity improves social cohesion. Because there are no other studies examining this association, the causal pathway between these two variables cannot be presumed with any certainty. However, the association in general cannot be questioned.

As compared to the literature on exercise, there are an even more limited number of investigations of area effects on obesity. Essentially, a deprived social environment is associated with poor dietary habits, specifically obesity. Another similarity between the physical activity and obesity literature is that most studies have looked at the impact of both the physical and social environment on risk behavior in order to adjust for alternative explanations. One commonality of the work in this area is how recent the studies are, with most empirical investigations published only in the last two years. In general, there is a positive association between area of residence and obesity (Ellaway, Anderson, & Macintyre, 1997; Ecob & Macintyre, 2000; Morland, Wing, & Diez Roux, 2002), with explicit evidence of the impact of a weakened social context and poor diet and obesity (Lee & Cubbin, 2002; Giles-Corti & Donovan, 2003; Vandegrift & Yoked, 2004). Specifically, there is an excess odds (OR 1.6, 95% CI 1.1-2.3) of being

overweight or obese for those residing in socially disorganized environments, controlling for individual demographic and other health behaviors (Caitlin et al, 2003). Other studies find that urban sprawl (including social disengagement) is positively associated with obesity (Ewing et al., 2003; Vandegrift & Yoked, 2004), partly through its effects on physical activity.

Although there is a growing literature on the social contextual influence of place on obesity, there is little examination of the relationship specifically between social capital and associated risk behavior. Currently, there is only one study of the role of social capital in obesity. This European work comes from the CVD literature and posits that social capital should be an essential consideration of an obesity prevention strategy aimed at reducing the prevalence of hypertension (Worsely, 2001).

Other health behaviors implicated in the etiology of CVD include smoking. Just as with the other risk factors, this area of study is quite new, with this specific literature the smallest of all. Findings indicated that living in a poor social environment is positively associated with smoking, although the effects of timing and extraneous variables have not been well studied. Recent qualitative studies found that the daily social environment one resides in assists or impedes smoking through contextual influences (Poland, 2000; Bancroft et al., 2003). The few quantitative studies that have been conducted provided findings consistent with these conclusions (Ecob & Macintyre, 2000; Diez Roux, Merkin, Hannan, Jacobs, & Kiefe, 2003).

Also consistent with the physical activity and obesity literature discussed, the evidence of impact of social capital on smoking is recent, limited, and predominantly comes from international studies. Findings from these investigations demonstrated a significant positive association of weak social capital on increased smoking, with strongest evidence on daily smoking (Lindstrom & Ostergren, 2001; Lindstrom, 2003). One such study found a positive relationship between correlates of social capital, such as voting behavior – an indicator of social engagement - and smoking (Kelleher, Timoney, Friel, & McKeown, 2002). Although the literature regarding social capital effects on CVD-related health behavior is incomplete, there are studies of its impact on other forms of behavior, such as sexual behavior and STD/HIV (Thomas & Thomas, 1999; Crosby, Holtgrave, DiClemente, Wingood, & Gayle, 2003), environmental risk (Wakefield, Elliott, Cole, & Eyles, 2001), and alcohol use (Weitzman & Kawachi, 2000).

Overall, there are three commonalities amongst this literature: preponderance of international investigations, clustering of risk behaviors, and the studying of the relationship between risk and social context is still in its infancy and therefore unable to establish if a causal relationship exists. When studying the initiation and maintenance of risk behavior, it is critical to not just focus on the proximate causes of such activity, but the more contextual influences that shape the environment within which risk occurs. Ultimately, the environment confers opportunity or barriers to engage in behavior. Although most studies examining the broader environmental context have looked at the impact

of the physical environment, there is evidence that a commensurate (Salmon et al., 2003; Vandegrift & Yoked, 2004) need exists to address related social environmental factors as well (Ford, Ahluwalia, & Galuska, 2000) when investigating social context on CVD risk factors such as physical activity, obesity, and smoking.

Health Behaviors and Hypertension

In general, there is a large knowledge base on the associations between health behavior and CVD. In point of fact, most of the information regarding the etiology of CVD comes from studies investigating individual factors. The majority of public health prevention efforts have targeted these proximal causes or associations to combat the increase in their prevalence, which then leads to increased rates of CVD and other chronic diseases. In regards to this study, the pertinent behavioral risk factors include physical inactivity, obesity, and smoking. Commonalities across the literatures linking these behaviors to CVD include the impact of individual-level moderators (e.g., age, gender, race/ethnicity, SES) and the role of clustering of risk. There are differences in the literatures as well. For example, some of the earliest work on behavioral risk is in regards to the deleterious effects of smoking on CVD, whereas obesity and physical activity studies are comparatively more recent. Overall, evidence for all three factors as casually related to CVD is robust; there is a large literature based on cohort, case-control, and randomized control studies from which to draw conclusions.

Physical Inactivity

Physical activity is associated with improved health. Initial studies suggest that vigorous exercise was the only way in which to obtain cardiorespiratory fitness. It has been subsequently ascertained that, at this time, the benefits of any form of regular exercise include a reduction in risk for a wide variety of chronic diseases, including CVD. Overall, these benefits consist of lowering the risk of: heart disease, development or worsening of hypertension, and premature mortality from CVD (USDHHS, 1996). Although the type (i.e., vigorous, moderate, light) and amount (i.e., minutes per week) are still being studied, the general recommendation for adults is 20 - 30 minutes or more of moderate activity at least 5 days a week to obtain optimum health benefits. Nevertheless, findings demonstrate that as little as 10 or more minutes a day has significant reduction in premature CHD mortality (Leon, Myers, & Connett, 1997). Although this recommendation is widely known, the majority of adults are physically inactive, with women, lower SES, African-American and Hispanic, and older individuals having higher rates of inactivity (USDHHS, 1996).

Up until relatively recently, the majority of findings on physical activity and CVD have been based on white men (USDHHS, 1996). Currently evidence on the impact of exercise on CVD in women has essentially paralleled the benefits to men (Oguma, Sesso, Paffenbarger, & Lee, 2002). Findings suggest reduced CVD (HRR 0.64, 95% CI 0.42-0.97) is associated with increasing activity levels

for older women, even adjusted for other risk factors such as smoking, BMI, and comorbidities (Gregg et al., 2003). These general conclusions have been replicated on middle-aged women as well (Owens, Matthews, Raikonen, & Kuller, 2003).

Additional evidence of the relationship between physical inactivity and CVD come predominately from cohort studies in which a gradient effect has been consistently demonstrated (USDHHS, 1996). On the whole, the effects of physical inactivity on this form of chronic disease are commonly divided into two classes – one on the effects on CVD in general and the other group of studies on CHD in particular, with a more limited number of investigations specifically on hypertension (USDHHS, 1996). In regards to CVD risk as a whole, evidence indicates an inverse dose-response effect with physical activity (Kannel & Sorlie, 1979; Kannel, Belanger, D'Agostino, & Israel, 1986; LaCroix, Leveille, Hecht, Grothaus, & Wagner, 1996; USDHHA, 1996). Kaplan et al (1996) finds that there is a significant protective effect of physical activity on CVD mortality (RR 0.81, 95% CI 0.71-0.93). Others have found a similar independent relationship, even after accounting for age, sex, BMI, and smoking (LaCroix, Leveille, Hecht, Grothaus, & Wagner, 1996). There are parallel findings with respect to CHD. Studies have shown that risk of CHD is inversely associated with exercise (Blair, 1994; USDHHS, 1996), with an overall RR ranging from 1.21 to 1.8 of CHD for inactivity (Paffenbarger, Wing, & Hyde, 1978; Kannel et al., 1986; Berlin & Colditz, 1990).

In regards to the pertinent pathways of effects, regular exercise influences some of the biological mechanisms associated with CHD – hypertension, dyslipidemia, obesity, and endothelial health (McKechnie & Mosca, 2003). In concordance with these findings, most of the literature proposes the physiological pathways to CVD include physical activity's beneficial effects on blood pressure, atherosclerosis, ischemia, levels of plasma fibrinogen, and plasma viscosity (Gordon & Scott, 1991; Leon & Connett, 1991; USDHHS, 1996; Lindstrom, Hanson, & Ostergren, 2001).

To specify, in regards to high blood pressure, evidence posits that exercise benefits both normotensive as well as hypertensive adults (Whelton, Chin, Xin, & He, 2002). Although there is support of moderating effects for race/ethnicity and gender, there is commensurate evidence of the independent effects of physical activity on the prevalence of hypertension (Bassett, Fitzhugh, Crespo, King, & McLaughlin, 2002). In addition, the progression of atherosclerosis is attenuated by regular aerobic activity (Nordstrom, Dwyer, Merz, Shircore, & Dwyer, 2003). Further, evidence suggests that those who are least physically active have a 30% greater risk of developing hypertension (USDHHS, 1996).

In general, patterns of findings demonstrate that physical activity and CVD (overall), CHD, or hypertension reflect a robust inverse gradient effect. By and large, findings indicate that the burden of CVD could be greatly decreased by increased physical activity. For example, regular exercise could reduce or prevent the 13.5 million who have CHD, the 1.5 million who suffer heart attacks,

and the 50 million who have hypertension in a year as well as positively impact the approximately 60 million (one-third of the U.S. population) who are overweight (USDHHS, 1996). Prevention efforts would also need to take into consideration that physical inactivity and obesity often co-occur (USDHHS, 1996; Blair & Brodney, 1999) with combined negative effects on CVD mortality (Fang, Wylie-Rosett, Cohen, Kaplan, & Alderman, 2003).

Obesity

Overweight and obesity result in excess risk for many forms of CVD (USDHHS, 1996, 2001; NIDDKD, 1998) in both men and women (Hu, 2003). Support for this causal relationship is found in multiple types of studies including quantitative (e.g., multiple cohort, RCT, longitudinal and cross-sectional; NIH, 1998), qualitative cross-cultural (Treloar et al., 1999), as well as reviews of the independent effect of obesity on more prominent forms or correlates of CVD such as hypertension, dyslipidemia, CHD, CHF (Labarthe, 1998; NIDDKD, 1998; Rashid, Fuentes, Touchon, & Wehner, 2003; Sowers, 2003) with additional evidence on increased risk even for coronary thrombosis (Wolk, Berger, Lennon, Brilakis, & Somers, 2003). The effects of obesity-related morbidity and mortality from CVD are well known, however only recently have studies demonstrated that there are effects not just for absolute weight, but weight gain as well.

There is support for the effect of excess weight in increasing CVD mortality, with exponential effects as weight increases (Meyer, Sogaard, Tverdal, & Selmer, 2002; Rogers, Hummer, & Krueger, 2003). For example, in a longitudinal cohort study, investigators find negative effects of both weight and weight gain on CVD risk for young adults (Norman, Bild, Lewis, Liu, & West, 2003). There is supplementary evidence on the deleterious health effects of weight gain (Willett et al, 1995). Additionally, Kawachi (1999), in a review, presented evidence that an increase of approximately 11-17 pounds in adulthood confers 25% excess risk of suffering from CHD, with risk increasing as weight increases (Galanis, Harris, Sharp, & Petrovitch, 1998; NIDDKD, 1998).

The disparities in prevalence of CVD may be due, in part to the differential effects of obesity on CVD risk by gender and race/ethnicity (Patt, Yanek, Moy, & Becker, 2003) as well as age and SES (USDHHS, 2001). Clearly, obesity is a multi-determinant risk factor with severe health consequences in and of itself, however the current literature has begun to focus more on how this feature impacts and is impacted by other forms of CVD risk behavior. Although there is a large evidence base for the direct effects of obesity on CVD (NIDDKD, 1998) and a growing literature on the combined effects of physical activity and obesity on CVD-related outcomes, currently, there is no clear consensus as to the directionality of effect between behaviors.

What is certain is the mounting evidence of clustering – those who are obese or overweight are less physically active by and large, with support for an inverse dose response between exercise and obesity (USDHHS, 1996, 2001).

For example, in a study by (Kannel, Wilson, Nam, & D'Agostino, 2002), the authors have found that the clustering of risk factors associated with being overweight or obese occurs in well over half the individuals, resulting in increasing RR for CHD for men (2.07) and women (10.9). Along with physical activity, findings indicate obesity and smoking co-occur in studies of CVD (Millen et al., 2002), with additional evidence that engaging in both behaviors compounds risk of CVD (NIDDKD, 1998). In point of fact, some suggest that morbidity due to obesity is as large as that from smoking (Sturm & Wells, 2001).

Smoking

Compared to the other behavioral risk factors (i.e., physical inactivity and obesity), smoking has by far the longest and strongest evidence base for a causal relationship with CVD. The link between smoking and CVD has been established in the scientific literature since the 19th century (USDHHS, 1983). In essence, smoking has been studied from a variety of designs, which have established its causal role in the development and progression of CVD. Empirical investigations, meta analyses, and reviews concur that most forms of CVD are affected including CHD, hypertension, arteriosclerosis, aortic aneurysm, peripheral vascular disease (USDHHS, 1983, 2001; Labarthe, 1998; Burns, 2003).

The knowledge base, however, is not equal amongst all groups. The majority of studies up until the past two decades have used men only in their

samples. More recent examinations of smoking effects on CVD for women find just as robust associations. For example, women smokers have a significantly greater risk of CHD (Mann, James, Wang, & Pickering, 1991; Kawachi et al., 1994) as well as hypertension (USDHHS, 1980; Mann et al, 1991).

There are numerous traditional mechanisms by which smoking confers physiological damage. Smoking causes weakening of vessel walls and exacerbates or hastens atherosclerosis and atherosclerotic lesions (USDHHS, 1983, 2001). In addition, nicotine and other toxins from smoking increase blood pressure and heart rate, resulting in an imbalance of oxygen supply to the myocardium, platelet aggregation and function, and related pathogenic insults to the cardiovascular system such as thrombosis, hemorrhage, and vasoconstriction (USDHHS, 1983).

Recent findings include studies branching from a focus on traditional effects of smoking on pathology to more novel factors, which are involved in CVD risk. For example, the physiological responses to smoking implicated in development of disease include increasing cholesterol and blood pressure (USDHHS, 2001). Currently, investigators find a dose-response association between smoking and other, less investigated biochemical processes such as elevated serum C-reactive protein, fibrinogen, and homocysteine levels (Bazzano, He, Muntner, Vupputuri, & Whelton, 2003). In addition to biochemical effects, there is evidence of smoking's structural damage implicated in the etiology of atherosclerosis (Pittilo, 2000; Burns, 2003).

In essence, much is known about the independent effects of smoking. However, there is a gap in the information regarding its effect on and with other forms of risk. To fill this space in knowledge, a growing aspect of this literature relates to the clustering effect of behavioral risk. It is not clear in current literature the relative and/or interactive effects of these behavioral risk factors (physical activity, obesity, and smoking) on the development of CVD. What is apparent is that, to some extent, all three are related in their associations with and prevention of CVD (e.g., a reduction in CHD is associated with not smoking similarly to the benefit of regular physical activity, which is causally linked to reduced rates of obesity) (USDHHS, 1996). There are many ways in which these CVD risk factors may be linked including physiological, psychological, and social factors.

Health Behaviors and General Health Status

Compared to the CVD literature, there are relatively fewer studies that examine the influence of health behavior on self-reported general health status, even though self-rated health is a well-established predictor of future morbidity and mortality. While most studies examine the role of individual risk behavior, such as smoking, obesity, or inactivity, a few studies simultaneously examine the role of multiple risk behaviors in independently predicting self-rated health, both

internationally (Manderbacka, Lundberg, & Martikainen, 1999; Abu-Omar, Rutten, & Robine, 2004; Froom, Melamed, Triber, Ratson, & Hermoni, 2004) and nationally (Brown et al., 2003; Strine et al., 2005).

Physical Inactivity

The evidence of the health benefits of physical activity comes from both cross-sectional and prospective studies. There is mounting evidence of both the direct as well as mediated influence of exercise on self-reported health. Findings suggested that the influence of health behaviors on self-rated health is mediated by health problems and functional limitations (Manderbacka, Lundberg, & Martikainen, 1999). Results demonstrated a salubrious influence of physical activity on general health status (Hassan, Joshi, Madhavan, & Amonkar, 2003; Atlantis, Chow, Kirby, & Singh, 2004), with regular recommended levels of exercise conferring health benefits from young adulthood through to old age (Brown et al., 2003). Longitudinal investigations have found that infrequent exercise predicts poor self-rated health in men (OR 1.67, 95% CI 1.04-2.17) more than 7 years later (Froom, Melamed, Triber, Ratson, & Hermoni, 2004).

Most of the studies of the role of physical activity in general health status evaluations have treated this behavior as either indicating regular activity, insufficiently active or inactive. More recently, a study examining three critical aspects of exercise (intensity, frequency, and duration) finds that insufficient activity levels doubled the likelihood of reporting poor health more than half the

month (OR 2.02, 95% CI 1.85-2.21), controlling for a range of sociodemographic and behavioral factors (Brown et al., 2004).

Obesity

Body weight influences both physiological as well as psychological aspects of self. Excess weight can harm health through its role in the etiology of many chronic diseases in addition to its influence on self-esteem and self-efficacy. Although there is a vast literature on the impact of obesity on health, there is a relatively small group of studies on obesity's impact on general health status, specifically. Findings underscored the negative impact of current weight (Ferraro & Yu, 1995; Manderbacka et al., 1999) as well as the positive influence of weight loss (Fontaine, Barofsky, Bartlett, Franckowiak, & Andersen, 2004) on self-reported general health status. Cross-sectional studies have found increased levels of BMI significantly associated with self-reported poor health (Mansson & Merlo, 2001; Kobau, Safran, Zack, Moriarty, & Chapman, 2004), with obesity predicting poor self-rated health in nationally representative studies (Ferraro & Yu, 1995).

Moreover, findings from national studies indicate gradient effect in that severely obese are more likely than obese to report poor health (Hassan, Joshi, Madhavan, & Amonkar, 2003), with excess odds increasing from 12% to 323% as weight increases (Ford, Moriarty, Zack, Mokdad, & Chapman, 2001). Consistent with this conclusion, a non-U.S. study indicates the presence of a

gradient; compared to normal weight individuals, poor health was more likely to be reported by persons overweight (OR 1.46, 95% CI 1.24-1.71) or obese (OR 2.67, 95% CI 2.04-3.48) (Manderbacka et al., 1999)

Smoking

Of all three health behaviors, smoking has the least amount of literature directly examining its association with general health status. Only recently has a national investigation of smoking, other risk behaviors, and self-reported health been conducted, with findings supporting the significantly negative impact of current smoking (Strine et al., 2005). Previous international studies have found the direct influence of smoking, whereby those who smoke are greater than 60% (OR 1.63, 95% CI 1.23-2.16) more likely to report worse general health seven to ten years later (Froom et al., 2004). Additional indirect associations indicated that those who smoke are at significantly greater odds (OR 1.81, 95% CI 1.49-2.19) of reporting poor health (Manderbacka et al., 1999).

Health Behaviors and Mental Distress

There is a growing literature on the impact of health behavior on mental distress. Studies have examined both type and amount of risk behavior on prevalence and treatment of mental distress. Specific disorders as well as general sub-syndromal moodiness (Kobau, Safran, Zack, Moriarty, & Chapman,

2004), which may result in an increased burden of disease, has been studied. Although the majority of studies have examined the role of individual risk behaviors, there are investigations of the association between a cluster of risk factors and mental health/distress as well. To compare, the evidence supporting the influence of physical activity on mental health is far more rigorous, with the obesity literature less advanced. Although the relationship between smoking and mental distress has been established, the evidence, by and large, is mixed as related to direction of influence.

Physical Inactivity

There is a growing literature on the benefits of physical activity for both the prevention and treatment of an array of mental disorders. There is evidence from a variety of studies, including cross-sectional, longitudinal, quasi-experimental (Dunn, Trivedi, & O'Neal, 2001; Salmon, 2001), and randomized controlled investigations (Blumenthal et al., 1999; Atlantis et al., 2004). Although the relationship between exercise and mental health is a complex one, the protective influence of physical activity is not questioned. Findings support the benefit of acute exercise on present state of self-reported stress and long-term benefits in reduction of risk of psychopathology (Dunn et al., 2001).

Positive influence of physical activity has been found with regards to specific disorders such as depression (Atlantis, Chow, Kirby, & Singh, 2004; Dunn, Trivedi, Kampert, Clark, & Chambliss, 2005) and anxiety (Cromarty,

Robinson, Callcott, & Freeston, 2004) as well as general stress responsivity (Scully, Kremer, Meade, Graham, & Dudgeon, 1998; Atlantis et al., 2004), mood state, and self-esteem (Scully, Kremer, Meade, Graham, & Dudgeon, 1998). Investigations on the public burden of mental disorders have found that even after controlling for sociodemographic factors, individuals who engage in regular exercise have a significantly lower prevalence of many mental disorders, including depression (OR 0.75, 95% CI 0.6-0.94), panic attacks (OR 0.73, 95% CI 0.56-0.96), social phobia (OR 0.65, 95% CI 0.53-0.8), specific phobia (OR 0.78, 95% CI 0.63-0.97), and agoraphobia (OR 0.64, 95% CI 0.43-0.94) (Goodwin, 2003).

There are multiple plausible mechanisms through which physical activity influences mental health. Biological explanations suggest that exercise impacts neurotransmitter production and response (Scully et al., 1998). Physical activity may also act as a buffer to the body's natural response to stress and stress hormones, thereby improving individual resilience (Scully et al., 1998). Psychological explanations posit that regular exercise may lead to enhanced self-esteem and improved body image (Scully et al., 1998). Finally, some propose that the advantage stems from a dose-response relationship with respect to prevalence (Goodwin, 2003; Abu-Omar, Rutten, & Lehtinen, 2004) as well as treatment (Dunn et al., 2005). Overall, most dose-related studies have found that it is regular exercise, that is physical activity meeting the recommended level, which confers benefit (Brown et al., 2003; Brown et al., 2004).

Obesity

There is evidence of the association between obesity and mental distress, with findings from a variety of correlational and prospective studies. Early studies examining the prevalence of depression among the obese have found mixed results with some finding no association, reciprocal association, depression leading to obesity, or obesity implicated in depression or mental distress (Friedman & Brownell, 1995). Cross-sectional studies have found that those who are obese are 17% to 41% more likely to report mental distress more than half the month (Hassan et al., 2003). More recently, findings from prospective cohort studies have indicated that being obese confers excess risk of future depression and other adverse mental health outcomes (Roberts, Deleger, Strawbridge, & Kaplan, 2003; Hasler et al., 2004). For example, controlling for a variety of sociodemographic and psychosocial factors, there is a 70% to 200% excess risk of depression up to five years later for those who are obese (Roberts, Kaplan, Shema, & Strawbridge, 2000; Roberts, Strawbridge, Deleger, & Kaplan, 2002; Roberts et al., 2003).

There is an array of explanatory models and hypothesized mechanisms underlying this relationship. Some suggest that it is not being overweight that is distressing, but rather the relationship is better explained by the negative experience of dieting and related stress involved in attempting (and often failing)

to meet societal norms which dictate thinness as a requirement for attractiveness (Ross, 1994). Others have proposed that there are multiple biopsychosocial pathways accounting for the association, which include disparate consumption of carbohydrates and reduced neurotransmitter production resulting from inactivity (Palinkas, Wingard, & Barrett-Connor, 1996; Roberts, Deleger, Strawbridge, & Kaplan, 2003).

Although more rigorous studies have established the role of weight in the etiology of mental distress, the effect is not consistent across subgroups. Sociodemographic differences have been found based on age as well as gender. Increased body weight and depression has been found more often among women than men (Carpenter, Hasin, Allison, & Faith, 2000) with evidence of an inverse relationship in older men (Palinkas et al., 1996).

Smoking

The use of tobacco and nicotine is associated with mental distress, although evidence regarding the directionality of the relationship is mixed (Lasser et al., 2000). There is evidence to support both those suffering from mental disorders are more prone to smoke as well as increased prevalence of mental disorders among smokers (Williams & Ziedonis, 2004). Some hypothesize that this complex relationship is due to genetic diathesis whereby one form of

neurobiological or endocrine response may be involved in the etiology of both outcomes (Picciotto, Brunzell, & Caldarone, 2002; Williams & Ziedonis, 2004).

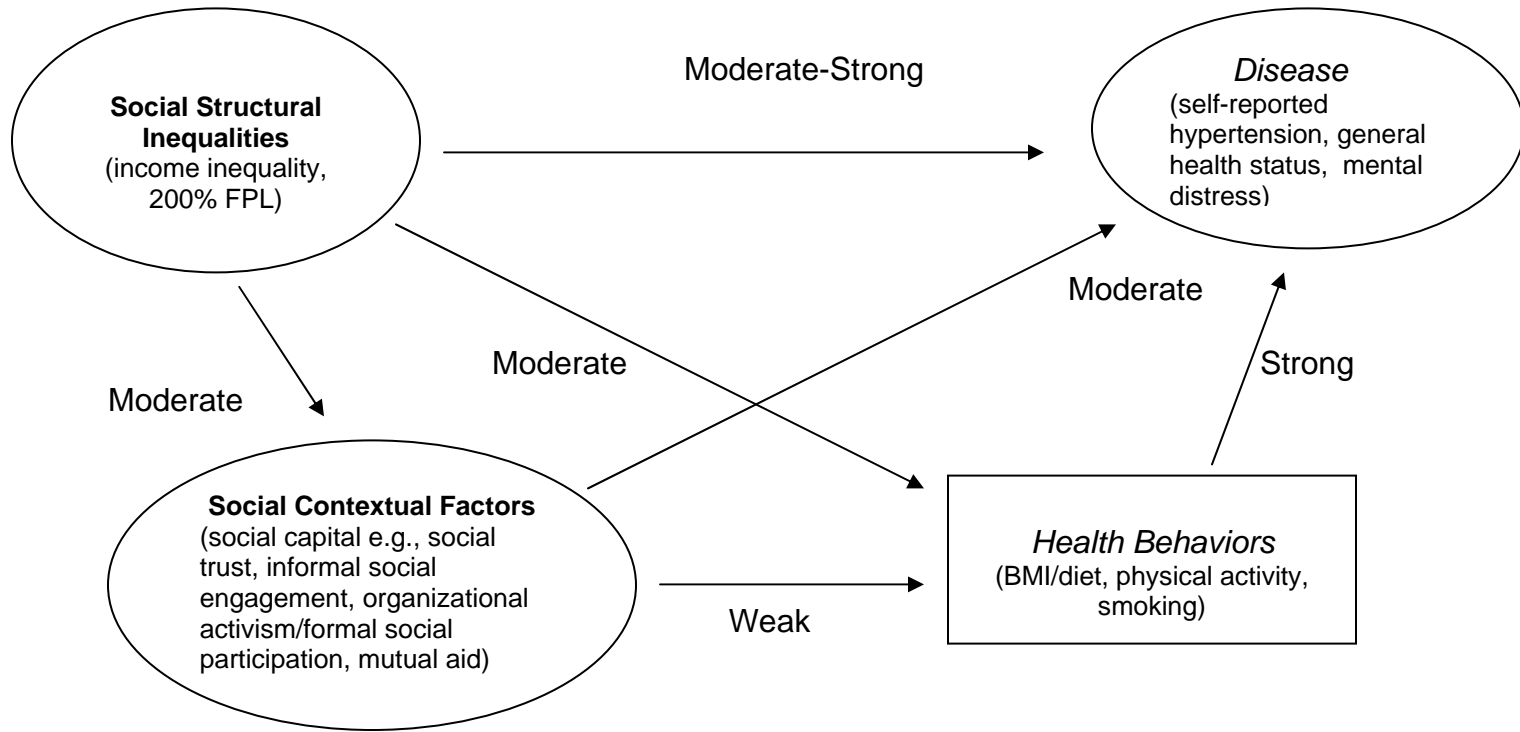
Additionally, findings demonstrate that these systems may also be implicated as mechanisms underlying the smoking - mental distress relationship. For example, smoking is posited to alter neurotransmitters associated with depression (Dierker, Avenevoli, Stolar, & Merikangas, 2002) as well as chronic use exacerbating more severe symptomatology associated with schizophrenia and Post-Traumatic Stress Disorder (Williams & Ziedonis, 2004).

There are a variety of mental disorders associated with previous smoking. In a study involving two national data sets, Breslau and Klein (1999) found that daily smoking conferred a significant increased risk of panic attacks for both men and women. There was no evidence to support the reverse – panic attacks were not associated with initiation of subsequent smoking behavior. There was additional evidence for the link from smoking to anxiety from a prospective longitudinal study. Controlling for a myriad of individual characteristics, smoking one pack or more per day in adolescence assigned excess risk in early adulthood of the following disorders: generalized anxiety disorder (OR 5.53, 95% CI 1.84-16.66), agoraphobia (OR 6.79, 95% CI 1.53-30.17), and panic disorder (OR 15.58 95% CI 2.31-105.14) (Johnson et al., 2000). There is also consistent evidence on the association between smoking and depressive symptomatology (Breslau, Kilbey, & Andreski, 1991; Dierker et al., 2002; Williams & Ziedonis, 2004), with findings indicating that chronic smoking results in almost four times

the risk of having major depression (OR 3.90, 95% CI 1.85-8.20) (Goodman & Capitman, 2000).

In sum, engaging in the risk behaviors examined in this study are significantly associated with frequent mental distress (Kobau et al., 2004; Strine et al., 2004). In addition, taken as a whole, there is sufficient empirical evidence to support associational relationships amongst social structure, social context, risk behaviors, and the three health outcomes under study, even after adjusting for various competing factors. The strength of causal evidence, however, is mixed (Figure 2.2). For example, there is moderate strength of evidence of a causal relationship between social structure and both social context and health behavior, with a moderate-strong level with CVD. In regards to social contextual factors, there is moderate strength of evidence with respect to CVD, however weak support of a causal link with health behaviors, which may be due to the dearth of studies in that area. By far, the strongest evidence base of a causal relationship is between health behaviors and CVD, with the majority of the literature demonstrating temporal ordering of effects. In essence, there are no studies that have attempted to examine these different levels of association and their possible causal effects on self-reported CVD. Therefore, based upon the above review of literature, this study has the potential to advance the knowledge base in this area and make a contribution to the field (Table 2.1).

Figure 2.2: Strength of Evidence



Weak = Correlational evidence only

Moderate = Some evidence ruling out of alternative explanations

Strong = Some causality established (evidence of temporal ordering)

Table 2.1: Hypotheses

Overall	Hypertension	General Health Status	Mental Distress
<i>Behavioral variables only partially mediate social structure and disease</i>	<i>Social structural inequality in the community in which one resides will positively influence self-reported hypertension.</i>	<i>Social structural inequality in the community in which one resides will negatively influence self-reported general health status.</i>	<i>Social structural inequality in the community in which one resides will negatively influence self-reported mental health.</i>
1a	1a	4a	7a
	Greater income inequality in the community in which one resides will positively influence self-reported hypertension.	Greater income inequality in the community in which one resides will negatively influence self-reported general health status.	Greater income inequality in the community in which one resides will negatively influence self-reported mental health.
	1a1	4a1	7a1
	Greater poverty in the community in which one resides will positively influence self-reported hypertension.	Greater poverty in the community in which one resides will negatively influence self-reported general health status.	Greater poverty in the community in which one resides will negatively influence self-reported mental health.
	1a2	4a2	7a2
	<i>The effect of social structure on self-reported hypertension is only partly mediated by known risk behaviors (BMI/diet, physical activity, smoking).</i>	<i>The effect of social structure on self-reported general health status is only partly mediated by known risk behaviors (BMI/diet, physical activity, smoking).</i>	<i>The effect of social structure on self-reported mental health is only partly mediated by known risk behaviors (BMI/diet, physical activity, smoking).</i>
	1b	4b	7b
	Greater income inequality in the community in which one resides will positively influence self-reported hypertension after controlling for individual risk behavior.	Greater income inequality in the community in which one resides will negatively influence self-reported general health status after controlling for individual risk behavior.	Greater income inequality in the community in which one resides will negatively influence self-reported mental health after controlling for individual risk behavior.
	1b1	4b1	7b1
	Greater poverty in the community in which one resides will positively influence self-reported hypertension after controlling for individual risk behavior.	Greater poverty in the community in which one resides will negatively influence self-reported general health status after controlling for individual risk behavior.	Greater poverty in the community in which one resides will negatively influence self-reported mental health after controlling for individual risk behavior.
	1b2	4b2	7b2
<i>Greater social structural inequalities in the community in which one resides positively influences engaging in high risk behavior.</i>			
1c			

Table 2.1: Hypotheses

Overall	Hypertension	General Health Status	Mental Distress
1c1	Greater income inequality in the community one resides in positively influences physical inactivity.		
1c2	Greater income inequality in the community one resides in positively influences overweight/obesity.		
1c3	Greater income inequality in the community one resides in positively influences smoking.		
1c4	Greater poverty in the community one resides positively influences physical inactivity.		
1c5	Greater poverty in the community one resides positively influences overweight/obesity.		
1c6	Greater poverty in the community one resides positively influences smoking.		
<i>Social context partially mediates social structure and disease.</i>	<i>The level of social capital in the community in which one resides influences self-reported hypertension.</i>	<i>The level of social capital in the community in which one resides influences self-reported general health status.</i>	<i>The level of social capital in the community in which one resides influences self-reported mental health.</i>
	2a	5a	8a
	Communities characterized by less social trust will positively influence self-reported hypertension.	Communities characterized by less social trust will negatively influence self-reported general health status.	Communities characterized by less social trust will negatively influence self-reported mental health.
	2a1	5a1	8a1

Table 2.1: Hypotheses

Overall	Hypertension	General Health Status	Mental Distress
2b	<i>Social structural inequalities will be negatively associated with a salubrious social context.</i>		
2b1	Income inequality will be negatively associated with social trust.		
2b2	Income inequality will be negatively associated with informal social engagement.		
2b3	Income inequality will be negatively associated with formal group involvements, or organizational activism.		
2b4	Income inequality will be negatively associated with mutual aid.		
2b5	Poverty will be negatively associated with social trust.		
2b6	Poverty will be negatively associated with informal social engagement.		
2b7	Poverty will be negatively associated with formal group involvements, or organizational activism.		
2b8	Poverty will be negatively associated with mutual aid.		

Table 2.1: Hypotheses

Overall	Hypertension	General Health Status	Mental Distress
	2c <i>Social structural inequality in the community in which one resides will positively influence self-reported hypertension after controlling for community social context.</i>	5b <i>Social structural inequality in the community in which one resides will negatively influence self-reported general health status after controlling for community social context.</i>	8b <i>Social structural inequality in the community in which one resides will negatively influence self-reported mental health after controlling for community social context.</i>
	2c1 Income inequality in the community in which one resides will positively influence self-reported hypertension after controlling for levels of social trust.	5b1 Income inequality in the community in which one resides will negatively influence self-reported general health status after controlling for levels of social trust.	8b1 Income inequality in the community in which one resides will negatively influence self-reported mental health after controlling for levels of social trust.
	2c2 Income inequality in the community in which one resides will positively influence self-reported hypertension after controlling for informal social engagement.	5b2 Income inequality in the community in which one resides will negatively influence self-reported general health status after controlling for informal social engagement.	8b2 Income inequality in the community in which one resides will negatively influence self-reported mental health after controlling for informal social engagement.
	2c3 Income inequality in the community in which one resides will positively influence self-reported hypertension after controlling for formal group involvements, or organizational activism.	5b3 Income inequality in the community in which one resides will negatively influence self-reported general health status after controlling for formal group involvements, or organizational activism.	8b3 Income inequality in the community in which one resides will negatively influence self-reported mental health after controlling for formal group involvements, or organizational activism.
	2c4 Income inequality in the community in which one resides will positively influence self-reported hypertension after controlling for levels of mutual aid.	5b4 Income inequality in the community in which one resides will negatively influence self-reported general health status after controlling for levels of mutual aid.	8b4 Income inequality in the community in which one resides will negatively influence self-reported mental health after controlling for levels of mutual aid.

Table 2.1: Hypotheses

Overall	Hypertension	General Health Status	Mental Distress
	Poverty inequality in the community in which one resides will positively influence self-reported hypertension after controlling for levels of social trust. 2c5	Poverty inequality in the community in which one resides will negatively influence self-reported general health status after controlling for levels of social trust. 5b5	Poverty inequality in the community in which one resides will negatively influence self-reported mental health after controlling for levels of social trust. 8b5
	Poverty in the community in which one resides will positively influence self-reported hypertension after controlling for informal social engagement. 2c6	Poverty in the community in which one resides will negatively influence self-reported general health status after controlling for informal social engagement. 5b6	Poverty in the community in which one resides will negatively influence self-reported mental health after controlling for informal social engagement. 8b6
	Poverty in the community in which one resides will positively influence self-reported hypertension after controlling for formal group involvements, or organizational activism. 2c7	Poverty in the community in which one resides will negatively influence self-reported general health status after controlling for formal group involvements, or organizational activism. 5b7	Poverty in the community in which one resides will negatively influence self-reported mental health after controlling for formal group involvements, or organizational activism. 8b7
	Poverty in the community in which one resides will positively influence self-reported hypertension after controlling for levels of mutual aid. 2c8	Poverty in the community in which one resides will negatively influence self-reported general health status after controlling for levels of mutual aid. 5b8	Poverty in the community in which one resides will negatively influence self-reported mental health after controlling for levels of mutual aid. 8b8
<i>Behavior only partially mediates social context and disease.</i> 3	<i>Engaging in risk behavior (BMI/diet, physical inactivity, smoking) is positively associated with self-reported hypertension.</i> 3a	<i>Engaging in risk behavior (BMI/diet, physical inactivity, smoking) is negatively associated with self-reported general health status.</i> 6a	<i>Engaging in risk behavior (BMI/diet, physical inactivity, smoking) is negatively associated with self-reported mental health.</i> 9a
<i>Weaker social context in the community in which one resides positively influences engaging in high-risk behavior.</i> 3b			

Table 2.1: Hypotheses

Overall	Hypertension	General Health Status	Mental Distress
3b1	Weaker social trust in the community in which one resides positively influences physical inactivity.		
3b2	Less informal social engagement in the community in which one resides positively influences physical inactivity.		
3b3	Less organizational activism in the community in which one resides positively influences physical inactivity.		
3b4	Less mutual aid in the community in which one resides positively influences physical inactivity.		
3b5	Weaker social trust in the community in which one resides positively influences overweight/obesity.		
3b6	Less informal social engagement in the community in which one resides positively influences overweight/obesity.		
3b7	Less organizational activism in the community in which one resides positively influences overweight/obesity.		

Table 2.1: Hypotheses

Overall	Hypertension	General Health Status	Mental Distress
3b9	Weaker social trust in the community in which one resides positively influences smoking.		
3b10	Less informal social engagement in the community in which one resides positively influences smoking.		
3b11	Less organizational activism in the community in which one resides positively influences smoking.		
3b12	Less mutual aid in the community in which one resides positively influences smoking.		
	<i>3c Weaker social context in the community in which one resides positively influences self-reported hypertension after controlling for individual risk behavior.</i>	<i>6b Weaker social context in the community in which one resides negatively influences self-reported general health status after controlling for individual risk behavior.</i>	<i>9b Weaker social context in the community in which one resides negatively influences self-reported mental health after controlling for individual risk behavior.</i>
	3c1 Weaker social trust in the community in which one resides positively influences hypertension after controlling for individual risk behavior.	6b1 Weaker social trust in the community in which one resides negatively influences general health status after controlling for individual risk behavior.	9b1 Weaker social trust in the community in which one resides positively influences mental distress after controlling for individual risk behavior.
	3c2 Less informal social engagement in the community in which one resides positively influences hypertension after controlling for individual risk behavior.	6b2 Less informal social engagement in the community in which one resides negatively influences general health status after controlling for individual risk behavior.	9b2 Less informal social engagement in the community in which one resides positively influences mental distress after controlling for individual risk behavior.

Table 2.1: Hypotheses

Overall	Hypertension	General Health Status	Mental Distress
	Less organizational activism in the community in which one resides positively influences hypertension after controlling for individual risk behavior. 3c3	Less organizational activism in the community in which one resides negatively influences general health status after controlling for individual risk behavior. 6b3	Less organizational activism in the community in which one resides positively influences mental distress after controlling for individual risk behavior. 9b3
	Less mutual aid in the community in which one resides positively influences hypertension after controlling for individual risk behavior. 3c4	Less mutual aid in the community in which one resides negatively influences general health status after controlling for individual risk behavior. 6b4	Less mutual aid in the community in which one resides positively influences mental distress after controlling for individual risk behavior. 9b4

CHAPTER 3: METHODOLOGY

Methods

Study Design

This study employed a multilevel, retrospective, nonexperimental design with longitudinal elements utilizing secondary data. Multilevel designs were utilized when data have been clustered or nested within different levels of analysis. The design was nonexperimental in that it employs data from a naturally occurring study population without randomization and although did not clearly include the temporal ordering necessary to generate evidence concerning causal relationships, it did allow for temporal precedence to a limited extent. This work was retrospective, in that it defined the study population in terms of 2001 data and then linked those data to exposures in 2000 data sources. The data utilized in this study were originally collected for different studies and purposes, but they have been judged to be suitable for addressing this study's objectives based upon criteria outlined by McCall and Applebaum (McCall & Applebaum, 1991) and Stewart and Kamins (Stewart & Kamins, 1993).

Until recently, the study of macro-level social determinants of health has relied on ecological studies, which have employed cross-sectional designs. The proposed study employed a multilevel design for two reasons. First, a multilevel design was selected because of the nature questions that were investigated and

the data that was utilized. Different data sources representing differing levels of analysis were critical to this study as no one data set had specific multilevel data related to CVD, general health status, or mental distress. This design was chosen also because it had multiple benefits over ecological approaches, including limiting problems related to fallacies (ecologic, atomistic) and allowing for the unique variance of contextual and compositional levels (e.g., to test whether income inequality and social capital effects on CVD were significant while adjusting for individual-level factors, such as SES and individual health behaviors) (Subramanian et al., 2003).

This retrospective study defined the sample in terms of availability of outcome data (CVD, general health status, mental distress) in 2001 and then examined associations with exposure (social structural and social contextual inequalities) from other sources of aggregation (from the 2000 Census for social structural variables and from the 2000 SCCBS for social contextual variables). Normally in this type of study, the concern making valid inferences included recall bias, however as this study was not relying on one group at one level of analysis, this threat was not applicable.

This study was a nonexperimental design with longitudinal elements in that the data was observational in nature and deliberately selected to address the temporal ordering of possible effects through observations gleaned from two different time points. Typically in a nonexperimental design, directionality cannot be established; however, because this design had a temporal component, exposure was speculated to precede disease (and not visa versa) to some

extent. As with most nonexperimentation, this study presented a more rigorous design than cross-sectional, however one still cannot infer causality due to nonrandomness of assignment to groups.

Secondary data sources were used in this study for a few reasons. First of all, in order to answer the research questions proposed, multiple sources of data were needed at various levels of aggregation (i.e., individuals in communities in counties). In addition, there was no single publicly available data set in the United States that included all the variables of interest. These three data sources were selected because they included some portion of the variables of interest in each and, as importantly, they all were linkable based on federal codes included in each. Furthermore, few studies existed that explicitly addressed the effects of social capital and related variables on specific health outcomes. At present, there have been no studies that link the BRFSS health-related data to a rich source of contextual data. The study linked the BRFSS with the Social Capital Benchmark Study by FIPS codes that were present in both – this way individuals were placed in their respective community or state contexts.

Sampling

The sample included in each dataset (Census, SCCBS, and BRFSS) was intended to represent the same population, but the data sources reflected different levels of aggregation and different timepoints. The sample for this study

was limited to individuals from the 2001 BRFSS sample who resided in communities represented in the SCCBS.

BRFSS

The BRFSS was an ongoing public health surveillance system of health behaviors of adults in the United States. Its purpose was to gather information on health practices, knowledge, and risk associated with major burdens of disease and disability. Its intent was to collect prevalence estimates on the lifestyle and health behaviors of adults in the U.S, which have been used to inform prevention policy and public health practice. The data reflected both national and state-specific trends on a variety of public health-related factors. It was an annual telephone survey administered by the CDC to a random sample of adults.

Implementation of the survey was conducted by state and local health departments. The reliability of the BRFSS has been evaluated through test-retest studies. Overall, the survey exhibited good reliability, with Kappa ranging from 0.60 (for minority participants) to 0.80 (for White respondents) in regards to sociodemographic items and ranging from 0.70 to 0.80 for behavioral items (Stein, Lederman, & Shea, 1993). Other examinations of the BRFSS have found it to have both moderate (physical activity) and high reliability and validity in most of its items (smoking, blood pressure, height, weight, and demographics), which come from the core instrument (Nelson, Holtzman, Bolen, Stanwyck, & Mack,

2001) (Table 3.1). Specifically in regards to CVD, the BRFSS has been used in many studies to assess trends in factors such as hypertension (Ayala, Greenlund, & Croft, 2002) as well as multiple risk (Jackson, Jatulis, & Fortmann, 1992; Greenlund et al., 2004).

There were three sections to the survey: core questions (all participate), optional modules (participation is decided upon by state), and state-added questions. The number of questions in any given survey ranged from 90 – 150 items. Probability sampling was used for all households with telephones in each state. The majority of participating states utilized a disproportionate stratified sample (DSS) design. Interviews were carried out using computer assisted telephone interviewing (CATI), with interviews lasting an average of 15-20 minutes.

Table 3.1: Behavioral Risk Factor Surveillance System Reliability and Validity¹

Variable	Topic	Reliability ²	Validity ³	Consistent with Other Surveys	
Smoking	Current Smoking Status	High	High	Yes	
Obesity	Height	High	High	Yes	
	Weight	High	High	Yes; slightly underreported	
Physical Activity	Level	Moderate	Moderate	Mixed	
CVD	Hypertension	High	Moderate	Yes; slightly underreported	
General Health Status	Health-Related Quality of Life	Moderate	High	Measures only from other surveys	
Mental Health/Distress	Health-Related Quality of Life	Difficult to determine	Moderate	Mixed	
² where reliability	High:	$\kappa > 0.60$	³ where validity	High:	Sensitivity & Specificity > 80% or correlation coefficients > 0.60
	Moderate:	$0.60 < \kappa > 0.40$		Moderate:	60% < Sensitivity & Specificity > 79% or 0.40 < correlation coefficients > 0.59
	Low:	$\kappa > 0.40$		Low	Sensitivity & Specificity < 60% or correlation coefficients < 0.40

¹Nelson, Holtzman, Bolen, Stanwyck, & Mack, 2001

SCCBS

The Social Capital Community Benchmark Survey (SCCBS) was to date the largest survey of civic engagement in the United States. The study was designed by scholars in social capital and measurement who attended the Saguaro Seminar at Harvard University in 1999 and was obtained for the purposes of this study from the Roper Center for Public Opinion Research. The SCCBS was the first of its kind to measure aspects and correlates of social capital across the United States conducted on individuals and then aggregated to community-level. Another intended use of the data was to provide the communities who participated with information to support efforts targeting improvement in community connectedness. Data was aggregated from individual responses to contextual level constructs, as communities, not individuals themselves, made up the sample. Communities were invited to participate during an annual meeting of Foundations in 1999. Thirty-four Foundations were selected for the range of communities they represented across the U.S. The communities consisted of counties, cities, and lightly populated states. Each Foundation selected the areas within their communities to be surveyed, with the majority using proportionate sampling. The purpose of this work was to provide researchers and practitioners a comprehensive benchmark database to enhance current knowledge and future initiatives. The survey interviews were approximately 26 minutes and were completed between July and November 2000 and was carried out using random-digit dialing. As certain

Foundations represented more than one community, there were 40 communities in the final sample, each represented by approximately 500-1500 residents (Table 3.2).

The contextual-level data from the SCCBS were linked to the individual-level data from the BRFSS through Federal Informational Processing Standards (FIPS) codes. FIPS codes were federally designated unique numbers that were assigned to every county in each state. Utilizing FIPS codes allowed each person in the BRFSS sample to be located within his/her community represented in the SCCBS sample. However, only 27 out of the 40 communities in the original study were used. These thirteen were omitted because the geography of the community could not be matched with Census data, did not have FIPS codes assigned, and therefore could not be linked to the BRFSS data (12) or did not have data collected for the 2001 BRFSS (1).

Census

Data was obtained from the 2000 Census. The Census has been conducted every ten years and was a survey of individuals and households in the United States. The Census provided statistical information regarding the population. Results have informed national and local public planning and program funding as well as research. Specifically, measures of social structural inequalities (i.e., absolute and relative deprivation) were employed. Relative deprivation was assessed by income inequality. Absolute or area measures of

poverty included the percent of families living at or below 200% of the Federal Poverty Level (FPL).

Community-level data from the SCCBS was linked to county-level data from the Census through the use of FIPS codes as well. The 27 communities from the SCCBS that make up this study's sample varied in the number of counties they represent. Therefore, some communities included only one county, some included several, and a few included lightly populated states.

Variable Measures

Variables selected were grouped into four categories: social structural, social contextual, health behavior, and outcome (Table 3.3). The social structural inequalities were represented by relative and absolute deprivation and were measured by the 2000 Census. The social contextual factors included social capital and its correlates and were measured by the SCCBS (2000). The third and fourth groups of variables, health behavior and outcomes, were both measured by the 2001 BRFSS.

Table 3.2: Social Capital Community Benchmark Survey Sample

Actual Sample Size	Location (State/County)	Response Rate (%)
500	Alabama/Jefferson, Shelby	31.6
501	Arizona/Maricopa	31.7
515	California/Los Angeles	24.1
504	California/San Diego	30.9
500	California/San Francisco	27.1
500	Colorado/Boulder	22.4
501	Colorado/Denver	14.9
1379	Delaware/state of	27.3
510	Georgia/DeKalb, Fulton, Cobb, Rockdale, Henry	29.8
1001	Indiana/state of	26.7
500	Louisiana/Baton Rouge	25.0
500	Michigan/Kalamazoo	27.1
501	Michigan/Wayne, Oakland, Macomb, St. Clair, Washtenaw, Monroe, Livingston	30.1
503	Minnesota/Dakota, Ramsey, Washington	39.2
502	Montana/state of	44.1
711	New Hampshire/state of	32.2
541	New York/Onondaga	24.8
988	New York/Monroe, Wayne, Ontario, Livingston, Genesee, Orleans	27.1
750	North Carolina/Forsyth	34.8
750	North Carolina/Guilford	32.7
1100	Ohio/Cuyahoga	20.0
1001	Ohio/Butler, Clermont, Hamilton, Warren Kentucky/Boone, Campbell, Kenton Indiana/Dearborn	38.7
500	Oregon/Crook, Deschutes, Jefferson	34.1
500	Pennsylvania/York	28.2
500	Texas/Harris	28.7
500	Washington/Yakima	34.6
500	West Virginia/Kanawha, Putnam, Boone	27.4

Table 3.3: Variable Sources & Definitions

Source	Variable	Defined as
Census	Relative Deprivation: Income Inequality	Qualitative and quantitative description of the dispersion or distribution or range of income in a population
Census	Absolute Deprivation: Poverty	Area-level socioeconomic status
SCCBS	Social Capital & Correlates	Features of social organization, such as participation in associations and civic engagement, interpersonal trust, and norms of reciprocity, which act as resources and facilitate collective action
BRFSS	Physical Activity	Any activity that can be considered exercise (e.g., not related to work)
BRFSS	Obesity/Overweight	Body Mass Index meeting USDHHS cutoffs
BRFSS	Smoking	History of or active tobacco smoking
BRFSS	CVD	Hypertension
BRFSS	General Health Status	Global health assessment
BRFSS	Mental Distress	Number of days in past 30 mental health not good

Income inequality was a form of relative deprivation. It was measured by the commonly used Gini coefficient. The Gini has been derived from the Lorenz curve, which was a diagram of the cumulative proportion of income plotted against the cumulative percentage of the population (Kawachi & Kennedy, 1997b; Soobader & LeClere, 1999). The proportion approaching 0 indicated perfect equality whereas 1 demonstrated perfect inequality. Area-level poverty or absolute deprivation was measured by the percent of the population in a specified area who lived at or below 200% of the Federal Poverty Line. For 2000, the FPL ranged from \$8,350 (family of 1) and \$11,250 (family of 2) to \$17,050 (family of 4). Specific data related to income, number of households, and population economic indicators were obtained from the 2000 Census in order to calculate both measures of social structural inequalities.

Social contextual variables consisted of social capital and its correlates including measures of social trust, participation in formal and informal organizations, and mutual aid. All items measuring these constructs came from the 2000 SCCBS. Social trust described a characteristic of the collective (e.g., general interpersonal trust, level of trust amongst neighbors, coworkers, etc.). There were five questions in this index (Table 3.4). Items related to this construct included whether most people can be trusted, to what extent one can trust the police in one's community, or to what extent one trusts people who work in the stores in which one shops. Response options for each item ranged from a five to seven point Likert scale.

Participation in formal organizations, or organizational activism (Table 3.5), included a count of eighteen various activities such as involvement in professional, neighborhood, service, charity groups whereas informal social activity or engagement (Table 3.6) included five items related to other relationship-based actions such as engaging with relatives, friends, or having people to your home.

Mutual aid was measured by several items (Table 3.7). Questions were related to volunteering (e.g., for a neighborhood or civic group, for a school or youth program, for a place of worship) and donating. Response options were either yes/no or a five to seven point Likert scale, depending upon the specific item.

For the purposes of this study, health risk was restricted to poor physical activity, being overweight or obese, and engaging in smoking behavior (Table 3.8). BRFSS items related to physical activity pertained specifically to non-work related activity. Physical activity was measured as meeting current recommended levels in type (light vs. moderate vs. vigorous), duration (minutes), and frequency (days/week or month), engaging in some activity, or not active. Activities were defined as those which caused small changes in respiration and heart rate, such as brisk walking, bicycling, vacuuming, and gardening. In addition, activity included those that cause significant increase in respiration and heart rate, such as running, aerobics, and heavy yard work. Reliability and validity estimates for BRFSS items related to physical activity were moderate (Table 3.1).

Table 3.4: Social Capital Community Benchmark Survey Social Trust Index

Variable	Item	Response		
TRUST	“Whether most people can be trusted ”	1. People can be trusted 2.You can't be too careful	3.Depends	8.Don't Know 9.Refused
TRNEI	"How much you can trust people in your neighborhood"	1.Trust them a lot 2.Trust them some	3.Trust them a little 4.Trust them not at all	5.Does not apply 8.Don't Know 9.Refused
TRWRK	“How much you can trust people you work with”	1.Trust them a lot 2.Trust them some	3.Trust them only a little 4.Trust them not at all	5.Does not apply 8.Don't Know 9.Refused
TRREL	“How much you can trust people at your church or place of ”	1.Trust them a lot 2.Trust them some	3.Trust them only a little 4.Trust them not at all	5.Does not apply 8.Don't Know 9.Refused
TRSHOP	“How much you can trust people who work in the stores where you shop”	1.Trust them a lot 2.Trust them some	3.Trust them only a little 4.Trust them not at all	5.Does not apply 8.Don't Know 9.Refused
TRCOP	“How much you can trust the police in your local community”	1.Trust them a lot 2.Trust them some	3.Trust them only a little 4.Trust them not at all	5.Does not apply 8.Don't Know 9.Refused

Table 3.5 : Social Capital Community Benchmark Survey Informal Social Engagement Index

Variable	Item	Response
CFRDVIST	“In the past twelve months, how often had friends over to your home”	Continuous
CFAMVISI	“In the past twelve months, how often visited with relatives”	Continuous
CJOBSOC	“In the past twelve months, how often socialized with co-workers outside of work”	Continuous
CFRDHANG	“In the past twelve months, how often hung out with friends in a public place”	Continuous
CCARDS	“In the past twelve months, how often played cards or board games with others”	Continuous

Table 3.6: Social Capital Community Benchmark Survey Formal Social Participation/Organizational Activism Index

Variable	Item	Response			
GRPREL	Participate in organization affiliated with religion	1. Yes	2. No	3. Don't Know	4. Refused
GRPSPORT	Participate in sports club, league, or outdoor activity	1. Yes	2. No	3. Don't Know	4. Refused
GRPYOUTH	Participate in youth organization	1. Yes	2. No	3. Don't Know	4. Refused
GRPPTA	Participate in parent association or other school support group	1. Yes	2. No	3. Don't Know	4. Refused
GRPVET	Participate in veterans group	1. Yes	2. No	3. Don't Know	4. Refused
GRPNEI	Participate in neighborhood association	1. Yes	2. No	3. Don't Know	4. Refused
GRPELD	Participate in seniors group	1. Yes	2. No	3. Don't Know	4. Refused
GRPSOC	Participate in charity or social welfare organization	1. Yes	2. No	3. Don't Know	4. Refused
GRPLAB	Participate in labor union	1. Yes	2. No	3. Don't Know	4. Refused
GRPPROF	Participate in professional, trade, farm, or business as	1. Yes	2. No	3. Don't Know	4. Refused
GRPFRAT	Participate in service or fraternal organization	1. Yes	2. No	3. Don't Know	4. Refused
GRPETH	Participate in ethnic, nationality, or civil rights org	1. Yes	2. No	3. Don't Know	4. Refused

Table 3.6: Social Capital Community Benchmark Survey Formal Social Participation/Organizational Activism Index

Variable	Item	Response			
GRPPOL	Participate in political group	1. Yes	2. No	3. Don't Know	4. Refused
GRPART	Participate in literary, art, or musical group	1. Yes	2. No	3. Don't Know	4. Refused
GRPHOB	Participate in hobby, investment, or garden club	1. Yes	2. No	3. Don't Know	4. Refused
GRPSELF	Participate in self-help program	1. Yes	2. No	3. Don't Know	4. Refused
GRPWWW	Involved in group that meets over the Internet	1. Yes	2. No	3. Don't Know	4. Refused
GRPOTHR	Belong to other kinds of clubs or organizations	1. Yes	2. No	3. Don't Know	4. Refused
OFFICER	"Served as an officer or on a committee"	1. Yes	2. No	3. Don't Know	4. Refused
CCLUBMET	"In the past twelve months – How often attended a club meeting"	4 Point Likert Scale			
CPUBMEET	"In the past twelve months - How often attended public meeting discussing school or"	4 Point Likert Scale			

Table 3.7: Social Capital Community Benchmark Survey Mutual Aid Index

Variable	Item	Response		
VOLARTS	Volunteered for cultural or arts organizations	1. Yes 2. No	3. Don't Know 4. Refused	
VOLHEA	Volunteered for health care or fight disease	1. Yes 2. No	3. Don't Know 4. Refused	
VOLHUM	Volunteered to help poor or elderly	1. Yes 2. No	3. Don't Know 4. Refused	
VOLNEI	Volunteered for neighborhood or civic group	1. Yes 2. No	3. Don't Know 4. Refused	
VOLREL	Volunteered for a place of worship	1. Yes 2. No	3. Don't Know 4. Refused	
VOLYOU	Volunteered for school or youth programs	1. Yes 2. No	3. Don't Know 4. Refused	
CVOLTIME	"In the past twelve months, number of times volunteered"	Continuous		
GIVEOTHR	Dollars contributed to non-religious charities	0. None 1. < \$100 2. \$100 < \$500	3. \$500 < \$1000 4. \$1000 < \$5000 5. > \$5000	8. Don't know 9. Refused
GIVEREL	Dollars contributed to church or religious causes	0. None 1. < \$100 2. \$100 < \$500	3. \$500 < \$1000 4. \$1000 < \$5000 5. > \$5000	8. Don't know 9. Refused

Table 3.8: Behavioral Risk Factor Surveillance System Health Behavior Items

Health Behavior	Item	Response
Physical Activity	"During the past 30 days, other than your regular job, did you participate in any physical activities or exercise such as running, calisthenics, golf, gardening, or walking for exercise?"	Yes or No
	"Now thinking about the moderate physical activities you do in a usual week, do you do moderate activities for at least 10 minutes at a time, such as brisk walking, bicycling, vacuuming, gardening or anything else that causes small increases in breathing or heart rate?"	Yes or No
	"How many days per week do you do these moderate activities for at least 10 minutes at a time?"	Days per week
	"On days when you do moderate activities for at least 10 minutes at a time, how much total time per day do you spend doing these activities?"	Hours and minutes per day
	"Now thinking about the vigorous physical activities you do in a usual week, do you do vigorous activities for at least 10 minutes at a time, such as running, aerobics, heavy yard work, or anything else that causes large increases in breathing or heart rate?"	Yes or No
	"How many days per week do you do these vigorous activities for at least 10 minutes at a time?"	Days per week

Table 3.8: Behavioral Risk Factor Surveillance System Health Behavior Items

Health Behavior	Item	Response
	"On days when you do vigorous activities for at least 10 minutes at a time, how much total time per day do you spend doing these activities?"	Hours and minutes per day
	Calculated physical activity level categorized	Meets recommendation/some activity/physically inactive
Tobacco Use	"Do you now smoke cigarettes every day, some days, or not at all?"	Current smoker(every day-some days)/former smoker/never smoked
	Smoking status (Derived)	Current &/or history of smoking/never smoked
Overweight/ Obesity	"About how much do you weigh without shoes?"	in pounds
	"About how tall are you without shoes?"	in feet/inches
	Calculated Body Mass Index categorized	Not overweight or obese (BMI<25)/ Overweight (25 ≥ BMI < 30)/ Obese (BMI ≥ 30)

Overweight or obesity was measured using the standard BMI cutoff points. The Body Mass Index has been calculated using height and weight, both of which were available from the BRFSS. Reliability and validity of self-report of height and weight in the BRFSS was high, although there have been slight underestimation of weight across populations (Nelson et al., 2001) (Table 3.1).

Smoking status was measured by a single composite variable. The specific questions reflected both history of smoking and whether the individual currently smoked every day, some days, or not at all. Reliability and validity of these items were high and were consistent with other surveys of smoking behavior (Nelson et al., 2001).

The outcome of interest was a restricted range of CVD, which has comprised a cluster of diseases, but for the purposes of this research study CVD indicator was limited to self-reported hypertension. The validity of utilizing self-report in assessments of CVD has been established for all race-sex groups (Giles, Croft, Keenan, Lane, & Wheeler, 1995). Reliability and validity were high to moderate, respectively, specifically for the BRFSS hypertension item (Nelson et al., 2001).

Hypertension was treated as a binary variable (yes/no), with an affirmative response on the one item indicating hypertension. Other forms of CVD were not being studied as there were either insufficient data in the 2001 BRFSS (Coronary Heart Disease and Myocardial Infarction) or they were either rare in occurrence (e.g., peripheral arterial disease, aortic aneurysm, deep vein thrombosis) or may have had a different physiological pathogenic process (e.g., stroke).

The original form of general health status was a Likert scale consisting of five response options: excellent, very good, good, fair, poor. Often due to the subjective nature of this variable, evaluation of reliability have been challenging. Although there are no reliability estimates for the BRFSS item per se, international studies on self-reported health were moderately reliable (Nelson et al., 2001). Validity issues were similar, in that although there have been no validity issues on the BRFSS item, other national surveillance surveys have demonstrated that self-reported health is highly valid and a strong predictor of future morbidity/mortality (Nelson et al., 2001). Consistent with previous studies of general health status and for ease of interpretation, the item was dichotomized in this study (0 = excellent/very good/good and 1 = fair/poor).

Mental distress was a self-report measure assessing the number of days out of the past 30 when one's mental health was not good (including feelings of sadness, anxiety, stress). There have been no studies of the reliability or validity of this BRFSS item. However, other scales with similar questions have found strong reliability and moderate validity (Nelson et al., 2001).

Analysis Procedures

To begin, preparation of the data for analysis included cleaning data and decisions regarding the treatment of missing data. The process of cleaning the data consisted of identifying and correcting errors in the data sets. The possible multiple sources of error that needed to be investigated included respondents or

coders mismarking responses, data entry errors, and “not applicable” or missing coded as 0. Detecting suspicious, erroneous, or illogical values involved examining the data through descriptive statistics, such as the range of possible values, outliers, frequencies, means, and standard deviations. Inconsistencies between related variables were explored as well. Errors that were found resulted in the variable in question being recoded, without jeopardizing the integrity of the item. Once the data were cleaned, arrangements were made for missing data.

Decisions regarding managing missing data included investigating the type and pattern of the missing information. First and foremost, it was imperative to assess whether the data was missing at random or was systematic. Every effort was made to retain or approximate the original distribution of responses in order to maintain the integrity of the data.

Following these steps in data preparation, the three data sources were linked and measures were taken to arrange the data for analysis. Data was weighted based upon the respective weighting schemes. The weight variable for the SCCBS was derived in a three step process whereby the initial weight (number of household adults/number of phone lines) was multiplied by the balancing weight derived from population distributions of variables such as gender, age, education, and race/ethnicity. The data weighting variable for the BRFSS was the product of several features of the sample and population. These factors included the probability of selection among strata of phone numbers, the number of phone lines in a respondents household, the number of adults in the respondents household, and an adjustment for non-coverage and non-response.

At this time, some variables were recoded and new variables were created (i.e., reporting health as fair/poor or not). Initially, univariate and bivariate analyses were conducted in order to see the ways in which the variables were distributed within the sample and their basic associations. Univariate statistics included mean, standard deviation, range, and distribution of responses on all items and composites, whether they represent Level-1 or Level-2 variables. Examination of the shape of responses was critical, as much of the analysis is based upon the assumption of normality. The only variables that were not anticipated to meet this criterion were the outcome variables (hypertension, general health status, and mental distress), which were assumed to be positively skewed in the population. The information permitted me to better understand the nature of the data employed and enhanced the process of analysis and interpretation.

The next step in examining the data was through bivariate analysis. Just as with univariate analysis, separate analyses were conducted for Level-1 and Level-2 data. As the range of variables in this study included nominal, ordinal, interval, and ratio levels of measurement, a variety of statistics were used, including Chi-Square, ANOVA, and Spearman and Pearson Correlations. Measures of association examined the relationships between, for example, each sociodemographic variable (e.g., SES, race/ethnicity, gender) and individual health behaviors and self-reported hypertension as well as covariation between social capital indicators and social structural variables (i.e., income inequality and

poverty). Bivariate analyses permitted the basic relationships between the variables to be elucidated and therefore informed the next step in the analysis.

Hypothesis testing included identification of first-level, second-level, and cross-level relationships utilizing Hierarchical Linear Modeling. Two outcomes were treated as binary (hypertension and general health status) and one comprised a count (mental distress). For the two outcomes treated as dichotomous, ($Y_{ij} \in \{0,1\}$), the probability distribution of the sampling model was Bernoulli and the logit link function was used for transformation. As mental distress was treated as a count of days out of last 30, ($Y_{ij} \in \{0,1,\dots, 30\}$), the probability distribution of the sampling model was Poisson and the log link function was used for transformation. Basic assumptions of a two level hierarchical generalized model were maintained. These assumptions included (Raudenbush & Bryk, 2002):

1. Each Level-1 random effect cannot be distributed normally, as it has either two discrete values or a count from 0-30, and variance σ^2 for each Level-1 unit within every Level-2 unit is heterogeneous.
2. There are restrictions on predicted values.
3. Level-1 predictors are independent of Level-1 random effects.
4. Level-2 random effects are multivariate normal, with a mean of 0 and variance of τ_{qq}
5. Level-2 predictors are independent of Level-2 random effects.
6. Level-1 and Level-2 errors are independent.

7. Predictors at each level are not correlated with random effects at the other level.

The first level variables included sociodemographic characteristics (i.e., gender, SES, race/ethnicity) and behavioral risk factors (i.e., physical inactivity, obesity, smoking). Control sociodemographic factors were modeled in a cluster with each behavior then added individually to the model. As behavioral risk factors tend to cluster in the population, different combinations of risk were examined.

The second level variables that were introduced in the model consisted of social contextual indicators (i.e., social capital and correlates) and social structural variables (i.e., income inequality and poverty). As with the level-1 equations, the relationship of level-2 predictors in explaining the outcome were investigated separately as well as together. For example, two-level models were tested utilizing income inequality and poverty (individually) and rotating social capital indicators as level-2 variables with differing combinations of level-1 predictors to explain self-reported hypertension, general health status, or mental distress.

Although multiple comparisons were analyzed, no statistical adjustments were made. In this study, the development of a priori hypotheses involved planned testing of theoretically-driven questions. As such, hypotheses testing of multiple comparisons was from a confirmatory mode, and not ad hoc, and therefore no adjustments were made. However, by not adjusting for multiple

tests within each model, an inflated Type I error rate may have resulted. This would have produced a rejection of the null hypothesis when it was actually true – in other words, concluding a statistically significant relationship existed when in reality it did not.

Additional effort was made to adjust for confounders. At level-1, a host of individual characteristics (i.e., gender, age, race/ethnicity, SES, education, marital status) were controlled for in order to isolate the relationships under study. At level-2, confounders under study included (for each community): median household income, percent unemployed, and percent who have completed high school or less. Because of the commonalities between the possible confounders, multicollinearity was thoroughly examined and controlled.

Chapter 4

The findings of this study are presented in the following chapter. Both univariate and bivariate statistical results are presented, followed by the results of multilevel models testing the study's hypotheses. For all variables and their associations, patterns of the pooled sample are described initially in order to give a global perspective of the relationships, followed by discussion of selected individual patterns by community. Finally, a summary of support for hypotheses is presented.

Univariate Analysis

Sociodemographic Factors

A description of the sociodemographic composition of the sample as a whole is presented in Table 4.1. Overall, the sample is 41% male and 59% female. One-third of the sample is between 45 and 64, with the next larger groups at age 20 to 34, 35 to 44, and 65 and older, respectively. In regards to race/ethnicity, the sample is 5.2% Hispanic, 84.1% White, 9.6% Black, and 6.4% other. The majority of individuals are married (54.3%), have completed college (32.6%), and have an annual income between \$20,000 and \$50,000 (42.9%). There is relatively small amount of missing data for these sociodemographic

variables, with means across communities ranging from none (e.g., sex) to 13.2% (e.g., income).

Table 4.1: Pooled Sample Sociodemographic Factors

Gender	Male	40.7%
	Female	59.3%
Age	20-34	24.9%
	35-44	22.5%
	45-64	33.8%
	65+	18.9%
Race/Ethnicity	Hispanic	5.2%
	White	84.1%
	Black	9.6%
	Other	6.4%
Income	< \$20,000	18.4%
	\$20,000 < \$50,000	42.9%
	\$50,000 < \$75,000	17.8%
	≥ \$75,000	20.9%
Education	< 12	9.9%
	12	30.7%
	13-15	26.9%
	16+	32.6%
Marital Status	Married	54.3%
	Separated/Widowed/Divorced	27.5%
	Never Married	18.2%

The distribution of these factors for individual communities is presented in Table 4.2. Comparing characteristics between communities reveals that there is a wide range of incomes represented across the sample, the highest proportion of residents earning \$75,000/year or more located in San Francisco (38.5%), Atlanta (36.3%), and St. Paul (34.4%). The three communities with the greatest

proportion of disadvantaged individuals earning less than \$20,000/year are Los Angeles (27.7%), Kalamazoo (27.6%), and Birmingham (26.3%). The sample is predominantly middle-aged, with the eldest population, on average, living in Yakima, Washington (32.2% age 65 and older) and the youngest residing in East Baton Rouge, Louisiana (36.3% between 20 and 34 years old).

Specifically, in comparing the 27 communities that make up the study sample, in regards to racial/ethnic composition, the most diverse community is Georgia (59.3% White, 34.1% Black, 6.6% other) and the most homogeneous is Oregon (96.9% White, 0% Black, 3.1% other).

Health Factors: Behavioral Variables

The distribution of health behaviors and outcomes for the pooled sample is displayed in Table 4.3. Overall, the sample is moderately active – over 85% report engaging in some activity, with less than half (44.8%) meeting current recommendations of regular exercise for sustaining health benefits (20 minutes of moderate activity most days). In addition, less than half of the pooled sample (42.1%) has a normal Body Mass Index; the majority of the sample is overweight or obese (58%), with only 4% missing data. Smoking is the least prevalent of the three risk behaviors under study, with under one-quarter (23.6%) engaging in this behavior. Approximately 26.8% of the sample suffers from hypertension. Although the majority report their general health to be very good or excellent (56.9%), one-third (34.2%) suffer from some form of mental distress each month.

In comparing the health activities of interest (physical activity, Body Mass Index, and smoking) between communities, there is a wide range in the frequency of behavior (Table 4.4).

The most active community is Boulder (CO), where 65% of the sample meets recommended levels of activity. The community with the least active inhabitants is East Baton Rouge (LA), with only 34% reporting engaging in the recommended levels of physical activity. In regards to Body Mass Index, Boulder (CO) is also the healthiest in the sample, with 60% of the residents reporting they are neither overweight nor obese, as opposed to Kanawha Valley (WV), where only 34% reported having a healthy Body Mass Index. In addition, the sample as a whole is largely comprised of non-smokers (76%), with a wide span of prevalence of 8.1% in Central Oregon to 27.4% of residents in the Kanawha Valley (WV) community.

Table 4.2: Frequencies of Sociodemographic Factors*

	N	Sex		Age				Race/Ethnicity			
		M	F	20-34	35-44	45-64	65+	Hisp	White	Black	Other
Birmingham (AL)	496	36.7	63.3	26.5	20.0	33.5	20.0	1.0	65.4	31.8	2.8
Maricopa (AZ)	856	42.5	57.5	26.8	21.7	32.1	19.5	13.8	82.4	4.1	13.6
Los Angeles (CA)	1002	43.1	56.9	31.7	22.4	32.0	13.9	34.4	75.8	11.4	12.8
San Diego (CA)	346	41.3	58.7	27.7	22.6	30.1	19.6	22.3	87.8	4.1	8.1
San Francisco (CA)	95	48.4	51.6	34.0	18.1	33.0	14.9	14.7	74.7	7.4	17.9
Boulder (CO)	124	44.4	55.7	30.8	25.8	29.2	14.2	7.3	90.2	0.8	9.0
Denver (CO)	228	39.9	60.1	34.6	19.4	31.8	14.3	22.8	76.1	12.0	12.0
Delaware (DE)	3514	38.7	61.3	23.3	21.8	33.3	21.6	2.8	81.0	14.6	4.5
Atlanta (GA)	646	40.3	59.8	28.5	27.7	31.6	12.2	2.0	59.3	34.1	6.6
Indiana (IN)	3993	40.4	59.6	26.4	21.4	33.1	19.1	2.9	90.7	6.2	3.0
E. Baton Rouge (LA)	461	39.9	60.1	36.3	16.8	32.7	14.3	1.7	63.8	32.1	4.0
Kalamazoo (MI)	89	44.9	55.1	36.1	15.1	30.2	18.6	2.3	85.4	9.0	5.6
Southeast (MI)	1554	38.2	61.8	24.7	23.9	34.1	17.4	3.2	72.8	20.5	6.7
St. Paul (MN)	844	41.2	58.8	26.2	27.0	31.0	15.8	1.9	91.7	4.4	3.9
Montana (MT)	3338	42.6	57.4	19.8	20.1	37.6	22.5	2.5	86.4	0.2	13.5
New Hampshire (NH)	4068	42.5	57.5	22.0	25.5	35.3	17.2	1.6	96.0	0.4	3.6
Central (NY)	106	37.7	62.3	20.0	23.0	34.0	23.0	2.8	93.3	4.8	1.9
Rochester (NY)	164	34.2	65.9	26.0	24.1	32.9	17.1	4.3	84.7	9.2	6.1
Winston-Salem (NC)	454	38.8	61.2	22.3	19.8	33.5	24.5	1.8	70.7	26.9	2.5
Greensboro (NC)	413	35.6	64.4	24.6	22.6	31.8	20.9	2.4	68.7	26.7	4.7
Cleveland (OH)	459	37.7	62.3	21.7	23.9	33.6	20.8	3.5	73.7	22.6	3.8
Cincinnati (OH)	1038	40.9	59.1	28.6	21.3	30.8	19.4	1.7	87.5	10.5	2.1
Central (OR)	99	47.5	52.5	29.8	17.0	34.0	19.2	5.1	96.9	0.0	3.1
York (PA)	127	40.9	59.1	30.9	15.5	40.7	13.0	2.4	92.9	4.0	3.2
Houston (TX)	802	41.0	59.0	31.5	25.8	30.7	12.0	24.9	68.7	16.0	15.3
Yakima (WA)	119	39.5	60.5	21.7	18.3	27.8	32.2	14.3	93.2	0.9	5.9
Kanawha Valley (WV)	497	40.4	59.6	22.5	21.1	36.4	20.0	1.0	93.6	3.8	2.6
TOTAL SAMPLE	25932	40.7	59.3	29.4	22.5	33.8	18.9	5.2	84.1	9.6	6.4

*NOTE: All numbers reflect percentages

Table 4.2: Frequencies of Sociodemographic Factors*

	N	Education				Income				Marital		
		<12th	12	13-15	16+	<20k	20<50k	50<75k	75k+	Married	Divorced/ Widowed/ Separated	Never
Birmingham (AL)	496	10.9	30.4	29.2	29.6	26.3	41.6	15.3	16.9	50.9	31.0	18.1
Maricopa (AZ)	856	8.9	25.5	33.3	32.3	13.2	45.8	17.5	23.5	54.4	27.7	17.9
Los Angeles (CA)	1002	17.0	21.4	26.5	35.2	27.7	31.5	16.5	24.4	46.0	28.2	25.8
San Diego (CA)	346	9.3	24.4	28.1	38.3	21.3	33.0	20.6	25.1	56.9	24.7	18.4
San Francisco (CA)	95	6.3	9.5	24.2	60.0	19.8	25.3	16.5	38.5	33.8	30.0	36.3
Boulder (CO)	124	3.2	12.1	25.8	58.9	14.2	40.7	16.8	28.3	57.4	15.7	27.0
Denver (CO)	228	17.1	18.0	21.1	43.9	24.2	40.6	16.4	18.8	39.5	29.4	31.2
Delaware (DE)	3514	9.3	34.5	25.7	30.5	20.1	40.4	17.9	21.6	53.5	27.7	18.8
Atlanta (GA)	646	7.0	17.5	22.5	53.0	11.4	36.3	16.1	36.3	49.5	22.2	28.3
Indiana (IN)	3993	10.4	38.7	24.1	26.8	18.7	47.0	18.7	15.6	56.7	28.1	15.3
E. Baton Rouge (LA)	461	7.6	24.6	28.1	39.7	20.1	41.7	16.6	21.6	48.1	25.2	26.7
Kalamazoo (MI)	89	12.4	23.6	22.5	41.6	27.6	38.2	6.6	27.6	54.7	23.3	22.1
Southeast (MI)	1554	8.5	27.7	30.5	33.4	14.8	39.7	18.8	26.8	50.6	27.8	21.6
St.Paul (MN)	844	5.1	21.1	32.0	41.8	8.2	36.6	20.7	34.4	55.4	25.1	19.5
Montana (MT)	3338	10.9	34.1	29.1	25.9	25.5	52.7	12.5	9.3	56.3	30.4	13.3
New Hampshire (NH)	4068	7.4	29.8	26.5	36.4	12.3	40.4	21.6	25.7	59.4	25.2	15.4
Central (NY)	106	7.7	26.0	27.9	38.5	19.8	34.1	22.0	24.2	51.5	30.1	18.5
Rochester (NY)	164	5.5	26.2	28.1	40.2	16.3	38.8	15.0	29.9	50.9	25.8	23.3
Winston-Salem (NC)	454	13.5	27.0	24.6	35.0	20.8	46.8	17.8	14.6	49.9	31.2	18.9
Greensboro (NC)	413	11.4	25.6	26.3	36.7	20.8	45.2	16.0	18.1	46.4	31.2	22.4
Cleveland (OH)	459	8.3	30.1	28.2	33.4	16.3	49.0	14.5	20.2	46.8	29.2	24.1
Cincinnati (OH)	1038	10.6	32.6	26.3	30.5	17.4	42.6	17.9	22.1	52.4	27.0	20.7
Central (OR)	99	3.0	38.4	34.3	24.2	12.4	55.1	16.9	15.7	62.0	22.8	15.2
York (PA)	127	8.7	40.9	23.6	26.8	16.4	50.9	23.3	9.5	61.8	23.6	14.6
Houston (TX)	802	15.7	22.0	23.7	38.7	19.7	38.5	17.2	24.6	51.7	26.7	21.6
Yakima (WA)	119	17.7	35.3	26.9	20.2	25.2	48.5	17.5	8.7	58.0	26.9	15.1
Kanawha Valley (WV)	497	14.5	36.9	26.4	22.2	22.9	46.9	16.8	13.4	57.1	27.6	15.3
TOTAL SAMPLE	25932	9.9	30.7	26.9	32.6	18.4	42.9	17.8	20.9	54.3	27.5	18.2

*NOTE: All numbers reflect percentages

Table 4.3: Pooled Sample Health Behavior and Outcome Factors

Physical Activity	Inactive	15.1%
	Some activity	40.1%
	Meets recommendations	44.8%
Body Mass Index	Normal	42.1%
	Overweight	36.1%
	Obese	21.8%
Smoking	Yes	23.6%
	No	76.4%
Hypertension	Presence	26.8%
	Absence	73.2%
General Health	Excellent	22.6%
	Very Good	34.3%
	Good	28.4%
	Fair	10.8%
	Poor	3.8%
Days of Mental Distress per Month	0	65.8%
	1	3.5%
	2	6.3%
	3	3.3%
	4-7	7.8%
	8-15	6.0%
	> 15	7.3%

Table 4.4: Comparison of Health Behavior Frequencies

	Physical Activity			Body Mass Index			Smoke	
	Inactive	Some Activity	Meets Recommendations	Normal	Overweight	Obese	Yes	No
Birmingham (AL)	16.21%	41.89%	41.89%	39.17%	34.79%	26.04%	22.67%	77.33%
Maricopa (AZ)	10.97%	37.53%	51.50%	45.01%	37.66%	17.33%	20.84%	79.16%
Los Angeles (CA)	13.10%	44.28%	42.62%	46.87%	32.72%	20.41%	16.00%	84.00%
San Diego (CA)	11.47%	40.88%	47.65%	40.29%	38.24%	21.47%	14.83%	85.17%
San Francisco (CA)	10.00%	36.67%	53.33%	55.91%	30.11%	13.98%	20.00%	80.00%
Boulder (CO)	5.83%	29.17%	65.00%	60.33%	32.23%	7.44%	14.52%	85.48%
Denver (CO)	11.42%	38.81%	49.77%	56.94%	28.71%	14.35%	24.12%	75.88%
Delaware (DE)	17.55%	41.83%	40.63%	40.67%	37.48%	21.85%	24.63%	75.37%
Atlanta (GA)	19.77%	37.87%	42.36%	47.66%	32.47%	19.87%	19.00%	81.00%
Indiana (IN)	14.13%	41.11%	44.76%	40.25%	35.59%	24.16%	27.20%	72.80%
E. Baton Rouge (LA)	28.88%	37.23%	33.89%	43.99%	35.83%	20.18%	18.70%	81.30%
Kalamazoo (MI)	11.76%	48.24%	40.00%	45.35%	39.53%	15.12%	17.98%	82.02%
Southeast (MI)	14.70%	41.87%	43.43%	39.76%	34.22%	26.06%	23.92%	76.08%
St.Paul (MN)	7.34%	42.46%	50.20%	44.23%	36.12%	19.66%	21.26%	78.74%
Montana (MT)	16.68%	35.70%	47.62%	40.66%	37.98%	21.36%	23.90%	76.10%
New Hampshire (NH)	11.89%	38.11%	50.00%	43.94%	36.58%	19.48%	23.61%	76.39%
Central (NY)	19.79%	35.42%	44.79%	46.08%	31.37%	22.55%	23.81%	76.19%
Rochester (NY)	14.19%	42.57%	43.24%	43.95%	36.31%	19.75%	24.54%	75.46%
Winston-Salem (NC)	17.73%	43.64%	38.64%	43.02%	34.42%	22.56%	25.00%	75.00%
Greensboro (NC)	18.16%	45.52%	36.32%	46.67%	31.54%	21.79%	22.68%	77.32%
Cleveland (OH)	15.90%	43.86%	40.24%	38.76%	36.01%	25.23%	26.36%	73.64%
Cincinnati (OH)	21.34%	40.62%	38.04%	41.76%	36.23%	22.01%	26.06%	73.94%
Central (OR)	7.22%	35.05%	57.73%	44.33%	41.24%	14.43%	8.08%	91.92%
York (PA)	10.08%	51.26%	38.66%	35.20%	38.40%	26.40%	22.83%	77.17%
Houston (TX)	15.19%	42.67%	42.14%	41.78%	35.41%	22.81%	21.13%	78.87%
Yakima (WA)	6.36%	41.82%	51.82%	34.51%	35.40%	30.09%	21.85%	78.15%
Kanawha Valley (WV)	18.70%	38.70%	42.61%	34.04%	39.32%	26.64%	27.42%	72.58%
Pooled Sample	15.07%	40.09%	44.84%	42.12%	36.10%	21.79%	23.58%	76.42%

Health Factors: Outcome Variables

A primary outcome of interest is a CVD-related factor, hypertension. In the overall sample, an average of 26.8% of participants reported that a professional has informed them that they have high blood pressure; the prevalence of self-reported hypertension ranged from 16.1% (Boulder) to 35.8% (Kanawha Valley). The five communities with the highest rates of hypertension are Kanawha Valley (WV), Birmingham (AL), Yakima (WA), York (PA), and Winston-Salem (NC). Table 4.5 compares each individual community's rate of disease.

Additional outcomes of interest include general health and mental distress. Approximately 14.6% of the sample rates their health as poor or fair. The communities with the highest frequency of residents reporting fair or poor health are: Kanawha Valley, WV (24.59%), Birmingham, AL (21.97%), and Denver, CO (19.30%). However, most residents report their general health status to be good to excellent (mean 3.61, sd 1.07); differences in distributions reflect a negative skew. Slight platykurtosis is observed. Descriptive statistics for all 27 communities are presented in Table 4.6.

Table 4.5: Comparison of Hypertension Outcome Frequency

Community	Prevalence of Hypertension ¹	Se	95% CI
Birmingham (AL)	33.7%	0.0212	(29.5%, 37.9%)
Maricopa (AZ)	24.1%	0.0146	(21.2%, 27%)
Los Angeles (CA)	23.3%	0.0134	(20.1%, 26%)
San Diego (CA)	26.3%	0.0237	(21.7%, 30.9%)
San Francisco (CA)	17.9%	0.0393	(10.2%, 25.6%)
Boulder (CO)	16.1%	0.0330	(9.6%, 22.6%)
Denver (CO)	21.9%	0.0271	(16.1%, 26.7%)
Delaware (DE)	29.7%	0.0077	(28.2%, 31.2%)
Atlanta (GA)	21.4%	0.0161	(18.2%, 24.6%)
Indiana (IN)	26.9%	0.0070	(25.5%, 28.3%)
E. Baton Rouge (LA)	27.2%	0.0207	(23.1%, 31.3%)
Kalamazoo (MI)	24.7%	0.0457	(15.7%, 33.7%)
Southeast (MI)	28.7%	0.0115	(26.5%, 31%)
St. Paul (MN)	20.2%	0.0138	(17.5%, 22.9%)
Montana (MT)	29.9%	0.0079	(28.4%, 31.5%)
New Hampshire (NH)	23.7%	0.0067	(22.4%, 25%)
Central (NY)	28.6%	0.0439	(20%, 37.2%)
Rochester (NY)	22.0%	0.0324	(15.7%, 28.3%)
Winston-Salem (NC)	31.8%	0.0219	(27.5%, 36.1%)
Greensboro (NC)	26.6%	0.0217	(22.3%, 30.9%)
Cleveland (OH)	24.8%	0.0202	(20.9%, 28.8%)
Cincinnati (OH)	26.4%	0.0137	(23.7%, 29.1%)
Central (OR)	25.3%	0.0437	(16.7%, 33.9%)
York (PA)	31.8%	0.0414	(23.7%, 39.9%)
Houston (TX)	23.6%	0.0150	(20.7%, 26.5%)
Yakima (WA)	32.2%	0.0428	(23.8%, 40.6%)
Kanawha Valley (WV)	35.8%	0.0215	(31.6%, 40%)
Pooled Sample	26.8%	0.0143	(24.0%, 29.6%)

¹where higher number reflects higher frequency of hypertension

Table 4.6: Comparison of Descriptive Statistics of General Health

Community	Excellent	Very Good	Good	Fair	Poor	Mean ¹	sd	Skew	Kurtosis
Birmingham (AL)	19.56%	33.06%	25.40%	15.12%	6.85%	3.43	1.16	-0.41	-0.66
Maricopa (AZ)	24.82%	32.71%	27.06%	11.18%	4.24%	3.63	1.10	-0.50	-0.44
Los Angeles (CA)	24.05%	31.94%	28.44%	10.98%	4.59%	3.60	1.10	-0.47	-0.44
San Diego (CA)	30.92%	34.68%	23.70%	9.83%	0.87%	3.85	1.00	-0.50	-0.58
San Francisco (CA)	26.32%	32.63%	24.21%	12.63%	4.21%	3.64	1.13	-0.52	-0.51
Boulder (CO)	33.06%	39.52%	24.19%	2.42%	0.81%	4.02	0.86	-0.57	0.02
Denver (CO)	20.18%	30.26%	30.26%	13.16%	6.14%	3.45	1.14	-0.37	-0.55
Delaware (DE)	20.23%	34.71%	29.98%	11.57%	3.51%	3.57	1.05	-0.41	-0.39
Atlanta (GA)	29.04%	37.73%	23.76%	7.45%	2.02%	3.84	0.99	-0.64	-0.07
Indiana (IN)	19.26%	34.39%	31.95%	10.13%	4.26%	3.54	1.05	-0.43	-0.26
E. Baton Rouge (LA)	31.89%	35.36%	21.91%	8.46%	2.39%	3.86	1.04	-0.69	-0.14
Kalamazoo (MI)	20.22%	38.20%	31.46%	6.74%	3.37%	3.65	0.99	-0.54	0.17
Southeast (MI)	20.04%	33.38%	30.35%	12.24%	3.99%	3.53	1.07	-0.39	-0.44
St.Paul (MN)	22.87%	40.17%	27.01%	7.58%	2.37%	3.74	0.97	-0.57	0.01
Montana (MT)	20.28%	34.02%	29.10%	12.15%	4.44%	3.54	1.08	-0.43	-0.43
New Hampshire (NH)	28.06%	36.63%	25.01%	7.56%	2.73%	3.80	1.02	-0.64	-0.07
Central (NY)	25.71%	34.29%	30.48%	5.71%	3.81%	3.72	1.03	-0.59	0.08
Rochester (NY)	21.95%	40.24%	25.00%	11.59%	1.22%	3.70	0.98	-0.44	-0.44
Winston-Salem (NC)	20.84%	31.49%	29.27%	12.42%	5.99%	3.49	1.13	-0.42	-0.51
Greensboro (NC)	21.84%	35.68%	24.03%	13.35%	5.10%	3.56	1.12	-0.51	-0.49
Cleveland (OH)	23.58%	32.53%	26.20%	12.88%	4.80%	3.57	1.12	-0.46	-0.54
Cincinnati (OH)	22.57%	30.67%	31.53%	11.67%	3.57%	3.57	1.07	-0.35	-0.51
Central (OR)	28.28%	37.37%	24.24%	7.07%	3.03%	3.81	1.03	-0.70	0.11
York (PA)	14.17%	38.58%	29.92%	13.39%	3.94%	3.46	1.02	-0.43	-0.27
Houston (TX)	24.13%	28.25%	29.38%	15.38%	2.88%	3.55	1.10	-0.26	-0.80
Yakima (WA)	21.01%	27.73%	33.61%	14.29%	3.36%	3.49	1.08	-0.21	-0.64
Kanawha Valley (WV)	17.54%	30.04%	27.82%	15.32%	9.27%	3.31	1.20	-0.33	-0.74
Pooled Sample	22.63%	34.34%	28.44%	10.75%	3.84%	3.61	1.07	-0.47	-0.36

¹where higher number indicates better self-reported health

Table 4.7: Comparison of Descriptive Statistics of Days of Mental Distress per Month

Community	0	1	2	3	4-7	8-15	>15	Mean¹	sd	Skew	Kurtosis
Birmingham (AL)	60.41%	4.08%	6.94%	4.08%	9.60%	4.90%	9.98%	4.06	8.14	2.27	4.03
Maricopa (AZ)	63.63%	3.91%	7.11%	4.62%	8.41%	6.17%	6.17%	3.28	7.19	2.76	6.94
Los Angeles (CA)	62.34%	3.60%	7.49%	4.10%	9.40%	5.90%	7.20%	3.48	7.40	2.63	6.12
San Diego (CA)	66.96%	2.61%	7.25%	3.77%	8.12%	5.80%	5.51%	3.01	6.94	2.94	8.10
San Francisco (CA)	49.47%	3.16%	3.16%	10.53%	13.69%	9.47%	10.52%	5.32	8.80	1.96	2.81
Boulder (CO)	60.16%	5.69%	9.76%	4.88%	10.57%	4.87%	4.06%	2.65	5.68	3.03	9.49
Denver (CO)	59.47%	4.85%	7.05%	3.08%	8.81%	7.92%	8.81%	4.14	8.17	2.28	4.16
Delaware (DE)	67.55%	3.72%	5.30%	3.05%	7.51%	5.21%	7.64%	3.44	7.78	2.61	5.69
Atlanta (GA)	62.75%	3.76%	6.42%	2.66%	8.45%	8.77%	7.20%	3.80	7.69	2.39	4.89
Indiana (IN)	62.05%	3.83%	6.64%	3.37%	9.05%	7.10%	8.01%	3.80	7.81	2.43	4.91
E. Baton Rouge (LA)	72.03%	2.86%	5.51%	1.98%	5.72%	5.94%	5.94%	2.93	7.09	2.83	7.19
Kalamazoo (MI)	67.05%	3.41%	7.95%	3.41%	10.23%	4.55%	6.82%	2.77	6.37	3.00	8.86
Southeast (MI)	59.22%	3.40%	7.78%	3.73%	8.89%	8.37%	8.65%	4.18	8.14	2.25	4.02
St. Paul (MN)	54.46%	7.47%	7.95%	5.18%	10.84%	7.58%	6.50%	3.64	7.04	2.52	5.81
Montana (MT)	76.14%	1.88%	4.31%	1.85%	5.52%	4.45%	5.85%	2.70	7.10	3.03	8.24
New Hampshire (NH)	66.42%	3.53%	7.03%	3.40%	7.51%	5.52%	6.64%	3.15	7.21	2.80	7.03
Central (NY)	60.38%	6.60%	10.38%	0.94%	8.49%	6.60%	6.60%	3.40	7.41	2.76	6.95
Rochester (NY)	61.96%	4.29%	7.36%	2.45%	8.59%	7.36%	7.97%	3.51	6.97	2.34	4.82
Winston-Salem (NC)	74.55%	1.79%	4.46%	3.57%	5.13%	3.12%	7.36%	2.93	7.57	2.92	7.29
Greensboro (NC)	71.22%	2.73%	4.71%	2.23%	6.70%	4.96%	7.45%	3.21	7.63	2.73	6.39
Cleveland (OH)	60.79%	4.41%	6.17%	5.51%	8.37%	7.04%	7.71%	3.71	7.60	2.48	5.25
Cincinnati (OH)	66.86%	2.63%	4.97%	3.31%	6.82%	6.63%	8.77%	3.85	8.27	2.39	4.48
Central (OR)	66.33%	5.10%	8.16%	3.06%	5.10%	3.06%	9.18%	3.53	8.45	2.64	5.57
York (PA)	53.17%	3.17%	11.90%	0.79%	12.69%	6.35%	11.90%	3.56	7.53	2.53	5.56
Houston (TX)	65.70%	4.18%	7.59%	4.30%	6.96%	5.57%	5.70%	2.95	6.83	2.94	8.08
Yakima (WA)	70.34%	2.54%	1.69%	2.54%	6.77%	8.47%	7.62%	3.68	7.86	2.38	4.80
Kanawha Valley (WV)	62.70%	1.84%	5.12%	2.87%	7.58%	6.96%	12.89%	5.09	9.53	1.86	1.98
Pooled Sample	65.77%	3.48%	6.26%	3.29%	7.82%	6.03%	7.32%	3.45	7.59	2.60	5.79

¹where higher number indicates higher mean number of days in past month self-reported mental health not good

In addition to general health status, mental distress was evaluated. As with the other outcome variables, there is only a very small amount of data missing (less than 2%). The overall sample reports a mean of 3.45 (range 2.65 – 5.32) days in the past month when their mental health was not good. The distributions of every community are positively skewed and leptokurtic (Table 4.7). Communities with the highest frequencies of residents who report their mental health is not good half of every month or more are: Kanawha Valley, WV (12.9%), York, PA (11.9%), and San Francisco, CA (10.5%). Table 4.8 compares the relative ranking of the communities in terms of each outcome. There is some consistency in the ranking of communities with respect to all three outcomes at the extreme ends of the distributions (which communities have the worst health and which have the best). However, there is not a stable pattern of ranking the burden of disease towards the middle of the distributions of investigated outcomes.

Social Contextual Factors

Descriptive statistics of both pooled and community-specific individual indices are presented in Table 4.9. There is very little missing data for any one index, with less than 1% for any community on every index. Scores for most of the indices have been standardized on national norms by the original investigators. Therefore, it is consistent that the communities' pooled mean

levels of social trust (0.02, 0.69), informal social engagement (-0.002, 0.64), and organizational activism or formal social engagement (0.068, 1.036) remain around zero, with a standard deviation just under one. As the mutual aid index is more of a count-based dimension, similar standardization has not been used (pooled mean 5.243, sd 4.312). Descriptions of the differences between individual communities are found in examining the type of distribution of scores. Overall, the shape of the distribution of scores for the social trust index pooled across communities is slightly negatively skewed (-0.892) and leptokurtic (0.517). Individual communities distributions range from moderate deviations (New Hampshire and Boulder, CO) to relatively normal shape (Houston, TX). The shape of pooled scores for informal social participation is slightly positively skewed (0.87) and barely leptokurtic (0.177). There are only very small distinctions between community-level distributions, with Atlanta, GA the most positively skewed (1.05) and slightly leptokurtic (0.48). The communities' organizational activism score distributions are very similar, with all communities both positively skewed and sharply peaked, which reflects a concentration of lower scores indicating less overall engagement in formal participation. Mutual aid is also consistent with respect to skewness, with Los Angeles the most deviant (positive skew .99). The peakedness of the distributions, however, are more diverse, ranging from flatter (Winston-Salem, NC) to more peaked (Yakima, WA).

Table 4.8: Comparison of Community Rankings on Outcome Variables

Community	Hypertension ¹	General Health ²	Mental Distress ³
Birmingham (AL)	2 nd	2 nd	5 th
Maricopa (AZ)	18 th	16 th	18 th
Los Angeles (CA)	21 st	15 th	15 th
San Diego (CA)	14 th	25 th	21 st
San Francisco (CA)	26 th	17 th	1 st
Boulder (CO)	27 th	27 th	27 th
Denver (CO)	23 rd	3 rd	4 th
Delaware (DE)	7 th	12 th	16 th
Atlanta (GA)	24 th	24 th	8 th
Indiana (IN)	11 th	9 th	7 th
E. Baton Rouge (LA)	10 th	26 th	24 th
Kalamazoo (MI)	17 th	18 th	25 th
Southeast (MI)	8 th	7 th	3 rd
St. Paul (MN)	25 th	21 st	11 th
Montana (MT)	6 th	8 th	26 th
New Hampshire (NH)	19 th	22 nd	20 th
Central (NY)	9 th	20 th	17 th
Rochester (NY)	22 nd	19 th	14 th
Winston-Salem (NC)	4 th	6 th	23 rd
Greensboro (NC)	12 th	11 th	19 th
Cleveland (OH)	16 th	14 th	9 th
Cincinnati (OH)	13 th	13 th	6 th
Central (OR)	15 th	23 rd	13 th
York (PA)	5 th	4 th	12 th
Houston (TX)	20 th	10 th	22 nd
Yakima (WA)	3 rd	5 th	10 th
Kanawha Valley (WV)	1 st	1 st	2 nd

¹where higher number reflects higher frequency of hypertension

²where higher number indicates worse self-reported health

³where higher number indicates higher mean number of days in past month self-reported mental health not good

Table 4.9: Comparison of Social Capital Descriptive Statistics

	Social Trust¹				Informal Participation¹				Organizational Activism¹				Mutual Aid¹			
	Mean	sd	Skew	Kurtosis	Mean	sd	Skew	Kurtosis	Mean	sd	Skew	Kurtosis	Mean	sd	Skew	Kurtosis
Birmingham (AL)	1.99	0.81	0.02	-1.49	1.98	0.81	0.04	-1.47	2.04	0.84	-0.08	-1.57	2.08	0.80	-0.15	-1.42
Maricopa (AZ)	1.99	0.83	0.02	-1.54	2.01	0.81	-0.02	-1.46	1.94	0.83	0.11	-1.55	1.97	0.82	0.05	-1.51
Los Angeles (CA)	1.74	0.80	0.50	-1.28	1.89	0.83	0.20	-1.51	1.93	0.85	0.14	-1.61	1.96	0.82	0.08	-1.51
San Diego (CA)	1.98	0.83	0.04	-1.54	1.96	0.81	0.07	-1.49	2.01	0.83	-0.03	-1.56	1.97	0.80	0.05	-1.43
San Francisco (CA)	1.96	0.83	0.08	-1.54	2.04	0.82	-0.08	-1.52	2.07	0.82	-0.12	-1.51	2.02	0.80	-0.04	-1.42
Boulder (CO)	2.27	0.76	-0.50	-1.11	2.10	0.81	-0.18	-1.44	2.17	0.82	-0.31	-1.46	2.10	0.81	-0.19	-1.47
Denver (CO)	1.94	0.81	0.11	-1.46	2.00	0.81	0.00	-1.49	2.05	0.87	-0.09	-1.67	2.05	0.81	-0.10	-1.46
Delaware (DE)	2.06	0.82	-0.11	-1.51	2.02	0.80	-0.04	-1.45	2.07	0.85	-0.13	-1.62	2.05	0.83	-0.10	-1.55
Atlanta (GA)	1.82	0.83	0.34	-1.45	1.90	0.82	0.19	-1.49	2.08	0.81	-0.14	-1.45	2.16	0.80	-0.29	-1.39
Indiana (IN)	2.14	0.81	-0.25	-1.44	2.13	0.78	-0.24	-1.33	1.99	0.85	0.03	-1.63	2.05	0.81	-0.08	-1.45
E. Baton Rouge (LA)	1.86	0.80	0.25	-1.41	2.09	0.83	-0.16	-1.51	2.17	0.81	-0.31	-1.42	2.18	0.78	-0.33	-1.30
Kalamazoo (MI)	2.16	0.82	-0.31	-1.45	2.19	0.80	-0.37	-1.36	2.06	0.83	-0.12	-1.55	2.12	0.84	-0.22	-1.56
Southeast (MI)	1.96	0.84	0.08	-1.58	2.09	0.83	-0.16	-1.53	2.02	0.84	-0.03	-1.57	2.05	0.80	-0.09	-1.42
St. Paul (MN)	2.30	0.77	-0.57	-1.11	2.01	0.81	-0.02	-1.45	2.04	0.82	-0.07	-1.50	2.19	0.81	-0.35	-1.40
Montana (MT)	2.35	0.75	-0.67	-0.92	2.09	0.79	-0.16	-1.37	2.10	0.86	-0.19	-1.61	2.09	0.83	-0.17	-1.53
New Hampshire (NH)	2.33	0.76	-0.64	-1.00	2.06	0.80	-0.11	-1.43	2.06	0.84	-0.12	-1.58	2.06	0.82	-0.12	-1.51
Central (NY)	2.10	0.81	-0.18	-1.47	2.11	0.82	-0.20	-1.49	2.03	0.85	-0.06	-1.61	2.10	0.80	-0.18	-1.43
Rochester (NY)	2.01	0.83	-0.01	-1.56	2.03	0.80	-0.06	-1.45	1.92	0.86	0.15	-1.63	2.00	0.80	0.00	-1.45
Winston-Salem (NC)	1.98	0.83	0.04	-1.53	1.89	0.82	0.20	-1.47	2.04	0.84	-0.08	-1.56	2.18	0.81	-0.34	-1.40
Greensboro (NC)	1.96	0.82	0.08	-1.52	1.96	0.81	0.08	-1.49	2.06	0.85	-0.12	-1.61	2.15	0.81	-0.28	-1.42

Table 4.9: Comparison of Social Capital Descriptive Statistics

	Social Trust¹				Informal Participation¹				Organizational Activism¹				Mutual Aid¹			
	Mean	sd	Skew	Kurtosis	Mean	Sd	Skew	Kurtosis	Mean	sd	Skew	Kurtosis	Mean	sd	Skew	Kurtosis
Cleveland (OH)	1.90	0.83	0.19	-1.52	2.00	0.83	0.00	-1.53	1.99	0.84	0.02	-1.58	1.94	0.82	0.12	-1.50
Cincinnati (OH)	2.08	0.80	-0.15	-1.42	2.04	0.82	-0.07	-1.49	2.01	0.84	-0.02	-1.58	2.06	0.81	-0.10	-1.46
Central (OR)	2.18	0.79	-0.33	-1.34	2.01	0.80	-0.03	-1.43	2.04	0.82	-0.07	-1.51	1.93	0.82	0.13	-1.50
York (PA)	2.18	0.78	-0.32	-1.29	2.06	0.81	-0.11	-1.48	1.92	0.83	0.15	-1.54	2.04	0.82	-0.08	-1.52
Houston (TX)	1.75	0.81	0.50	-1.30	1.87	0.83	0.24	-1.50	1.84	0.82	0.30	-1.45	1.89	0.82	0.20	-1.48
Yakima (WA)	1.99	0.82	0.02	-1.51	2.01	0.85	-0.01	-1.61	1.91	0.86	0.17	-1.64	1.92	0.84	0.15	-1.55
Kanawha Valley (WV)	2.07	0.85	-0.13	-1.60	2.03	0.79	-0.06	-1.39	2.00	0.86	0.00	-1.64	1.99	0.81	0.02	-1.47
Mean	2.04	0.81	-0.07	-1.40	2.02	0.81	-0.04	-1.47	2.02	0.84	-0.04	-1.56	2.05	0.81	-0.09	-1.46

¹where 1 = low, 2 = moderate, 3 = high

Table 4.10: Community Ranking on Social Capital Variables

Rank	Social Trust	Informal Social Participation	Organizational Activism	Mutual Aid
1	Montana MT	Kalamazoo MI	Boulder CO	Winston-Salem NC
2	New Hampshire NH	Indiana IN	East Baton Rouge LA	Greensboro NC
3	St.Paul MN	East Baton Rouge LA	Greensboro NC	East Baton Rouge LA
4	Boulder CO	Central New York NY	Montana MT	St.Paul MN
5	York PA	SE Michigan MI	Central Oregon OR	Atlanta GA
6	Central Oregon OR	Boulder CO	Birmingham AL	Kalamazoo MI
7	Kalamazoo MI	Montana MT	Delaware DE	Boulder CO
8	Indiana IN	Yakima WA	New Hampshire NH	Birmingham AL
9	Cincinnati OH	New Hampshire NH	San Francisco CA	Delaware DE
10	Central New York NY	Cincinnati OH	Kalamazoo MI	Montana MT
11	Delaware DE	Central Oregon OR	Denver CO	Cincinnati OH
12	Kanawha Valley WV	San Francisco CA	Atlanta GA	New Hampshire NH
13	Winston-Salem NC	Rochester NY	St.Paul MN	Central New York NY
14	Rochester NY	Arizona AZ	Central New York NY	SE Michigan MI
15	Birmingham AL	York PA	Winston-Salem NC	Denver CO
16	Arizona AZ	Kanawha Valley WV	Cleveland OH	Indiana IN
17	SE Michigan MI	Denver CO	Kanawha Valley WV	York PA
18	Yakima WA	Delaware DE	Cincinnati OH	San Francisco CA
19	Greensboro NC	Cleveland OH	SE Michigan MI	Rochester NY
20	San Diego CA	St.Paul MN	Indiana IN	Kanawha Valley WV
21	San Francisco CA	Birmingham AL	San Diego CA	Arizona AZ
22	Denver CO	Greensboro NC	Rochester NY	Los Angeles CA
23	East Baton Rouge LA	San Diego CA	Yakima WA	Central Oregon OR
24	Cleveland OH	Atlanta GA	Los Angeles CA	San Diego CA
25	Atlanta GA	Winston-Salem NC	York PA	Cleveland OH
26	Houston TX	Los Angeles CA	Arizona AZ	Yakima WA
27	Los Angeles CA	Houston TX	Houston TX	Houston TX

Comparisons of the communities on the four indices related to social capital are displayed in Table 4.10, in which the 27 communities are ranked. The community with the most social trust is Montana, followed by New Hampshire and St.Paul (MN). Residents in Kalamazoo (MI), Indiana, and East Baton Rouge (LA) are the most active in informal social engagement whereas organizational activism or formal social engagement is at its greatest in Boulder (CO), East

Baton Rouge (LA), and Greensboro (NC). Those living in Winston-Salem (NC), Greensboro (NC), and East Baton Rouge (LA) report the highest amount of mutual aid within their communities. Houston (TX) is consistently last on almost every dimension of social capital in this study.

Social Structural Factors

Social structural variables, including income inequality (Gini Coefficient) and absolute deprivation (200% below FPL), are reported at the state-level for each community in Table 4.11. The mean Gini across communities is .453 (range .414 - .499), indicating substantively significant relative deprivation. The shape of the distribution is both positively skewed and platykurtic and is illustrated in Figure 4.1. The most income inequality is observed in New York, Louisiana, and California, with the least in New Hampshire. In regards to absolute deprivation, approximately 30% of residents within the sample live at or below 200% FPL on average. Figure 4.2 displays the distribution of poverty rates. Communities range from the most impoverished areas, Louisiana (40.4%) and West Virginia (40.3%), to least deprived locales, New Hampshire (19%) and Minnesota (21.6%).

Table 4.11: Comparison of Social Structural Characteristics

	Gini Coefficient	Percent Below 200% FPL	Median HH Income	% Unemployed	% Completed High School or Less
Birmingham (AL)	0.475	36.1	\$34,135	16.9	55.1
Maricopa (AZ)	0.450	33.5	\$40,558	15.2	43.3
Los Angeles (CA)	0.475	33.1	\$47,493	19.5	43.3
San Diego (CA)	0.475	33.1	\$47,493	19.5	43.3
San Francisco (CA)	0.475	33.1	\$47,493	19.5	43.3
Boulder (CO)	0.438	24.2	\$47,203	13.2	36.2
Denver (CO)	0.438	24.2	\$47,203	13.2	36.2
Delaware (DE)	0.429	23.2	\$47,381	15.1	48.8
Atlanta (GA)	0.461	30.5	\$42,433	15.7	50.1
Indiana (IN)	0.424	25.8	\$41,567	14.0	55.1
E. Baton Rouge (LA)	0.483	40.4	\$32,566	18.4	57.6
Kalamazoo (MI)	0.440	25.4	\$44,667	16.7	47.8
Southeast (MI)	0.440	25.4	\$44,667	16.7	47.8
St. Paul (MN)	0.426	21.6	\$47,111	13.7	40.9
Montana (MT)	0.436	37.1	\$33,024	18.6	44.2
New Hampshire (NH)	0.414	19.0	\$49,467	14.0	42.7
Central (NY)	0.499	30.5	\$43,393	20.9	48.7
Rochester (NY)	0.499	30.5	\$43,393	20.9	48.7
Winston-Salem (NC)	0.452	30.5	\$39,184	15.9	50.2
Greensboro (NC)	0.452	30.5	\$39,184	15.9	50.2
Cleveland (OH)	0.441	26.4	\$40,956	14.2	53.2
Cincinnati (OH)	0.441	26.4	\$40,956	14.2	53.2
Central (OR)	0.438	29.6	\$40,916	18.5	41.1
York (PA)	0.452	27.4	\$40,106	17.5	56.1
Houston (TX)	0.470	36.0	\$39,927	16.5	49.1
Yakima (WA)	0.436	25.9	\$45,776	18.3	37.8
Kanawha Valley (WV)	0.468	40.3	\$29,696	18.3	64.2

Figure 4.1: Gini Coefficient Distribution

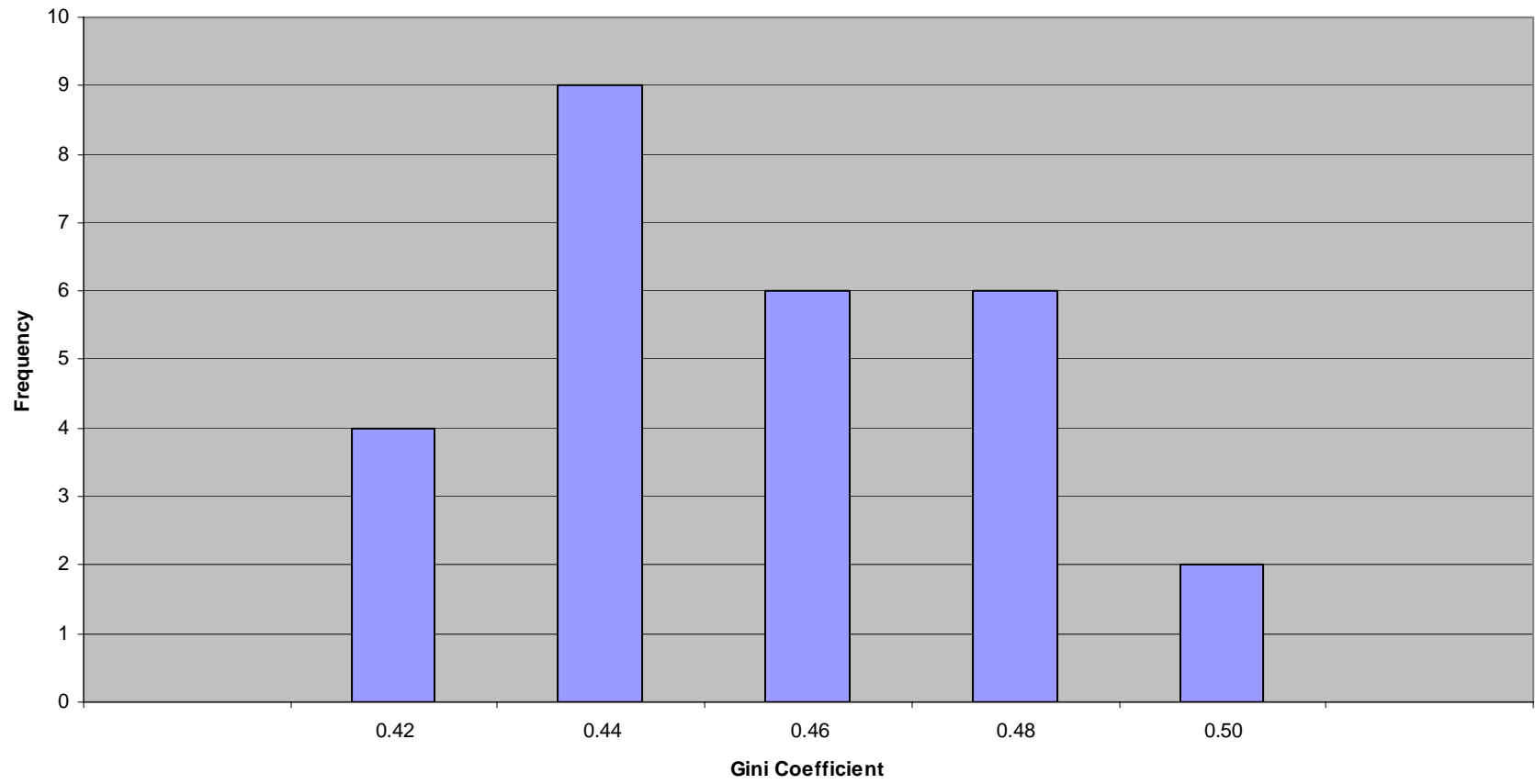
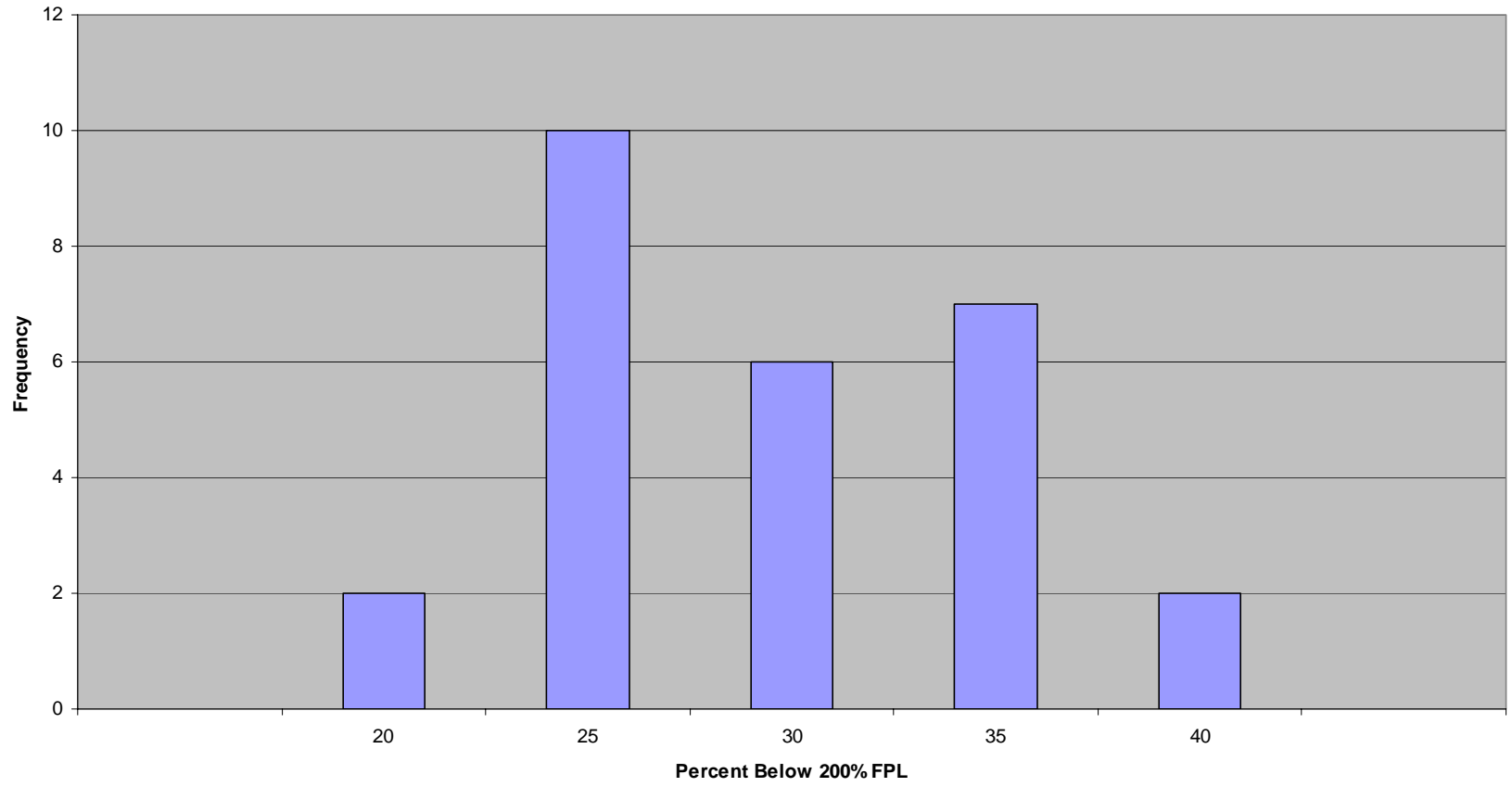


Figure 4.2: Below 200% FPL Distribution



Bivariate Analysis

Relationships between sociodemographic characteristics of the sample are presented in Table 4.12. In general, the majority of associations between factors are statistically significant at $p < .0001$ level. Specifically, men and women statistically significantly differ on almost all other factors ($p < .001$), with the exception of the gender distribution of Hispanics. Racial/ethnic differences are also apparent with respect to age, education, income and marital status ($p < .0001$).

There is also a consistent pattern of statistically significant associations between sociodemographic factors and the frequencies of health behaviors (Table 4.13). Those engaging in health behaviors such as physical activity or diet differ significantly based upon personal characteristics. For example, both education and income are positively correlated with physical activity ($r_{sp} = .14$, $p < .0001$ for both), with those who are more educated and have more income reporting more activity. A statistically significant negative association is reported for the same factors and Body Mass Index, with the more educated and wealthier individuals reporting lower Body Mass Index ($r_{sp} = -.09$, $p < .0001$ and $r_{sp} = -.06$, $p < .0001$, respectively). In regards to smoking behavior, significant associations are observed with all factors, except for race/ethnicity. Generally, smokers tend to be white, male, younger, less educated, have less income, and not married.

The majority of the sample engages in multiple risk behaviors, with only 21.3% both active and not overweight. In addition, slightly more smokers than

non-smokers are overweight or obese (52.7% vs. 47.4%) and do not meet recommended physical activity levels (55.4% vs. 44.6%). Although there is some evidence for the clustering effect of behavior in this sample, the associations among risk factors are not all positive (Table 4.14). Specifically, those who are inactive have higher Body Mass Index ($r_{sp} = -.12$, $p < .0001$), however are less likely to smoke with an OR 0.95 (95% CI 0.91 - 0.99). Moreover, smokers tend to have lower Body Mass Index with OR 0.82 (95% CI 0.79 - 0.86).

The sociodemographic distribution of hypertension is reported in Table 4.15. There are no gender differences, with both men and women reporting little more than a quarter (26.8%) suffer from hypertension. As expected, hypertension is more prevalent in older individuals (52.6%). The racial/ethnic spread demonstrated disparities: over one-third of Blacks report hypertension whereas only about one-quarter of Whites do. Two-thirds (66.3%) of those reporting hypertension have a high school diploma or less and over one-third (36%) make \$20,000 or less per year, which also is consistent with current literature.

The distribution of risk behavior amongst those with hypertension demonstrates a trend evident in the relationship between hypertension and both physical activity and Body Mass Index (Table 4.16). Individuals who reported having hypertension also reported that they were less active and had higher Body Mass Index on average than those who reported no hypertension.

Table 4.12: Select Sociodemographic by Sociodemographic Frequencies

	Gender			Age				
	Male	Female		20 - 34	35 - 44	45 - 64	65+	
Gender								
Male				25.4%	23.6%	34.7%	16.4%	
Female				24.6%	21.7%	33.2%	20.5%	
Age			$\chi^2 = 69.38$ df3 p<.0001					
20 – 34	41.4%	58.6%						
35 – 44	42.7%	57.3%						
45 – 64	41.7%	58.3%						
65 +	35.4%	64.6%						
Race/Ethnicity								p < .0001
White	41.1%	58.9%	$\chi^2 = 10.6$ df1 p<.0011	22.8%	22.2%	34.7%	20.3%	
Black	33.0%	67.0%	$\chi^2 = 65.58$ df1 p<.0001	32.9%	23.6%	30.6%	12.9%	
Other	46.4%	53.6%	$\chi^2 = 23.77$ df1 p<.0001	39.9%	23.6%	27.0%	9.4%	
Hispanic	40.4%	59.6%	NS	47.2%	23.6%	22.8%	6.4%	
Education			$\chi^2 = 87.27$ df3 p<.0001					p < .0001
>12	41.8%	58.2%		20.2%	16.2%	27.9%	35.7%	
12	38.9%	61.0%		23.0%	22.1%	32.8%	22.1%	
13-15	37.7%	62.3%		26.6%	22.5%	34.4%	16.4%	
16+	44.5%	55.5%		26.6%	24.6%	35.9%	12.9%	
Income			$\chi^2 = 340.55$ df3 p<.0001					p < .0001
<\$20k	30.6%	69.4%		26.4%	14.9%	28.1%	30.7%	
\$20<\$50k	41.5%	58.5%		29.2%	22.2%	31.2%	17.5%	
\$50<\$75k	45.9%	54.1%		27.6%	28.4%	36.7%	7.3%	
\$75k+	49.3%	50.7%		19.4%	30.3%	43.7%	6.6%	
Marital Status			$\chi^2 = 559.36$ df2 p<.0001					p < .0001
Married	44.1%	55.9%		20.4%	25.5%	38.5%	15.6%	
Separated/Divorced/Widowed	29.0%	71.0%		8.8%	17.3%	38.1%	35.9%	
Never Married	48.2%	51.8%		61.1%	20.8%	14.1%	4.1%	

¹Hispanic vs. non-Hispanic

NOTE: Rows within factors sum to 100%

Table 4.12: Select Sociodemographic by Sociodemographic Frequencies

	Race/Ethnicity				Education			
	Hispanic ¹	White	Black	Other	<12	12	13 - 15	16+
Gender								
Male	5.2%	85.0%	7.8%	7.3%	10.1%	29.4%	24.9%	35.6%
Female	5.3%	83.5%	10.8%	5.8%	9.7%	31.6%	28.2%	30.5%
Age								
20 - 34	9.8%	77.4%	12.5%	10.1%	7.8%	28.0%	28.8%	35.4%
35 - 44	5.4%	83.4%	9.9%	6.6%	7.0%	29.8%	27.0%	36.3%
45 - 64	3.5%	86.4%	8.6%	5.0%	8.0%	29.4%	27.5%	35.2%
65 +	1.8%	90.4%	6.4%	3.1%	18.3%	35.6%	23.5%	22.7%
Race/Ethnicity								
White					8.7%	30.8%	26.8%	33.6%
Black					14.6%	33.4%	28.7%	23.4%
Other					17.7%	26.2%	25.0%	31.2%
Hispanic					31.7%	28.3%	23.4%	16.6%
								p < .0001
Education								
>12	16.8%	74.4%	14.2%	11.5%				
12	4.8%	84.2%	10.4%	5.4%				
13-15	4.6%	83.9%	10.2%	5.9%				
16+	2.7%	87.0%	6.9%	6.1%				
Income								
<\$20k	10.6%	74.3%	15.6%	10.0%	24.8%	39.5%	24.3%	11.4%
\$20<\$50k	5.2%	83.1%	10.8%	6.2%	8.6%	36.1%	29.6%	25.6%
\$50<\$75k	3.6%	88.5%	6.4%	5.1%	2.4%	23.7%	30.0%	43.9%
\$75k+	2.7%	90.2%	5.0%	4.9%	1.0%	14.2%	22.6%	62.2%
Marital Status								
Married	5.1%	88.6%	5.6%	5.9%	7.7%	29.9%	25.8%	36.7%
Separated/Divorced/Widowed	3.9%	83.7%	11.1%	5.3%	14.4%	33.6%	28.3%	23.8%
Never Married	6.3%	72.5%	19.1%	8.4%	9.2%	28.7%	28.2%	34.0%
								p < .0001

¹Hispanic vs. non-Hispanic

NOTE: Rows within factors sum to 100%

Table 4.12: Select Sociodemographic by Sociodemographic Frequencies

	Income				Marital Status		
	<\$20k	\$20<\$50k	\$50<\$75k	\$75k+	Married	Separated/Widowed/Divorced	Never
Gender							
Male	13.5%	42.5%	19.5%	24.6%	58.8%	19.6%	21.6%
Female	22.0%	43.2%	16.6%	18.2%	51.2%	32.9%	15.9%
Age							
20 - 34	18.3%	47.6%	18.7%	15.4%	47.1%	10.2%	42.8%
35 - 44	11.5%	40.2%	21.5%	26.8%	62.9%	21.5%	15.6%
45 - 64	15.0%	39.1%	19.2%	26.7%	62.1%	31.1%	6.9%
65 +	35.5%	47.5%	8.3%	8.7%	44.6%	51.9%	3.6%
Race/Ethnicity							
					p < .0001		
White	16.3%	42.5%	18.8%	22.4%	57.0%	27.3%	15.7% $\chi^2 = 664.07$ df2 p<.0001
Black	29.7%	47.7%	11.8%	10.7%	31.6%	32.0%	36.5% $\chi^2 = 746.93$ df2 p<.0001
Other	28.8%	41.3%	14.1%	15.9%	51.8%	23.4%	24.8% $\chi^2 = 51.22$ df2 p<.0001
Hispanic	36.1%	41.6%	11.8%	10.5%	55.2%	21.7%	23.1% $\chi^2 = 33.11$ df2 p<.0001
Education							
					p < .0001		
>12	51.3%	41.5%	4.8%	2.4%	42.6%	40.3%	17.1%
12	24.2%	51.8%	14.1%	9.9%	52.9%	30.1%	17.0%
13-15	16.4%	46.7%	19.6%	17.4%	52.0%	28.9%	19.1%
16+	6.2%	32.5%	23.0%	38.3%	61.0%	20.0%	19.0%
Income							
<\$20k					23.5%	50.1%	26.4%
\$20<\$50k					47.6%	31.3%	21.1%
\$50<\$75k					70.7%	15.7%	13.6%
\$75k+					82.1%	8.7%	9.2%
Marital Status							
					p < .0001		
Married	7.9%	37.4%	23.1%	31.6%			
Separated/Divorced/Widowed	33.7%	49.3%	10.3%	6.7%			
Never Married	26.5%	49.6%	13.3%	10.6%			

¹Hispanic vs. non-Hispanic

NOTE: Rows within factors sum to 100%

Table 4.13: Sociodemographic Factors by Health Behavior Frequencies

	Physical Activity			BMI			Smoking		
	None	Some	Meets	Normal	Overweight	Obese	Yes	No	
Gender	p < .0001						p < .0001		$\chi^2 = 34.85$ df 1 p<.0001
Male	14.4	38.1	47.5	32.3	45.6	22.2	25.5	74.5	
Female	15.6	41.5	43.0	49.3	29.2	21.5	22.3	77.7	
Age	$r_{sp} = -.13, p<.0001$						$r_{sp} = .08, p<.0001$		OR 0.75 (0.73, 0.77)
20 – 34	10.9	38.8	50.3	50.7	31.4	17.9	28.3	71.7	
35 – 44	11.6	40.9	47.5	41.6	36.0	22.4	29.0	71.0	
45 – 64	14.8	42.4	42.8	34.7	38.6	26.7	23.1	76.9	
65 +	26.3	37.5	36.2	41.2	39.5	19.3	11.7	88.3	
Race/Ethnicity	p < .0001								
White	13.7	39.9	46.4	43.4	36.2	20.4	23.4	76.6	
Black	23.7	42.3	34.0	29.4	37.1	33.5	23.4	76.6	NS
Other	19.4	39.7	40.9	43.8	33.5	22.7	26.9	73.2	OR 1.2 (1.07, 1.35)
Hispanic	20.0	39.0	41.1	36.6	37.3	26.1	20.6	79.4	$\chi^2 = 7.15$ df 1 p<.0075
Education	$r_{sp} = .14, p<.0001$						$r_{sp} = -.09, p<.0001$		OR 0.64 (0.62, 0.66)
>12	31.2	35.1	33.7	37.1	35.1	27.9	36.0	64.0	
12	18.1	40.3	41.7	39.3	36.6	24.2	30.0	70.0	
13-15	14.1	41.0	44.9	41.8	35.2	23.1	24.7	75.3	
16+	8.5	40.7	50.9	46.5	36.7	16.8	12.8	87.2	
Income	$r_{sp} = .14, p<.0001$						$r_{sp} = -.06, p<.0001$		OR 0.72 (0.70, 0.74)
<\$20k	25.8	37.1	37.1	40.2	32.1	27.7	32.4	67.6	
\$20<\$50k	14.3	41.4	44.4	40.5	36.4	23.1	27.0	73.1	
\$50<\$75k	9.1	42.2	48.7	40.1	38.6	21.2	21.7	78.3	
\$75k+	7.0	40.7	52.3	45.1	37.6	17.4	14.9	85.1	
Marital Status	p < .0001						p < .0001		$\chi^2 = 395.94$ df 2 p<.0001
Married	13.0	41.2	45.7	39.3	38.5	22.2	18.5	81.5	
Separated/Widowed/Divorced	21.0	39.2	39.9	42.3	35.5	22.2	28.2	71.9	
Never Married	13.0	38.8	48.2	49.3	30.3	20.4	29.4	70.6	

Note: all numbers reflect percentages

Table 4.14: Health Behavior by Health Behavior Frequencies*

	Physical Activity				BMI				Smoking	
	None	Some	Meets		Normal	Overweight	Obese		Yes	No
Physical Activity										
None					35.0%	34.6%	30.5%		26.3%	73.7%
Some					38.9%	36.7%	24.4%		22.7%	77.3%
Meets Recommendations					47.0%	36.1%	17.0%		23.4%	76.6%
BMI										
Normal	12.2%	37.1%	50.7%	$r_{sp} = -.12, p < .0001$					26.8%	73.2%
Overweight	14.1%	40.7%	45.2%						22.6%	77.4%
Obese	20.4%	44.6%	35.0%						20.1%	79.9%
Smoking										
Yes	16.8%	38.6%	44.6%	OR .95 (.91, .99)	47.4%	34.4%	18.4%	OR .82 (.79, .86)		
No	14.5%	40.5%	45.0%		40.5%	36.7%	22.9%			

*rows within behaviors sum to 100%

Table 4.15: Sociodemographic Factors by Hypertension Frequencies

	<u>Hypertension</u>
Gender	
Male	26.77%
Female	26.81%
Age	
20 – 34	9.16%
35 – 44	16.14%
45 – 64	33.70%
65 +	52.62%
Race/Ethnicity	
White	26.33%
Black	34.57%
Other	21.45%
Hispanic	17.23%
Education	
>12	36.36%
12	29.92%
13-15	26.58%
16+	21.04%
Income	
<\$20k	36.04%
\$20<\$50k	26.62%
\$50<\$75k	21.62%
\$75k+	19.05%
Marital Status	
Married	25.80%
Separated/Widowed/Divorced	37.83%
Never Married	15.08%

Table 4.16: Health Behavior by Hypertension Frequencies

	<u>Hypertension</u>	
	Yes	No
Physical Activity		
None	37.1%	62.9%
Some	27.0%	73.0%
Meets Recommendations	22.4%	77.6%
BMI		
Not Overweight	16.5%	83.5%
Overweight	28.8%	71.2%
Obese	42.7%	57.3%
Smoking		
Yes	22.0%	78.0%
No	28.3%	71.7%

The sociodemographic distribution of general health status is reported in Table 4.17. In regards to racial/ethnic division of responses, Hispanics report fair or poor health most often (22.3%), followed by Blacks (20.1%), Others (19.1%), and Whites (13.6%). The economic breakdown of responses displays a well-established trend – as income increases, so does reported general health. Expected patterns in general health are reported across health behaviors as well (Table 4.18). In regards to physical activity, almost one-third of those who meet current recommendations for activity report excellent general health (whereas only just over one-tenth of non-active individuals do). The majority of those who are not overweight (65.4%) report their general health as very good or excellent while fewer obese individuals report the same (41.4%).

The distribution of sociodemographic factors and reported days when mental health not good is displayed in Table 4.19. Men consistently have fewer days per month of poor mental health, with those who are younger, less educated, and poorer having the most. For example, only slightly more than one-third as many higher income individuals (4.4%) as lowest income individuals (12.8%) report more than half the month with mental distress. In regards to behavior and mental distress, a pattern emerges whereby there are similar frequencies across all health behaviors for the first half of the month, after which differences in behavior become more evident (Table 4.20). While all levels of physical activity have similar number of no poor mental health days/month (64%-66%), differences exist especially among those reporting more than half the

month with mental distress. These distinctions are most evident between those who engage in no activity (10.9%) or some activity (6.8%). Of the same group who report at least two weeks of mental distress, there are more obese persons (10.2%) compared to normal weight individuals (6.7%). In regards to smoking, although nonsmokers report 10% more days of no poor mental health (68.5%) compared to smokers (57%), there are over twice as many nonsmokers (12.5%) as smokers (5.8%) who report 15 or more days per month their mental health is not good.

Table 4.21 displays the relative frequencies of all three outcomes in this study. There is a consistency where those who suffer from hypertension report poor general health more often than those without hypertension. An interesting finding is that the frequencies of reported days when mental health is not good is relatively similar whether one suffers from hypertension or not. Of the individuals who report excellent health, approximately 75% also report no days/month of mental distress (Table 4.22). Alternatively, of the individuals who report poor general health, approximately 31% report poor mental health at least two weeks/month.

Table 4.17: Sociodemographic Factors by General Health Status Frequencies

	General Health				
	poor	fair	good	very good	excellent
Gender					
Male	3.4%	9.9%	28.1%	34.6%	24.0%
Female	4.1%	11.3%	28.7%	34.2%	21.7%
Age					
20 - 34	1.1%	6.3%	26.4%	38.5%	27.7%
35 - 44	1.8%	8.2%	26.0%	37.3%	26.7%
45 - 64	4.8%	11.6%	28.4%	33.0%	22.2%
65 +	8.5%	18.7%	34.1%	27.4%	11.3%
Race/Ethnicity					
White	3.6%	10.0%	27.6%	35.6%	23.1%
Black	4.7%	15.4%	33.5%	27.4%	19.1%
Other	5.4%	13.7%	31.1%	28.7%	21.1%
Hispanic	5.0%	17.3%	32.2%	25.0%	20.5%
Education					
>12	12.2%	24.6%	34.4%	18.7%	10.1%
12	4.3%	13.2%	33.0%	32.5%	17.1%
13-15	3.2%	9.9%	28.8%	36.4%	21.7%
16+	1.4%	4.9%	22.1%	39.2%	32.4%
Income					
<\$20k	10.4%	20.1%	32.7%	24.3%	12.5%
\$20<\$50k	2.9%	10.9%	31.2%	35.1%	20.0%
\$50<\$75k	1.1%	6.0%	25.1%	40.6%	27.1%
\$75k+	0.8%	3.8%	20.5%	39.7%	35.3%
Marital Status					
Married	2.7%	9.1%	27.3%	35.8%	25.2%
Separated/Widowed/Divorced	7.4%	15.3%	30.8%	30.0%	16.6%
Never Married	2.3%	8.7%	28.0%	36.3%	24.7%

Table 4.18: Health Behavior by General Health Status Frequencies

	General Health				
	poor	Fair	good	very good	excellent
Physical Activity					
None	12.1%	19.5%	32.8%	24.0%	11.6%
Some	2.8%	10.4%	30.9%	36.4%	19.6%
Meets Recommendations	1.9%	7.6%	24.1%	36.6%	29.8%
BMI					
Not Overweight	3.1%	8.3%	23.3%	35.7%	29.7%
Overweight	3.2%	9.8%	29.2%	36.4%	21.4%
Obese	6.2%	16.5%	35.9%	29.5%	11.9%
Smoking					
Yes	5.2%	13.5%	33.1%	33.5%	14.7%
No	3.4%	9.9%	27.0%	34.6%	25.1%

Table 4.19: Sociodemographic Factors by Mental Distress Frequencies

	Days of Mental Distress per Month						
	0	1	2	3	4 - 7	8 - 15	>15
Gender							
Male	72.6%	2.8%	5.2%	2.7%	6.2%	4.4%	6.1%
Female	61.1%	4.0%	7.0%	3.7%	9.0%	7.2%	8.7%
Age							
20 - 34	56.1%	4.8%	8.3%	4.4%	11.2%	8.0%	7.3%
35 - 44	60.1%	4.1%	7.5%	3.9%	8.9%	6.8%	8.5%
45 - 64	68.1%	3.2%	5.9%	2.9%	6.6%	5.3%	8.0%
65 +	82.9%	1.5%	2.5%	1.5%	3.7%	3.4%	4.6%
Race/Ethnicity							
White	65.9%	3.5%	6.4%	3.2%	7.7%	6.0%	7.2%
Black	64.3%	3.3%	5.2%	4.0%	8.0%	6.6%	8.7%
Other	65.8%	3.7%	6.6%	3.3%	7.2%	6.2%	7.2%
Hispanic	61.5%	3.7%	6.8%	4.1%	8.7%	6.3%	8.8%
Education							
>12	65.9%	1.9%	3.9%	3.2%	6.5%	7.0%	11.5%
12	67.2%	2.6%	5.4%	2.9%	7.6%	6.1%	8.3%
13-15	62.7%	3.6%	6.9%	3.2%	8.5%	7.0%	8.2%
16+	67.0%	4.7%	7.3%	3.8%	8.0%	4.9%	4.5%
Income							
<\$20k	59.3%	2.5%	5.6%	3.1%	7.8%	8.9%	12.8%
\$20<\$50k	64.7%	3.3%	6.3%	3.4%	8.5%	6.6%	7.2%
\$50<\$75k	63.7%	4.6%	7.4%	3.4%	9.3%	5.5%	6.1%
\$75k+	67.9%	4.7%	7.8%	3.8%	7.1%	4.3%	4.4%
Marital Status							
Married	69.7%	3.5%	6.1%	3.0%	7.0%	4.8%	5.9%
Separated/Widowed/Divorced	65.6%	2.8%	5.5%	3.0%	7.0%	6.4%	10.2%
Never Married	56.5%	4.5%	7.6%	4.3%	10.5%	8.7%	8.0%

Table 4.20: Health Behavior by Mental Distress Frequencies

	Days of Mental Distress per Month						
	0	1	2	3	4 - 7	8 - 15	>15
Physical Activity							
None	66.6%	2.5%	4.6%	2.4%	6.2%	6.9%	10.9%
Some	64.1%	3.8%	7.0%	3.7%	8.3%	6.2%	6.8%
Meets Recommendations	65.8%	3.6%	6.6%	3.4%	8.3%	5.8%	6.6%
BMI							
Not Overweight	64.4%	4.2%	6.8%	3.5%	8.7%	5.8%	6.7%
Overweight	69.4%	2.9%	5.9%	3.2%	7.0%	5.4%	6.3%
Obese	62.2%	3.1%	5.9%	3.1%	8.0%	7.5%	10.2%
Smoking							
Yes	57.0%	3.1%	6.4%	3.2%	8.9%	8.9%	5.8%
No	68.5%	3.6%	6.2%	3.3%	7.5%	5.2%	12.5%

Table 4.21: Hypertension by General Health and Mental Distress Frequencies

	General Health Status					Days of Mental Distress per Month						
	Excellent	Very Good	Good	Fair	Poor	0	1	2	3	4 - 7	8 - 15	> 15
Hypertension												
No	27.3%	36.8%	26.0%	7.7%	2.2%	65.0%	3.9%	6.8%	3.6%	8.1%	6.1%	6.7%
Yes	10.1%	27.7%	34.9%	19.1%	8.2%	67.9%	2.5%	4.9%	2.6%	7.1%	6.0%	9.1%

*rows within outcomes sum to 100%

Table 4.22: General Health Status by Mental Distress Frequencies

General Health Status	Days of Mental Distress per Month						
	0	1	2	3	4 - 7	8 - 15	> 15
Excellent	74.3%	4.2%	6.0%	2.6%	5.9%	3.9%	3.1%
Very Good	65.7%	4.2%	7.6%	4.0%	8.6%	5.2%	4.7%
Good	64.9%	2.8%	6.1%	3.3%	8.3%	7.0%	7.7%
Fair	55.9%	2.5%	4.6%	3.0%	9.0%	9.3%	15.8%
Poor	48.6%	0.4%	2.3%	1.4%	5.4%	10.9%	31.0%

Summary of Significant Bivariate Associations among Key Study Constructs

As presented in Table 4.23, the majority of relationships between structural, contextual, individual and outcome variables are statistically significant. Of the three outcomes in this study, general health status has the most number of significant associations, followed by mental distress and hypertension. There is mixed evidence of the relationship between one structural factor, income inequality, and one contextual factor, informal social engagement, and the outcomes. Results are consistent across all health behaviors with significant findings for every outcome.

Within associations between predictor variables, there is evidence of significant relationships between both structural factors and most contextual factors, with one exception (Table 4.24). Organizational activism or formal social engagement is not significantly related to the majority of other predictors. In regards to health behavior and broader characteristics under study, only poverty and informal social engagement show a similar pattern of results with respect to physical activity, BMI, and smoking.

Table 4.23 Summary of Significance of Bivariate Associations: Outcomes

	Hypertension	General Health	Mental Health
Income inequality	NS	S	NS
Poverty	S	S	S
Social trust	NS	S	S
Informal social engagement	S	S	NS
Organizational activism	S	S	S
Mutual aid	NS	S	S
Physical Activity	S	S	S
Body Mass Index	S	S	S
Smoking	S	S	S

Table 4.24 Summary of Significance of Bivariate Associations: Predictors

	Income Inequality	Poverty	Physical Activity	Body Mass Index	Smoking
Social trust	S	S	S	NS	S
Informal social engagement	S	S	S	S	S
Formal social participation	S	NS	S	NS	NS
Mutual aid	S	S	S	S	NS
Income inequality		S	S	NS	S
Poverty	S		S	S	S

Correlates of Hypertension

There is mixed to weak evidence of the possible role of structural factors in influencing the occurrence of hypertension, as seen in Table 4.25. Income inequality is non-significant while although poverty is statistically significant, living in poverty has a very small influence on the odds of having hypertension (OR 1.01, 95% CI 1.01-1.02). Social contextual factors fare the same, with only two dimensions of social capital significantly associated with hypertension – informal social engagement (OR 1.60, 95% CI 1.02-2.49) and formal social engagement (OR 1.86, 95% CI 1.26-2.74). An interesting note is that both of these associations are in the counterintuitive direction in that the odds of a resident reporting hypertension are higher if s/he lives in a community characterized by higher levels of informal or formal social engagement. As expected, all health behaviors are significantly associated with hypertension.

Correlates of General Health Status

Of all outcomes, findings of associations with general health status are most consistent. All structural, contextual, and behavioral variables are statistically significantly related to this variable (Table 4.26). Although numerically small, both income inequality and poverty are correlated with it at the $p < .0001$ level. While all social capital indicators are associated with general

health status, some results are in the unexpected direction. One such unanticipated finding is the negative association between health status and living in a community with more informal social engagement while there is a positive association between living in a community with more organizational activism and self-reported general health. All three health behaviors are significantly associated with this outcome at the $p < .0001$ level.

Correlates of Mental Distress

As with hypertension, social structural factors have mixed associations with self-reported mental distress (Table 4.27). Income inequality is not significantly related to the number of days per month for which mental health is reported as not good. Although poverty is statistically significantly associated at the $p < .0001$ level, directionality of the relationship is unexpected; living in an impoverished community is negatively associated with reports of mental distress. A more stable pattern exists between social context and mental distress. Findings indicate that living in a community with more social trust, Organizational activism, or mutual aid, is significantly associated with reporting fewer days per month of mental distress. Only informal social engagement is not statistically significantly related to mental distress. Consistent results are also observed with respect to the health behaviors, with all three behaviors significantly associated in the anticipated direction with this outcome.

Table 4.25: Outcome Bivariate Associations: Hypertension

	Hypertension	Level	Interpretation
<i>Social Structural Factors</i>			
Income inequality	NS	p > .25	
Poverty	S	OR 1.01 (1.01, 1.02)	Although statistically significant, living in poverty has a very small influence on the odds of reporting hypertension
<i>Social Contextual Factors</i>			
Social trust	NS	p > .41	
Informal social engagement	S	OR 1.6 (1.02, 2.49)	Almost 60% more likely to report hypertension if live in a community with high levels of informal social engagement
Organizational activism	S	OR 1.86 (1.26, 2.74)	Almost twice as likely to report hypertension if live in a community with high levels of organizational activism
Mutual aid	NS	p > .59	
<i>Health Risk Behavior</i>			
Physical Activity	S	OR 0.71 (0.69, 0.74)	Odds of reporting hypertension lower as activity increases
Body Mass Index	S	OR 1.94 (1.87, 2.02)	Odds of reporting hypertension almost 2x higher as BMI increases
Smoking	S	$\chi^2 = 95.11$ df 1 p<.0001	Non-smoking associated with reporting hypertension

Table 4.25: Outcome Bivariate Associations: Hypertension

	Hypertension	Level	Interpretation
<i>Sociodemographic Factors</i>			
Sex	NS	$p > .95$	
Income	S	OR 0.75 (0.73, 0.77)	Odds of having hypertension lower as income increases
Race/Ethnicity			
Hispanic	S	OR 0.55 (0.48, 0.64)	Odds of reporting hypertension lower if Hispanic (vs. Non-Hispanic)
Black	S	OR 1.48 (1.35, 1.62)	Odds of reporting hypertension higher for Blacks compared to Whites
Other	S	OR 0.76 (0.68, 0.86)	Odds of reporting hypertension lower for Other compared to Whites
Age	S	OR 2.29 (2.22, 2.36)	Odds of reporting hypertension higher with age
Education	S	OR 0.79 (0.76, 0.81)	Odds of reporting hypertension lower as education level increases
Marital Status	S	$\chi^2 = 745.28$ df 2 $p < .0001$	Marital status significantly associated with reporting hypertension

Table 4.26: Outcome Bivariate Associations: General Health Status

	General Health	Level	Interpretation
<i>Social Structural Factors</i>			
Income inequality	S	$r_{sp} = -.03,$ $p < .0001$	Living in a community with higher income inequality is significantly associated with reporting poorer general health
Poverty	S	$r_{sp} = -.05,$ $p < .0001$	Living in a community with higher levels of poverty is significantly associated with reporting poorer general health
<i>Social Contextual Factors</i>			
Social trust	S	$r_{sp} = .02,$ $p = .0077$	Living in a community with more social trust is significantly associated with reporting better general health
Informal social engagement	S	$r_{sp} = -.01,$ $p = .0434$	Living in a community with more ISE is significantly associated with reporting poorer general health
Organizational activism	S	$r_{sp} = .01,$ $p = .0166$	Living in a community with more OA is significantly associated with reporting better general health
Mutual aid	S	$r_{sp} = .02,$ $p = .0063$	Living in a community with more mutual aid is significantly associated with reporting better general health
<i>Health Risk Behavior</i>			
Physical Activity	S	$r_{sp} = .23,$ $p < .0001$	Physical activity is significantly associated with reporting better general health
Body Mass Index	S	$r_{sp} = -.20,$ $p < .0001$	Higher BMI is significantly associated with reporting poorer general health
Smoking	S	$p < .0001$	Nonsmoking is significantly associated with reporting better general health

Table 4.26: Outcome Bivariate Associations: General Health Status

	General Health	Level	Interpretation
<i>Sociodemographic Factors</i>			
Sex	S	$p > .0001$	Men report significantly better levels of general health
Income	S	$r_{sp} = .29,$ $p < .0001$	Higher income is significantly associated with reporting better general health
Race/Ethnicity			
Hispanic	S	$p > .0001$	Hispanics report significantly poorer general health (compared to non-Hispanics)
Black	S	$p > .0001$	Blacks report significantly poorer general health compared to Whites
Other	S	$p > .0001$	Other race/ethnicities report significantly poorer general health compared to Whites
Age	S	$r_{sp} = -.21,$ $p < .0001$	Older individuals report significantly poorer general health
Education	S	$r_{sp} = .28,$ $p < .0001$	Education level positively associated with general health
Marital Status	S	$p > .0001$	Marital status significantly associated with level of general health

Table 4.27: Outcome Bivariate Associations: Mental Distress

	Mental Health	Level	Interpretation
<i>Social Structural Factors</i>			
Income inequality	NS	$p > .83$	
Poverty	S	$r_{sp} = -.03,$ $p < .0001$	Living in a community with higher levels of poverty is significantly associated with reporting mental health not good fewer days/month
<i>Social Contextual Factors</i>			
Social trust	S	$r_{sp} = -.05,$ $p < .0001$	Living in a community with more social trust is significantly associated with reporting mental health not good fewer days/month
Informal social engagement	NS	$p > 0.58$	
Organizational activism	S	$r_{sp} = -.07,$ $p < .0001$	Living in a community with more organizational activism is significantly associated with reporting mental health not good fewer days/month
Mutual aid	S	$r_{sp} = -.04,$ $p < .0001$	Living in a community with more mutual aid is significantly associated with reporting mental health not good fewer days/month
<i>Health Risk Behavior</i>			
Physical Activity	S	$r_{sp} = -.02,$ $p = .02$	Physical activity is significantly associated with reporting fewer days/month mental health not good
Body Mass Index	S	$r_{sp} = .01,$ $p = .04$	Higher BMI is significantly associated with reporting more days/month mental health not good
Smoking	S	$p < .0001$	Smoking is significantly associated with reporting more days/month of mental health not good

Table 4.27: Outcome Bivariate Associations: Mental Distress

	Mental Health	Level	Interpretation
<i>Sociodemographic Factors</i>			
Sex	S	$p < .0001$	Significantly more women report more days/month of poor mental health than men
Income	S	$r_{sp} = -.08, p < .0001$	Lower income is significantly associated with reporting more days/month mental health not good
Race/Ethnicity			
Hispanic	S	$p = .01$	Significantly more Hispanics report more days/month of poor mental health than Non-Hispanics
Black	S	$p = .002$	Significantly more Blacks report more days/month of poor mental health than Whites
Other	NS	$p > .60$	
Age	S	$r_{sp} = -.17, p < .0001$	Younger adults report significantly more days/month mental health not good compared to older adults
Education	S	$r_{sp} = -.02, p = .0002$	Less educated report significantly more days/month mental health not good compared to more educated
Marital Status	S	$p < .0001$	Marital status significantly associated with number of days/month poor mental health reported

Associations between Social Contextual Factors and Health Behavior

Overall, the relationship between indicators of social capital and health behaviors is mixed. Table 4.28 demonstrates that there is no pattern of association across all behaviors. Moreover, some of the significant associations are in a counterintuitive direction. In regards to the associations of the dimensions of social capital within type of health behavior, the most consistent results are with physical activity. Every aspect of social capital is statistically significantly associated with physical activity, although direction of the relationships is inconsistent across dimensions. Results indicate that the greater the social trust ($r_{sp} = .05$, $p < .0001$) and informal social engagement ($r_{sp} = .02$, $p = .0034$) in the community, the more active a resident is. Less Organizational activism ($r_{sp} = -.01$, $p = .0426$) and less mutual aid ($r_{sp} = -.03$, $p < .0001$) are significantly associated with inactivity within a locale.

In contrast, both BMI and smoking have inconsistent patterns, with respect to both statistical significance and directionality of associations. Findings indicate that neither social trust nor Organizational activism is significantly associated with BMI. Informal social engagement is associated with this risk factor, albeit in the positive direction ($r_{sp} = .02$, $p = .0019$). Only mutual aid influences BMI in the anticipated manner ($r_{sp} = -.01$, $p = .048$). Aspects of social capital either have no relationship to smoking or are positively associated.

Viewing the results by dimension of social capital yields additional information. For example, the influence of social trust is different across health behaviors; an individual living in a community with greater social trust is more likely to smoke and be more active. Although informal social engagement is significantly associated with all health behaviors, directionality is inconsistent; residing in a community with more informal social engagement is associated with an inhabitant being more active, having higher BMI, and engaging in smoking. The third dimension of social capital under study, informal social participation, is the least associated with the behaviors, with a negative relationship only with physical activity. Mutual aid is also inconsistent in its influence on behaviors; individuals residing in communities with less mutual aid are both less active and have a lower BMI. These results suggest that there may be different mechanisms underlying the relationships between the dimensions of social capital and the three behaviors.

Table 4.28: Social Capital Bivariates with Health Behavior

	Physical Activity	Body Mass Index	Smoking
Social trust	$r_{sp} = .05, p < .0001$	$p > .95$	OR 1.47 (1.22, 1.77)
	The greater the social trust in the community, the more active a resident is		More likely to smoke if living in a community with high social trust
Informal social engagement	$r_{sp} = .02, p = .0034$	$r_{sp} = .02, p = .0019$	OR 4.71 (2.95, 7.54)
	The more informal social engagement in the community, the more active a resident is	The more informal social engagement in the community, the higher a resident's BMI	Almost 5 times as likely to smoke if live in a community with more informal social engagement
Organizational activism	$r_{sp} = -.01, p = .0426$	$p > .14$	$p > .36$
	The more Organizational activism in the community, the less active a resident is		
Mutual aid	$r_{sp} = -.03, p < .0001$	$r_{sp} = -.01, p = .0480$	$p > .78$
	The more mutual aid in the community, the less active a resident is	The more mutual aid in a community, the lower a resident's BMI	

Table 4.29: Social Structure Bivariates with Health Behavior

	Physical Activity	Body Mass Index	Smoking
Income inequality	$r_{sp} = -.05, p < .0001$	$p > 0.91$	OR 0.01 (0.002, 0.044)
	The greater the income inequality in a community, the less active a resident is		Less likely to smoke if live in a community with higher income inequality
Poverty	$r_{sp} = -.03, p < .0001$	$r_{sp} = .01, p = .0359$	OR 0.993 (0.989, 0.998)
	The greater the poverty in a community, the less active a resident is	The greater the poverty on the community, the higher a resident's BMI	Less likely to smoke if live in a community with more poverty

Associations between Social Structural Factors and Health Behavior

Both income inequality and poverty are significantly associated with the majority of health behaviors. An unexpected finding is that while the greater the income inequality in a community is significantly associated with less activity ($r_{sp} = -.05, p < .0001$), it is not significantly related to a resident's BMI (Table 4.29). Poverty does influence both physical activity and BMI similarly; the greater the poverty in a community, the less active ($r_{sp} = -.03, p < .0001$) and heavier ($r_{sp} = .01, p < .0359$) a resident is. As with the social contextual factors, smoking is associated with social structure, but in the unanticipated direction. One is less likely to smoke if s/he lives in a community with higher income inequality (OR 0.01, 95% CI 0.002-0.044) or more impoverished (OR 0.993, 95% CI 0.989-0.998).

Associations between Social Structural and Social Contextual Factors

The most consistent bivariate relationships are found between social contextual and social structural factors (Table 4.30). Every aspect of social capital is negatively associated with structural inequalities, other than the finding that the level of poverty in a community appears to be unrelated to formal social engagement. Of the two structural factors, income inequality seems to have the

stronger association with social capital, with correlations ranging from -.13 (organizational activism) to -.68 (social trust).

Table 4.30: Social Capital Bivariates with Social Structure

	Income Inequality	Poverty
Social Trust	$r_{sp} = -.68, p < .0001$	$r_{sp} = -.19, p < .0001$
	The greater the income inequality in a community, the less social trust	The greater the poverty in a community, the less social trust
Informal social engagement	$r_{sp} = -.46, p < .0001$	$r_{sp} = -.02, p = .0026$
	The greater the income inequality in a community, the less informal social engagement	The greater the poverty in a community, the less informal social engagement
Formal social participation	$r_{sp} = -.13, p < .0001$	$p > .54$
	The greater the income inequality in a community, the less formal social participation	
Mutual aid	$r_{sp} = -.05, p < .0001$	$r_{sp} = -.12, p < .0001$
	The greater the income inequality in a community, the less mutual aid	The greater the poverty in a community, the less mutual aid

Multivariate Analysis

For each set of hypotheses, a series of hierarchical linear models were analyzed using HLM 6 (Raudenbush & Bryk, 2004). A two-level model was used, where individual attributes and behaviors were considered level-1 factors and social contextual and social structural variables were considered level-2 factors. A random-intercept model was one where only the level-1, or individual level, intercept was treated as random. Essentially, this form of modeling is comparable to a one-way ANOVA with random effects. Only the intercept at level-1 was modeled as a function of level-2 predictors.

A basic model building approach was used for testing of the random-intercepts models. The sequence of steps began with testing an unconditional model, followed by a control and then full model. The unconditional model was one in which no predictor variables were entered at either level – it was used to test whether there were basic differences between communities in the outcome. The control model accounted for both level-1 sociodemographic and level-2 social structural controls, whereas the full model included controls and the level-2 predictor under study. The control model at level-1 included individual sociodemographic variables coded predominantly as dummy variables in order to facilitate interpretation of the intercept, as the intercept in multilevel modeling did not have the same interpretation as in OLS analysis. In these multilevel models,

the intercept did not equate to the overall mean, but rather the mean for the referent group (where all dummy variables representing sociodemographic characteristics of the individual at level 1 = 0) – here, a poor, young, white male. The selection of level-2 controls was based upon which social structural factor was included in the model. The choice of the possible level-2 control variables (median household income, percent completed high school or less, and percent unemployed) was specifically tailored to each social structural indicator to reduce the possible effects of multicollinearity; only a subset was used. For models testing the effects of income inequality, only median household income was utilized as a control in level-2. For tests of the effects of poverty, only percent completed high school or less and percent unemployed were accounted for in the models.

Results are organized by cluster of hypotheses. For each section, the overall findings are characterized, followed by detailed discussion of results.

Cluster 1: Behavioral variables only partially mediate social structure and disease.

Only very limited evidence was found to support the relationships between social structural inequalities and any of the outcomes under study (hypertension, general health status, or mental distress). Most tests of direct relationships between either income inequality or poverty and disease state were statistically

non-significant at the $p < .05$ level. Because the lack of a statistically significant direct effect of social structure on disease may indicate full mediation by health behaviors in that the relationship between income inequality or poverty and any outcome only exists through their effects on physical activity, BMI, or smoking, additional models were examined which included possible mediators. Again, there was no support found for the relationships between social structure and outcomes. As only limited evidence of a direct relationship was found between social structure and hypertension, general health status, or mental distress, it was not surprising that indirect effects would be insignificant as well. Although the results for the three outcomes were similar, some notable differences in parameter estimates were found.

Hypertension

The possible direct effects of a community's social structural environment on residents' reporting of hypertension were examined. In these models, individual odds of reporting hypertension were regressed on community income inequality and community rates of poverty separately. The unconditional model, testing between community differences in the odds of a resident reporting hypertension, was significant at $p < .0001$ level (Table 4.31). The odds of a typical resident in an average community to report hypertension is OR 0.35 (95% CI 0.32 – 0.39). The control model was significant as well ($p < .0001$), indicating

individual sociodemographic variation in individual odds of having hypertension in these communities. Once other variables were entered into the model, no statistically significant associations were found (income inequality without ($p = .715$) and with ($p = .946$) control variables; poverty without ($p = .071$) and with ($p = .464$) control variables). As there was no support for a direct relationship, no evidence was found for the possible influence of behavior mediating such relationship between social structure and hypertension. Therefore, variation in the odds of a resident reporting hypertension is not related to the level of income inequality or poverty in the community within which s/he lived. Some of the models testing these hypotheses either did not converge (poverty) and/or had non-significant between level-2 variance, Tau. Non-convergence occurred when the model was not able to be estimated, given the type of data and/or parsimony of the model. The majority of the models having a non-significant Tau (at $p < .05$) indicated that there may not be significant differences between communities, with respect to hypertension, in these data once additional variables are considered.

Table 4.31 Community Social Structural Influences on Individual Hypertension

Community Characteristic	Unconditional ¹				Conditional ¹				Hypothesis
	τ	Odds Ratio	95% confidence interval	p value	τ	Odds Ratio	95% confidence interval	p value	
No predictors model									
Intercept, γ_{00}	0.037	0.35	0.32 - 0.39	<.0001***					
Control model for income inequality									
Intercept, γ_{00}					0.011	0.33	0.20 - 0.54	<.0001***	
Model for income inequality									
Intercept, γ_{00}	0.039	0.25	0.04 - 1.70	0.15	0.012	0.31	0.06 - 1.75	0.176	1a1
income inequality, γ_{01}		2.14	0.03 - 147.55	0.715		1.12	0.04 - 28.90	0.946	
Control model for poverty									
Intercept, γ_{00}					0.003	0.05	0.03 - 0.09	<.0001***	
Model for poverty									
Intercept, γ_{00}	0.033	0.24	0.15 - 0.37	<.0001***	0.002	0.05	0.03 - 0.09	<.0001***	1a2
poverty, γ_{01}		1.01	1.00 - 1.03	0.071		1.00	0.98 - 1.01	0.464	
Control health behavior model for income inequality									
Intercept, γ_{00}					0.003	0.12	0.08 - 0.18	<.0001***	
Full health behavior model for income inequality									
Intercept, γ_{00}					0.003	0.08	0.02 - 0.30	0.001**	1b1
income inequality, γ_{01}						2.45	0.19 - 31.71	0.477	
Control health behavior model for poverty									
			Did not converge						1b2

¹Unconditional models do not consider any additional covariates in the model at level 1; conditional models account for individual sociodemographic factors, including gender, income, race/ethnicity, and age at level 1 and median household income (for income inequality) or percent unemployed and percent completed high school or less (for poverty) at level 2.

*significant at p<.05

**significant at p<.01

***significant at p<.0001

General Health Status

The potential direct effects of a community's social structural environment on residents' ratings of general health status were examined. In these models, individual odds of reporting fair or poor health were regressed on income inequality and poverty separately. The unconditional model, testing between community differences in self-reported general health status, was significant at $p < .0001$ level (table 4.32). The odds of a typical resident in an average community to report his/her health as fair or poor was OR 0.17 (95% CI 0.15 – 0.20). The control model was significant as well ($p < .032$), indicating individual sociodemographic variation in individual odds of reporting health as fair or poor in these communities. Once social structural factors were entered individually into the model (where no controls were present), some significant associations were found. Although income inequality was not significantly associated with general health status ($p = .567$), significant results were obtained ($p = .047$) in the model with only poverty entered as a predictor. However, as control variables were entered into the model, no statistically significant associations were found for either income inequality ($p = .629$) or poverty ($p = .474$). As there was little support for a direct relationship between social structure and general health status, no evidence was found for the possible influence of behavior a mediating this association. Therefore, income inequality and poverty did not significantly

explain differences in self-reported health status. Variation in the odds of a resident reporting fair or poor health was not related to the level of income inequality or poverty in the community within which s/he lived, once individual sociodemographic characteristics were taken into account. All of the models had a statistically significant Tau (at $p < .05$) indicating that there were still significant differences between communities, with respect to general health status, in these data once additional variables are considered. These results suggest that there were other variables not considered in this model which may explain the between community differences in the odds of residents reporting fair or poor health.

Mental Distress

The possible direct effects of a community's social structural environment on residents' reports of mental distress were examined. In these models, individual reports of the number of days of mental distress per month were regressed on income inequality and poverty separately. The unconditional model, testing between community differences in mental distress, was significant at $p < .0001$ level (table 4.33) indicating that the communities differed significantly in residents' days of mental distress per month. The typical resident in an average community reported 3.54 (3.29 – 3.81) days of mental distress out of the last thirty days. The control model was significant as well ($p < .002$),

Table 4.32 Community Social Structural Influences on Individual General Health Status

Community Characteristic	Unconditional ¹				Conditional ¹				Hypothesis
	τ	Odds Ratio	95% confidence interval	p value	τ	Odds Ratio	95% confidence interval	p value	
No predictors model									
intercept, γ_{00}	0.102	0.17	0.15 - 0.20	<.0001***					
Control model for income inequality									
Intercept, γ_{00}					0.053	0.37	0.15 - 0.91	0.032*	
Model for income inequality									4a1
Intercept, γ_{00}	0.104	0.07	0.004 - 1.44	0.083	0.055	0.19	0.01 - 3.78	0.262	
income inequality, γ_{01}		6.37	0.01 - 4534.17	0.567		3.82	0.01 - 1088.11	0.629	
Control model for poverty									
Intercept, γ_{00}					0.048	0.08	0.03 - 0.24	<.0001***	
Model for poverty									4a2
Intercept, γ_{00}	0.085	0.09	0.04 - 0.17	<.0001***	0.050	0.09	0.03 - 0.30	<.0001***	
poverty, γ_{01}		1.02	1.00 - 1.05	0.047*		1.01	0.98 - 1.04	0.474	
Control health behavior model for income inequality									
Intercept, γ_{00}					0.054	0.10	0.04 - 0.27	<.0001***	
Full health behavior model for income inequality									4b1
Intercept, γ_{00}					0.058	0.09	0.004 - 2.18	0.133	
income inequality, γ_{01}						1.27	0.003 - 483.65	0.935	
Control health behavior model for poverty									
Intercept, γ_{00}					0.059	0.04	0.01 - 0.15	<.0001***	
Full health behavior model for poverty									4b2
Intercept, γ_{00}					0.056	0.06	0.02 - 0.20	<.0001***	
poverty, γ_{01}						1.02	0.99 - 1.05	0.24	

¹Unconditional models do not consider any additional covariates in the model at level 1; conditional models account for individual sociodemographic factors, including gender, income, race/ethnicity, and age at level 1 and median household income (for income inequality) or percent unemployed and percent completed high school or less (for poverty) at level 2.

*significant at p<.05

**significant at p<.01

***significant at p<.0001

indicating individual sociodemographic variation in days of mental distress reported in these communities. As such, this variability was partly explained by individual sociodemographic characteristics. However, once other variables were entered into the model, no statistically significant associations were found (income inequality without ($p = .627$) and with ($p = .603$) control variables; poverty without ($p = .969$) and with ($p = .165$) control variables). As there was no support for a direct relationship, no evidence was found for the possible influence of behavior mediating such relationship between social structure and mental distress. Therefore, variation in the days a resident reported mental distress was not related to the level of income inequality or poverty in the community within which s/he lived. All of the models had a statistically significant Tau (at $p < .05$) indicating that there were still significant differences between communities, with respect to mental distress, in these data even after additional variables were considered. This situation suggests that there were other variables not included in this model which may better explain the between community differences in days of reported mental distress.

Health Behavior

The possible direct influence of a community's social structural environment on residents' health behaviors was examined. In these models,

individual health behaviors were regressed on income inequality and poverty separately. The unconditional models, testing between community differences in health behaviors, were significant at $p < .0001$ level (Table 4.34). The odds of a typical resident in an average community to report limited or no activity is OR 1.24 (95% CI 1.12 – 1.37), overweight or obese OR 1.31 (95% CI 1.21 – 1.43), or smoking OR 0.28 (95% CI 0.26 – 0.31). The control model was significant only for one health behavior, Body Mass Index ($p < .0001$), demonstrating individual sociodemographic variation in individual odds of reporting being overweight or obese in these communities. Once income inequality was entered into the models, no statistically significant associations were found for physical activity ((income inequality without ($p = .109$) and with ($p = .196$) control variables) or Body Mass Index ((income inequality without ($p = .828$) and with ($p = .361$) control variables). The results did indicate, though, that the odds of smoking were significantly influenced by the level of income inequality in one's community ($p = .032$), controlling for sociodemographic characteristics of the resident and median household income of the community.

There were some differences in results regarding tests of the poverty's influence on health behavior (Table 4.35). The control model was significant only for one health behavior, physical activity ($p = .008$), demonstrating individual sociodemographic variation in individual odds of reporting limited or no activity in these communities. Once poverty was entered into the models, no statistically significant associations were found for physical activity ((poverty without ($p =$

.141) and with ($p = .266$) control variables) or Body Mass Index ((poverty without ($p = .316$) and with ($p = .084$) control variables). The results did indicate, though, that the influence of the level of poverty in one's community on odds of smoking was not significant ($p = .055$), albeit by a relatively small degree, controlling for sociodemographic factors of the resident and the percent unemployed and percent completed high school or less within the community.

Table 4.33 Community Social Structural Influences on Individual Mental Distress

Community Characteristic	Unconditional ¹				Conditional ¹				Hypothesis
	τ	Event Rate Ratio	95% confidence interval	p value	τ	Event Rate Ratio	95% confidence interval	p value	
No predictors model									
intercept, γ_{00}	0.033	3.54	3.29 - 3.81	<.0001***					
Control model for income inequality									
Intercept, γ_{00}					0.040	3.18	1.63 - 6.18	0.002**	
Model for income inequality									7a1
intercept, γ_{00}	0.034	2.46	0.54 - 11.34	0.237	0.041	1.94	0.25 - 14.96	0.509	
income inequality, γ_{01}		2.24	0.08 - 64.95	0.627		2.66	0.06 - 123.65	0.603	
Control model for poverty									
intercept, γ_{00}					0.040	2.76	1.26 - 6.02	0.014*	
Model for poverty									7a2
intercept, γ_{00}	0.035	3.52	2.33 - 5.32	<.0001***	0.038	2.51	1.15 - 5.44	0.023*	
poverty, γ_{01}		1.00	0.99 - 1.01	0.969		0.99	0.97 - 1.01	0.165	
Control health behavior model for income inequality									
intercept, γ_{00}					0.037	1.68	0.89 - 3.20	0.108	
Full health behavior model for income inequality									7b1
intercept, γ_{00}					0.038	0.97	0.14 - 6.94	0.974	
income inequality, γ_{01}						3.01	0.07 - 121.93	0.545	
Control health behavior model for poverty									
intercept, γ_{00}					0.039	1.85	0.85 - 4.01	0.114	
Full health behavior model for poverty									7b2
intercept, γ_{00}					0.039	1.71	0.78 - 3.75	0.169	
poverty, γ_{01}						0.99	0.97 - 1.01	0.266	

¹Unconditional models do not consider any additional covariates in the model at level 1; conditional models account for individual sociodemographic factors, including gender, income, race/ethnicity, and age at level 1 and median household income (for income inequality) or percent unemployed and percent completed high school or less (for poverty) at level 2.

*significant at p<.05

**significant at p<.01

***significant at p<.0001

Table 4.34 Community Income Inequality Influences on Individual Health Behavior

Community Characteristic	Unconditional ¹				Conditional ¹				Hypothesis
	τ	Odds Ratio	95% confidence interval	p value	τ	Odds Ratio	95% confidence interval	p value	
Physical Activity²									
No predictors model									
intercept, γ_{00}	0.050	1.24	1.12 - 1.37	<.0001***					
Control model					0.042	1.62	0.75 - 3.48	0.206	
Model for income inequality									1c1
intercept, γ_{00}	0.045	0.25	0.04 - 1.82	0.164	0.040	0.37	0.03 - 4.05	0.402	
income inequality, γ_{01}		34.05	0.43 - 2690.96	0.109		18.22	0.20 - 1631.17	0.196	
Body Mass Index³									
No predictors model									
intercept, γ_{00}	0.030	1.31	1.21 - 1.43	<.0001***					
Control model					0.028	7.79	4.04 - 15.01	<.0001***	
Model for income inequality									1c2
intercept, γ_{00}	0.033	1.58	0.28 - 9.01	0.593	0.028	19.15	2.35 - 156.10	0.008**	
income inequality, γ_{01}		0.66	0.01 - 31.25	0.828		0.17	.003 - 8.69	0.361	
Smoking⁴									
No predictors model									
intercept, γ_{00}	0.038	0.28	0.26 - 0.31	<.0001***					
Control model					0.048	1.28	0.56 - 2.92	0.547	
Model for income inequality									1c3
intercept, γ_{00}	0.031	1.10	0.19 - 6.52	0.915	0.030	13.88	1.41 - 136.75	0.026*	
income inequality, γ_{01}		0.05	.001 - 2.56	0.129		0.01	0.000 - 0.64	0.032*	

¹Unconditional models do not consider any additional covariates in the model at level 1; conditional models account for individual sociodemographic factors, including gender, income, race/ethnicity, and age at level 1 and median household income at level 2

²where 0 = meets recommended levels of activity, 1 = limited/no activity

³where 0 = normal BMI, 1 = overweight/obese BMI

⁴where 0 = non-smoker, 1 = smoker

*significant at p<.05

**significant at p<.01

***significant at p<.0001

Table 4.35 Community Poverty Influences on Individual Health Behavior

Community Characteristic	Unconditional ¹				Conditional ¹				Hypothesis
	τ	Odds Ratio	95% confidence interval	p value	τ	Odds Ratio	95% confidence interval	p value	
Physical Activity²									
No predictors model									
intercept, γ_{00}	0.050	1.24	1.12 - 1.37	<.0001***					
Control model					0.023	0.33	0.15 - 0.72	0.008**	
intercept, γ_{00}									
Model for poverty									1c4
intercept, γ_{00}	0.047	0.85	0.51 - 1.43	0.530	0.023	0.29	0.13 - 0.65	0.005**	
poverty, γ_{01}		1.01	1.00 - 1.03	0.141		0.99	0.97 - 1.01	0.266	
Body Mass Index³									
No predictors model									
intercept, γ_{00}	0.030	1.31	1.21 - 1.43	<.0001***					
Control model					0.017	1.23	0.60 - 2.50	0.559	
intercept, γ_{00}									
Model for poverty									1c5
intercept, γ_{00}	0.031	1.06	0.69 - 1.64	0.779	0.015	1.01	0.49 - 2.10	0.972	
poverty, γ_{01}		1.01	0.99 - 1.02	0.316		0.99	0.97 - 1.002	0.084	
Smoking⁴									
No predictors model									
intercept, γ_{00}	0.038	0.28	0.26 - 0.31	<.0001***					
Control model					0.022	1.11	0.49 - 2.51	0.786	
intercept, γ_{00}									
Model for poverty									1c6
intercept, γ_{00}	0.038	0.34	0.21 - 0.56	<.0001***	0.019	0.86	0.37 - 1.98	0.709	
poverty, γ_{01}		0.99	0.98 - 1.01	0.404		0.98	0.96 - 1.00	0.055	

¹Unconditional models do not consider any additional covariates in the model at level 1; conditional models account for individual sociodemographic factors, including gender, income, race/ethnicity, and age at level 1 and percent unemployed and percent completed high school or less at level 2.

²where 0 = meets recommended levels of activity, 1 = limited/no activity

³where 0 = normal BMI, 1 = overweight/obese BMI

⁴where 0 = non-smoker, 1 = smoker

*significant at p<.05

**significant at p<.01

***significant at p<.0001

Cluster 2: Social context partially mediates social structure and disease.

Mixed evidence was found to support the hypothesis that that the level of social capital in the community in which one resides directly influences self-reported health (e.g., hypertension, general health, mental health). Consistent with the findings for social structure, most tests of a direct relationship between indicators of social capital and disease state were statistically non-significant at the $p < .05$ level. All four social capital dimensions (social trust, informal social engagement, organizational activism, and mutual aid) were tested separately.

Social Capital and Disease

Hypertension

The possible direct effects of a community's social contextual environment on residents' reporting of hypertension were examined. In these models, individual odds of reporting hypertension were regressed on social trust, informal social engagement, organizational activism, and mutual aid individually. As stated earlier, the unconditional model, testing between community differences in resident's odds of hypertension, was significant at $p < .0001$ level (Table 4.36).

Table 4.36 Community Social Capital Influences on Individual Hypertension

Community Characteristic	Unconditional ¹				Conditional ¹				Hypothesis
	τ	Odds Ratio	95% confidence interval	<i>p</i> value	τ	Odds Ratio	95% confidence interval	<i>p</i> value	
No predictors model									
intercept, γ_{00}	0.037	0.35	0.32 - 0.39	<.0001***					
Level 1 control model									
intercept, γ_{00}					0.015	0.16	0.14 - 0.19	<.0001***	
Model for social trust									
intercept, γ_{00}	0.039	0.35	0.32 - 0.39	<.0001***	0.016	0.16	0.14 - 0.19	<.0001***	2a1
social trust, γ_{01}		1.11	0.58 - 2.11	0.742		0.96	0.60 - 1.54	0.863	
Model for informal social engagement									
intercept, γ_{00}	0.039	0.36	0.32 - 0.39	<.0001***	0.015	0.16	0.14 - 4.24	<.0001***	2a2
informal social engagement, γ_{01}		1.33	0.32 - 5.59	0.689		1.44	0.50 - 4.24	0.498	

Table 4.36 Community Social Capital Influences on Individual Hypertension

Community Characteristic	Unconditional ¹				Conditional ¹				Hypothesis
	τ	Odds Ratio	95% confidence interval	<i>p</i> value	τ	Odds Ratio	95% confidence interval	<i>p</i> value	
Model for organizational activism									
intercept, γ_{00}	0.040	0.35	0.31 - 0.40	<.0001***	0.016	0.17	0.14 - 0.20	<.0001***	2a3
organizational activism, γ_{01}		0.98	0.32 - 3.05	0.978		0.89	0.37 - 2.16	0.795	
Model for mutual aid									
intercept, γ_{00}	0.040	0.38	0.13 - 1.13	0.079	0.016	0.18	.080 - 0.43	<.0001***	2a4
mutual aid, γ_{01}		0.99	0.80 - 1.22	0.913		0.98	0.83 - 1.16	0.833	

¹Unconditional models do not consider any additional covariates in the model at level 1; conditional models account for individual sociodemographic factors, including gender, income, race/ethnicity, and age.

*significant at $p < .05$

**significant at $p < .01$

***significant at $p < .0001$

The odds of a typical resident in an average community reporting hypertension was OR 0.35 (0.32 – 0.39). The control model was significant as well ($p < .0001$), indicating individual sociodemographic variation in individual odds of having hypertension in these communities. Once other variables were entered into the model, no statistically significant associations were found (social trust without ($p = .742$) and with ($p = .863$) control variables; informal social engagement without ($p = .689$) and with ($p = .498$) control variables; organizational activism without ($p = .978$) and with ($p = .795$) control variables; mutual aid without ($p = .913$) and with ($p = .833$) control variables). As there was no support for a direct relationship, no tests were performed on the possible influence of behavior mediating such relationship between social context and hypertension. Results suggested that variation in the odds of a resident reporting hypertension was not related to the level of social trust, informal social engagement, organizational activism, or mutual aid in the community within which s/he lived. In sum, these findings indicated that above and beyond sociodemographic factors, social capital did not explain a resident's odds of reporting hypertension.

General Health Status

The potential direct effects of a community's social contextual environment on residents' reports of general health status were examined. In these models,

individual odds of reporting fair or poor health were regressed on social trust, informal social engagement, organizational activism, and mutual aid individually. As stated earlier, the unconditional model, testing between community differences in resident's odds of reporting fair or poor health, was significant at $p < .0001$ level (Table 4.37). The odds of a typical resident in an average community to report his/her health as fair or poor was OR 0.17 (95% CI 0.15 – 0.20). The control model was significant as well ($p < .0001$), indicating individual sociodemographic variation in individual odds of reporting fair or poor health in these communities. Once other variables were entered into the model, several statistically significant associations were found. The level of social trust in the community in which one resided decreased the odds of reporting fair or poor health (OR 0.47, 95% CI 0.23 – 1.00, $p = .05$), controlling for individual sociodemographic characteristics. Additionally, living in a community with strong organizational activism also decreased the odds of reporting fair or poor health (OR 0.19, 95% CI 0.05 – 0.69, $p = .01$), controlling for individual sociodemographic characteristics. There was no evidence of a direct relationship for the other two dimensions of social capital (informal social engagement without ($p = .157$) and with ($p = .081$) control variables; mutual aid without ($p = .15$) and with ($p = .081$) control variables). As there was support for a direct relationship between social trust and organizational activism and general health status, tests were performed on the possible influence of behavior mediating such relationship between social context and general health status. In

essence, results suggested that variation in the odds of a resident reporting his/her health as fair or poor was related to the level of organizational activism, after controlling for both sociodemographic characteristics and health behavior of the individual. However, once the sociodemographic characteristics and health behavior of the individual was considered, social trust no longer significantly predicted the odds of a resident reporting his/her health as fair or poor. In sum, these findings indicated that above and beyond sociodemographic factors and behavior, only certain indicators of social capital explained a resident's odds of reporting his/her health as fair or poor.

Table 4.37 Community Social Capital Influences on Individual General Health Status

Community Characteristic	Unconditional ¹				Conditional ¹				Hypothesis
	τ	Odds Ratio	95% confidence interval	<i>p</i> value	τ	Odds Ratio	95% confidence interval	<i>p</i> value	
No predictors model									
intercept, γ_{00}	0.102	0.17	0.15 - 0.20	<.0001***					
Level 1 control model									
intercept, γ_{00}					0.057	0.18	0.15 - 0.23	<.0001***	
Model for social trust									
intercept, γ_{00}	0.096	0.17	0.15 - 0.20	<.0001***	0.048	0.19	0.15 - 0.23	<.0001***	5a1
social trust, γ_{01}		0.43	0.16 - 1.14	0.087		0.47	0.23 - 1.002	0.051*	
Model for informal social engagement									
intercept, γ_{00}	0.101	0.17	0.15 - 0.19	<.0001***	0.055	0.18	0.15 - 0.23	<.0001***	5a2
informal social engagement, γ_{01}		0.21	0.02 - 1.91	0.157		0.20	0.03 - 1.23	0.081	

Table 4.37 Community Social Capital Influences on Individual General Health Status

Community Characteristic	Unconditional ¹				Conditional ¹				Hypothesis
	τ	Odds Ratio	95% confidence interval	<i>p</i> value	τ	Odds Ratio	95% confidence interval	<i>p</i> value	
Model for organizational activism									
intercept, γ_{00}	0.098	0.19	0.16 - 0.23	<.0001***	0.440	0.21	0.17 - 0.26	<.0001***	5a3
organizational activism, γ_{01}		0.22	0.04 - 1.20	0.078		0.19	0.05 - 0.69	0.014*	
Model for mutual aid									
intercept, γ_{00}	0.096	0.55	0.11 - 2.80	0.457	0.049	0.59	0.16 - 2.23	0.422	5a4
mutual aid, γ_{01}		0.80	0.59 - 1.09	0.15		0.80	0.62 - 1.03	0.081	

¹Unconditional models do not consider any additional covariates in the model at level 1; conditional models account for individual sociodemographic factors, including gender, income, race/ethnicity, and age.

*significant at $p < .05$

**significant at $p < .01$

***significant at $p < .0001$

Mental Distress

The possible direct effects of a community's social contextual environment on residents' reporting of mental distress were examined. In these models, an individual's days per month of reported mental distress were regressed on social trust, informal social engagement, organizational activism, and mutual aid individually. As stated earlier, the unconditional model, testing between community differences in resident's days per month of reported mental distress, was significant at $p < .0001$ level (Table 4.38). The typical resident in an average community reported 3.54 (3.29 – 3.81) days per month when their mental health was not good. The control model was significant as well ($p < .0001$), indicating individual sociodemographic variation in days per month of mental distress in these communities. Once other variables were entered into the model, no statistically significant associations were found (social trust without ($p = .553$) and with ($p = .825$) control variables; informal social engagement without ($p = .864$) and with ($p = .974$) control variables; organizational activism without ($p = .217$) and with ($p = .235$) control variables; mutual aid without ($p = .255$) and with ($p = .318$) control variables). As there was no support for a direct relationship, no tests were performed on the possible influence of behavior mediating such relationship between social context and mental distress. Results suggested that variation in the days per month a resident reported mental distress was not related to the level of social trust, informal social engagement,

organizational activism, or mutual aid in the community within which s/he lived. In sum, these findings indicated that above and beyond sociodemographic factors, social capital did not explain a resident's mental distress.

Social Structure and Social Capital

There was limited evidence to support the relationship between social structural factors (income inequality and poverty) and social context (social capital indicators such as social trust, informal social engagement, organizational activism, and mutual aid). As all variables are ecological in nature and pertinent hypotheses were related to tests of association only, correlational analysis was performed. Only social trust was consistently negatively associated with both poverty and income inequality (Table 4.39). Correlations of $-.402$ ($p = .037$) and $-.549$ ($p = .003$), respectively, point to 16% to 30% of the variance in social trust explained by social structure, controlling for no other predictors. However, no support was found regarding any other social capital indicator and income inequality or poverty.

Table 4.38 Community Social Capital Influences on Mental Distress

Community Characteristic	Unconditional ¹				Conditional ¹				Hypothesis
	τ	Event Rate Ratio	95% confidence interval	<i>p</i> value	τ	Event Rate Ratio	95% confidence interval	<i>p</i> value	
No predictors model									
intercept, γ_{00}	0.033	3.54	3.29 - 3.81	<.0001***					
Level 1 control model									
intercept, γ_{00}					0.04	3.70	3.40 - 4.03	<.0001***	
Model for social trust									
intercept, γ_{00}	0.034	3.56	3.30 - 3.84	<.0001***	0.040	3.71	3.39 - 4.05	<.0001***	8a1
social trust, γ_{01}		0.85	0.50 - 1.47	0.553		0.94	0.52 - 1.68	0.825	
Model for informal social engagement									
intercept, γ_{00}	0.035	3.54	3.29 - 3.82	<.0001***	0.040	3.70	3.39 - 4.03	<.0001***	8a2
informal social engagement, γ_{01}		0.91	0.28 - 2.94	0.864		0.98	0.28 - 3.49	0.974	

Table 4.38 Community Social Capital Influences on Mental Distress

Community Characteristic	Unconditional ¹				Conditional ¹				Hypothesis
	τ	Event Rate Ratio	95% confidence interval	<i>p</i> value	τ	Event Rate Ratio	95% confidence interval	<i>p</i> value	
Model for organizational activism									
intercept, γ_{00}	0.033	3.69	3.35 - 4.07	<.0001***	0.038	3.86	3.46 - 4.31	<.0001***	8a3
organizational activism, γ_{01}		0.58	0.24 - 1.40	0.217		0.57	0.22 - 1.47	0.235	
Model for mutual aid									
intercept, γ_{00}	0.033	5.79	2.43 - 13.78	<.0001***	0.039	5.89	2.30 - 15.11	0.001**	8a4
mutual aid, γ_{01}		0.91	0.77 - 1.07	0.255		0.91	0.76 - 1.10	0.318	

¹Unconditional models do not consider any additional covariates in the model at level 1; conditional models account for individual sociodemographic factors, including gender, income, race/ethnicity, and age.

*significant at $p < .05$

**significant at $p < .01$

***significant at $p < .0001$

Table 4.39: Associations between Social Structural Factors and Social Capital Indicators¹

Indicator	Income Inequality	Poverty	p-value	Hypotheses
Social Trust	-.549		.003**	2b1
			-.402	.037*
Informal Social Engagement	-.286		.148	2b2
			-.246	.215
Organizational Activism	-.197		.324	2b3
			-.100	.619
Mutual Aid	-.113		.574	2b4
			-.167	.404

¹Analysis completed using correlational analysis as hierarchical linear modeling not appropriate.

*significant at p<.05

**significant at p<.01

Cluster 3: Behavior only partially mediates social context and disease

Tests were performed to investigate the possible direct effects of health behaviors (physical activity, Body Mass Index, and smoking) on the three outcomes under study (hypertension, general health status, mental distress), direct effects of social capital on the health behaviors, and the extent of the mediating role of these behaviors on the relationship between social context and the outcomes. The strongest evidence was found for the direct effect of behavior on all three outcomes. In addition, some findings pointed to a direct effect of social capital on all three behaviors. There was limited evidence to support the role of health behaviors as only mediating the association between social capital and hypertension, general health status, or mental distress. Significant results

were found for organizational activism's direct and indirect influence on general health status. In general, although data revealed the significant role of health behavior in shaping hypertension, general health status, or mental distress directly, findings were mixed regarding social contextual influences on behavior.

Health Behavior and Health Outcomes

The testing of the effects of behavior on hypertension, general health status, and mental distress was modeled using OLS, as these variables represented only the individual level of analysis and therefore were not appropriate for hierarchical linear modeling. As displayed in Table 4.40, results demonstrated that the odds of reporting hypertension increased if one is inactive (OR 1.11, 95% CI 1.03 – 1.19, $p = .006$) or overweight or obese (OR 2.52, 95% CI 2.34 – 2.73, $p < .0001$), but no significant association was found for smoking (OR 0.95, 95% CI 0.87 – 1.03, $p = .2063$), after accounting for individual sociodemographic characteristics. In regards to general health status, one was more likely to report fair or poor health if one was inactive (OR 1.62, 95% CI 1.48 – 1.77, $p < .0001$), overweight (OR 1.47, 95% CI 1.34 – 1.61, $p < .0001$), or smoked (OR 1.66, 95% CI 1.50 – 1.83, $p < .0001$), after individual sociodemographic characteristics were controlled. Deleterious mental health effects of engaging in risk behavior were found as well. Days of reported mental

distress significantly increased with physical inactivity (parameter estimate 0.35, se 0.079, $p < .0001$), overweight/obesity (parameter estimate 0.62, se 0.071, $p < .0001$) or smoking (parameter estimate 2.12, se 0.127, $p < .0001$). Standardized estimates showed that this negative influence was not uniform across behavior, with smoking (0.11963) comparably the most influential, and weight (0.06331) and inactivity (0.03246) following.

Health Behavior and Social Capital

Overall, very little evidence was found supporting the role of community social context in shaping individual health behavior. This finding was consistent across behavior, regardless of which social capital indicator was modeled, with and without accounting for individual sociodemographic characteristics (Table 4.41 – 4.43). The only exception was the influence of social trust on physical activity. Both unconditional and conditional models demonstrated that the odds of a resident reporting limited or no activity was significantly lower (OR 0.51, 95% CI 0.28 – 0.90, $p = .023$), if s/he lives in a community with higher levels of social trust (Table 4.41). Living in a community with higher levels of social capital did not significantly influence the odds of a resident being overweight or obese (p -values ranged from .158 to .537) or smoking (p -values ranged from .269 to .646).

Table 4.40: Influence of Risk Behavior on Self-Reported Health¹

Model	Hypertension			General Health Status			Mental Distress		
	Odds Ratio	95% confidence interval	p-value	Odds Ratio	95% confidence interval	p-value	Parameter estimate	p-value	Standardized estimate
Control variables:									
gender	0.94	0.88 - 1.01	0.095	1.01	0.92 - 1.10	0.8605	1.37 (0.11)	<.0001***	
age	2.24	2.16 - 2.33	<.0001***	1.65	1.58 - 1.73	<.0001***	-0.51 (0.053)	<.0001***	
income	0.81	0.78 - 0.84	<.0001***	0.51	0.48 - 0.54	<.0001***	-0.65 (0.055)	<.0001***	
race/ethnicity	1.20	1.13 - 1.28	<.0001***	1.26	1.17 - 1.35	<.0001***	-0.2 (0.1)	0.0466*	
Health behaviors:									
physical activity	1.11	1.03 - 1.19	0.006**	1.62	1.48 - 1.77	<.0001***	0.35 (0.079)	<.0001***	0.03246
body mass index	2.52	2.34 - 2.73	<.0001***	1.47	1.34 - 1.61	<.0001***	0.62 (0.071)	<.0001***	0.06331
smoking	0.95	0.87 - 1.03	0.2063	1.66	1.50 - 1.83	<.0001***	2.12 (0.127)	<.0001***	0.11963
	Hypothesis 3a			Hypothesis 6a			Hypothesis 9a		

¹Analysis completed using OLS and logistic regression as hierarchical linear modeling not appropriate.

*significant at p<.05

**significant at p<.01

***significant at p<.0001

Table 4.41 Community Social Capital Influence on Individual Physical Activity

Community Characteristic	Unconditional ¹				Conditional ¹				Hypothesis
	τ	Odds Ratio	95% confidence interval	<i>p</i> value	τ	Odds Ratio	95% confidence interval	<i>p</i> value	
No predictors model									
intercept, γ_{00}	0.500	1.24	1.12 - 1.37	<.0001***					
Level 1 control model									
intercept, γ_{00}					0.041	1.02	0.88 - 1.19	0.777	
Model for social trust									
intercept, γ_{00}	0.040	1.27	1.15 - 1.39	<.0001***	0.031	1.04	0.90 - 1.21	0.542	3b1
social trust, γ_{01}		0.50	0.27 - 0.96	0.037*		0.51	0.28 - 0.90	0.023*	
Model for informal social engagement									
intercept, γ_{00}	0.050	1.24	1.12 - 1.37	<.0001***	0.041	1.02	0.88 - 1.18	0.811	3b2
informal social engagement, γ_{01}		0.47	0.10 - 2.26	0.336		0.48	0.11 - 2.09	0.314	

Table 4.41 Community Social Capital Influence on Individual Physical Activity

Community Characteristic	Unconditional ¹				Conditional ¹				Hypothesis
	τ	Odds Ratio	95% confidence interval	p value	τ	Odds Ratio	95% confidence interval	p value	
Model for organizational activism									3b3
intercept, γ_{00}	0.053	1.27	1.11 - 1.46	0.002**	0.044	1.04	0.88 - 1.24	0.620	
organizational activism, γ_{01}		0.71	0.21 - 2.43	0.567		0.74	0.23 - 2.39	0.600	
Model for mutual aid									3b4
intercept, γ_{00}	0.047	0.50	0.16 - 1.58	0.226	0.038	0.37	0.13 - 1.10	0.072	
mutual aid, γ_{01}		1.19	0.96 - 1.48	0.114		1.21	0.99 - 1.49	0.064	

¹Unconditional models do not consider any additional covariates in the model at level 1; conditional models account for individual sociodemographic factors, including gender, income, race/ethnicity, and age.

*significant at p<.05

**significant at p<.01

***significant at p<.0001

Table 4.42 Community Social Capital Influence on Individual Body Mass Index

Community Characteristic	Unconditional ¹				Conditional ¹				Hypothesis
	τ	Odds Ratio	95% confidence interval	<i>p</i> value	τ	Odds Ratio	95% confidence interval	<i>p</i> value	
No predictors model									
intercept, γ_{00}	0.030	1.31	1.21 - 1.43	<.0001***					
Level 1 control model									
intercept, γ_{00}					0.033	3.79	3.26 - 4.39	<.0001***	
Model for social trust									3b5
intercept, γ_{00}	0.033	1.31	1.20 - 1.43	<.0001***	0.034	3.76	3.23 - 4.37	<.0001***	
social trust, γ_{01}		1.11	0.62 - 1.98	0.724		1.25	0.69 - 2.29	0.446	
Model for informal social engagement									3b6
intercept, γ_{00}	0.033	1.31	1.21 - 1.43	<.0001***	0.033	3.79	3.27 - 4.40	<.0001***	
informal social engagement, γ_{01}		1.09	0.29 - 4.05	0.895		1.51	0.39 - 5.86	0.537	

Table 4.42 Community Social Capital Influence on Individual Body Mass Index

Community Characteristic	Unconditional ¹				Conditional ¹				Hypothesis
	τ	Odds Ratio	95% confidence interval	<i>p</i> value	τ	Odds Ratio	95% confidence interval	<i>p</i> value	
Model for organizational activism									
intercept, γ_{00}	0.031	1.40	1.25 - 1.56	<.0001***	0.034	4.00	3.38 - 4.73	<.0001***	3b7
organizational activism, γ_{01}		0.42	0.16 - 1.15	0.088		0.47	0.16 - 1.37	0.158	
Model for mutual aid									
intercept, γ_{00}	0.029	2.60	1.002 - 6.73	0.049*	0.033	6.42	2.28 - 18.12	0.001**	3b8
mutual aid, γ_{01}		0.88	0.73 - 1.05	0.152		0.9	0.74 - 1.10	0.300	

¹Unconditional models do not consider any additional covariates in the model at level 1; conditional models account for individual sociodemographic factors, including gender, income, race/ethnicity, and age.

*significant at $p < .05$

**significant at $p < .01$

***significant at $p < .0001$

Table 4.43 Community Social Capital Influence on Individual Smoking

Community Characteristic	Unconditional ¹				Conditional ¹				Hypothesis
	τ	Odds Ratio	95% confidence interval	<i>p</i> value	τ	Odds Ratio	95% confidence interval	<i>p</i> value	
No predictors model									
intercept, γ_{00}	0.038	0.28	0.26 - 0.31	<.0001***					
Level 1 control model									
intercept, γ_{00}					0.045	1.02	0.87 - 1.20	0.801	
Model for social trust									3b9
intercept, γ_{00}	0.036	0.28	0.26 - 0.31	<.0001***	0.041	1.01	0.86 - 1.19	0.892	
social trust, γ_{01}		1.34	0.72 - 2.50	0.345		1.44	0.74 - 2.82	0.277	
Model for informal social engagement									3b10
intercept, γ_{00}	0.034	0.28	0.26 - 0.31	<.0001***	0.041	1.03	0.87 - 1.21	0.747	
informal social engagement, γ_{01}		2.23	0.56 - 8.92	0.244		2.31	0.50 - 10.58	0.269	

Table 4.43 Community Social Capital Influence on Individual Smoking

Community Characteristic	Unconditional ¹				Conditional ¹				Hypothesis
	τ	Odds Ratio	95% confidence interval	<i>p</i> value	τ	Odds Ratio	95% confidence interval	<i>p</i> value	
Model for organizational activism									
intercept, γ_{00}	0.042	0.29	0.25 - 0.33	<.0001***	0.049	1.04	0.86 - 1.26	0.672	3b11
organizational activism, γ_{01}		0.76	0.24 - 2.44	0.628		0.75	0.21 - 2.68	0.646	
Model for mutual aid									
intercept, γ_{00}	0.041	0.24	0.08 - 0.74	0.016*	0.048	0.73	0.21 - 2.49	0.596	3b12
mutual aid, γ_{01}		1.03	0.83 - 1.28	0.78		1.07	0.85 - 1.35	0.571	

¹Unconditional models do not consider any additional covariates in the model at level 1; conditional models account for individual sociodemographic factors, including gender, income, race/ethnicity, and age.

*significant at $p < .05$

**significant at $p < .01$

***significant at $p < .0001$

Social Capital, Health Behavior, and General Health Status

As evidence was found for the direct association between two correlates of social capital, organizational activism and social trust, and general health status, models were constructed that included health behaviors in order to test for mediating effects. Results demonstrated that the stronger organizational activism in the community in which one resided was statistically significantly associated with lower odds of a resident reporting his/her health as fair or poor (Table 4.44), both when controlling for individual characteristics (OR 0.19, 95% CI 0.05 – 0.69, $p = .014$) and accounting for individual health behaviors (OR 0.19, 95% CI 0.05 – 0.66, $p = .012$). This finding established that the influence of social context on health status was not completely mediated by individual factors in these data. Different results were obtained for models testing social trust. Although findings demonstrated that higher levels of social trust in the community in which one resided was statistically significantly associated with lower odds of a resident reporting his/her health as fair or poor (Table 4.45), this only occurred when controlling for individual characteristics (OR 0.47, 95% CI 0.23 – 1.00, $p = .05$), but was non significant after accounting for individual health behaviors (OR 0.54, 95% CI 0.25 – 1.18, $p = .117$). Once a resident's level of activity, weight, and smoking status were added to the model, the level of social trust in the community no longer explained the odds of reporting fair or poor health.

Table 4.44 Community Organizational Activism Influence on General Health Status, With and Without Mediators Added

Community Characteristic	Without Health Behaviors ¹				With Health Behaviors Added ¹				Hypothesis
	τ	Odds Ratio	95% confidence interval	p value	τ	Odds Ratio	95% confidence interval	p value	
Organizational activism model									6b3
intercept, γ_{00}	0.044	0.21	0.17 - 0.26	<.0001***	0.037	0.07	0.06 - 0.09	<.0001***	
physical activity, γ_{10}						1.57	1.48 - 1.67	<.0001***	
body mass index, γ_{20}						1.43	1.35 - 1.51	<.0001***	
smoking, γ_{30}						1.70	1.54 - 1.88	<.0001***	
organizational activism, γ_{01}		0.19	0.05 - 0.69	0.014*		0.19	0.05 - 0.66	0.012*	

¹Both models account for individual sociodemographic covariates including gender, income, race/ethnicity, and age.

*significant at p<.05

**significant at p<.01

***significant at p<.0001

Table 4.45 Community Social Trust Influence on General Health Status, With and Without Mediators Added

Community Characteristic	Without Health Behaviors ¹				With Health Behaviors Added ¹				Hypothesis
	τ	Odds Ratio	95% confidence interval	<i>p</i> value	τ	Odds Ratio	95% confidence interval	<i>p</i> value	
Social Trust Model									6b1
intercept, γ_{00}	0.048	0.19	0.15 - 0.23	<.0001***	0.050	0.07	0.05 - 0.08	<.0001***	
physical activity, γ_{10}						1.57	1.48 - 1.67	<.0001***	
body mass index, γ_{20}						1.43	1.35 - 1.51	<.0001***	
smoking, γ_{30}						1.70	1.54 - 1.88	<.0001***	
social trust, γ_{01}		0.47	0.23 - 1.00	0.051*		0.54	0.25 - 1.18	0.117	

¹Both models account for individual sociodemographic covariates including gender, income, race/ethnicity, and age.

*significant at $p < .05$

**significant at $p < .01$

***significant at $p < .0001$

Summary of Findings

Although the data did not support the majority of hypotheses in this study, there were several significant findings. Table 4.46 summarizes overall results. The data demonstrated some evidence of the mediating role of health behavior on the relationships between social structural and social contextual inequalities and self-reported health. Direct effects were found between dimensions of social capital, organizational activism and social trust, and general health status. The direct effects of income inequality or poverty did not explain an individual's health status once behavioral factors were considered. In essence, the level of social structural inequality in the community in which one lived did not emerge as an independent influence on a resident's self-reported health. The negative influence of income inequality or poverty in the community in which one lived appeared to negatively affect health through its erosion of social trust, which impacted a resident's health behavior, thereby worsening self-reported health, including hypertension, general health, or mental distress.

Table 4.46: Summary of Significance

Cluster	Hypothesis	Support
Behavioral variables only partially mediate social structure and disease		
	1a Social structural inequality in the community in which one resides will positively influence self-reported <i>hypertension</i> .	No
	4a Social structural inequality in the community in which one resides will negatively influence self-reported <i>general health status</i> .	Partial
	7a Social structural inequality in the community in which one resides will negatively influence self-reported <i>mental health</i> .	No
	1b The effect of social structure on self-reported <i>hypertension</i> is only partly mediated by known risk factors (BMI, physical activity, smoking).	No
	4b The effect of social structure on self-reported <i>general health status</i> is only partly mediated by known risk factors (BMI, physical activity, smoking).	No
	7b The effect of social structure on self-reported <i>mental health</i> is only partly mediated by known risk factors (BMI, physical activity, smoking).	No
	1c Greater social structural inequalities in the community in which one resides positively influences engaging in high risk behavior.	Partial
Social context partially mediates social structure and disease		

Table 4.46: Summary of Significance

Cluster	Hypothesis	Support
2a	The level of social capital in the community in which one resides influences self-reported <i>hypertension</i> .	No
5a	The level of social capital in the community in which one resides influences self-reported <i>general health status</i> .	Partial
8a	The level of social capital in the community in which one resides influences self-reported <i>mental health</i> .	No
2b	Social structural inequalities will be negatively associated with a salubrious social context.	Partial
2c	Social structural inequality in the community in which one resides will positively influence self-reported <i>hypertension</i> after controlling for community social context.	N/A*
5b	Social structural inequality in the community in which one resides will negatively influence self-reported <i>general health status</i> after controlling for community social context.	N/A*
8b	Social structural inequality in the community in which one resides will negatively influence self-reported <i>mental health</i> after controlling for community social context.	N/A*

Behavior only partially mediates social context and disease

Table 4.46: Summary of Significance

Cluster	Hypothesis	Support
3a	Engaging in risk behavior (BMI, physical activity, smoking) is positively associated with self-reported <i>hypertension</i> .	Partial
6a	Engaging in risk behavior (BMI, physical activity, smoking) is negatively associated with self-reported <i>general health status</i> .	Supported
9a	Engaging in risk behavior (BMI, physical activity, smoking) is negatively associated with self-reported <i>mental health</i> .	Partial
3b	Weaker social context in the community in which one resides positively influences engaging in high risk behavior.	Partial
3c	Weaker social context in the community in which one resides positively influences self-reported <i>hypertension</i> after controlling for individual risk behavior.	N/A*
6b	Weaker social context in the community in which one resides negatively influences self-reported <i>general health status</i> after controlling for individual risk behavior.	Partial
9b	Weaker social context in the community in which one resides negatively influences self-reported <i>mental health</i> after controlling for individual risk behavior.	N/A*

*Tests of mediating effects no longer pertinent as direct effects are non-significant.

CHAPTER 5: DISCUSSION AND IMPLICATIONS

Discussion

Summary of Findings

The purpose of this study was to investigate the associations between social structural factors (e.g., income inequality and poverty), community social context (e.g., social capital dimensions), and individual characteristics (e.g., risk behavioral factors) and self-reported correlates of disease (hypertension, general health status, and mental distress). This study examined the extent to which upstream structural and contextual factors indirectly affect disease through their influence on risk behavior and the degree to which social structure and context independently influence self-reported disease.

The majority of findings supported the role of mediating factors in social structural and social contextual influences on self-reported health. In addition, little support was observed for most of the hypotheses in this study. No direct effects of social structural inequalities on any of the outcomes were found. There were only two instances of the direct effect of social capital. As the organizational activism of the community decreased, the odds of a resident reporting fair/poor health increased. No other indicator of social capital exerted direct influence, once individual characteristics were considered. In regards to

contextual influences on health behavior, as the resident's community's level of social trust decreased, the odds of reporting not meeting recommended levels of physical activity increased. Other significant findings in this study confirmed those of previous investigations. Specifically, income inequality and poverty were negatively associated with community social trust. In addition, risk behaviors were associated with individuals increased odds of reporting hypertension, fair/poor general health, and more days of mental distress.

Once individual characteristics of the residents were included in the models, any significant direct effect of social structural inequalities on the health outcomes disappeared. There was only mixed evidence supporting the influence of social contextual factors on self-reported health. Results suggested that social structural and contextual inequalities did shape certain individual behaviors. In addition, results confirmed those of numerous previous studies to indicate that engaging in risk behavior did explain self-reported hypertension, poor/fair health status, and mental distress. In essence, it appeared that any significant negative influence of social structural inequalities on health was mediated by their effects on reducing social trust. Living in a community with lower levels of social trust was associated with engaging in limited/no physical activity. Reduced physical activity increased the odds of reporting hypertension. Inactivity was also associated with reporting fair/poor general health as well as more days per month of mental distress.

Neither income inequality nor poverty in the community in which a resident lived explained reports of hypertension or mental distress. Because no direct relationships were found, tests of hypotheses that behavioral variables only partially mediate social structure and disease were not performed for these outcomes. One exception to the dearth of evidence to support associated hypotheses was the finding that living in an impoverished community did increase one's odds of reporting fair/poor health. However, once adjustments were made for individual sociodemographic factors, poverty no longer directly explained general health status.

Findings indicated that most of the social contextual factors under study did not have any direct influence on any of the three health outcomes. No significant associations were observed between informal social engagement or mutual aid and hypertension, general health status, or mental distress. Significant negative effects of the other two correlates of social capital on general health status were noted. Lower levels of either community social trust or organizational activism, which includes organizational activism, were associated with increased odds of a resident reporting fair/poor health. However, once sociodemographic and behavioral variables were included in the model, only organizational activism retained its direct effects. Therefore, this association was the only direct relationship found between a social contextual factor and a self-reported outcome. None of the social capital indicators significantly influenced reports of hypertension or mental distress directly. In regards to support of

indirect or mediated relationships, only social trust was indirectly associated with greater odds of reporting hypertension and mental distress, through its effects on health behavior. These results confirmed prior evidence that the social environment exerts its influence primarily through more proximal individual behavior.

Additional previous findings were confirmed as well. Consistent with some previous literature, both income inequality and poverty were negatively associated with social trust, although these social structural variables were not related to any of the other indicators of social capital. In turn, community levels of social trust significantly predicted a resident's activity level, however had no influence on whether the individual was overweight or smoked. No other dimension of social capital significantly influenced individual risk. In addition, only limited direct influence of social structure on health behaviors was observed. Income inequality and poverty had no statistically significant effect on whether a resident was either inactive or overweight or obese, but did approach significance in explaining smoking. Again, similar to previous findings, most of the statistically significant relationships were observed between individual risk behavior and self-reported health. Physically active individuals and those who were of normal BMI reported less hypertension, better general health status, and less mental distress. Smoking was not associated with reporting hypertension, although it was related to general and mental health status.

In sum, findings from this study were not only inconsistent as a whole, but several were contrary to previous literature as well. In earlier studies, the direct negative influences of income inequality and poverty on general health status were found at multiple levels of aggregation (e.g., state, metropolitan area, and county). In addition, results had supported a stronger effect of social trust on health outcomes. In comparison, social structural and social contextual characteristics of the community in which one lived had relatively little direct significant influence on either engaging in risk behavior or reporting poor health in this investigation. Tests of hypothesized mediating roles of context and behavior were conducted only where direct effects were found between environment and individual factors. Solely organizational activism retained its significant direct effect on general health status, once individual characteristics (sociodemographic and behavioral) were considered. These somewhat unanticipated results point to several possible explanations for such findings.

Limitations of Study

There were a number of possible limitations of this study, which may have resulted in few significant findings. Methodological issues included: use of secondary data sources and sampling, variable selection and measurement, and design issues. The use of secondary data and the linking of data sets constrained the use of the data and restricted the power with which conclusions

could be drawn regarding the hypotheses. Variable selection and measurement were also negatively influenced by the data in that the scale of measurement of the constructs under study and prior construction of indices used served to introduce bias and reduce reliability and validity. Finally, the most limiting factor of the study was the design type. Cross-sectional studies such as this one cannot infer causality, but rather only suggest associations, as exposure and disease were measured at the same time.

Data Sources and Sampling

Both the data sources selected and sampling procedure used may have limited the possibility of finding support for the hypotheses in this study. Weaknesses of this study related to the linked nature of the data and the small sample size at level-2. Because this was a study using secondary data, the researcher was limited to using only those communities where structural and contextual data could be linked by FIPS codes to individual data obtained from the BRFSS. The three data sources employed may have resulted in somewhat different comparable sociodemographic characteristics of each community, thereby resulting in different, less than valid or reliable comparisons. Another related difficulty was the linking of data, which may not be specific to this study, but rather a growing issue in multilevel investigations. Sampling bias was introduced through the use of the Social Capital Community Benchmark Survey,

as this survey was not a probability sample, but rather was a convenience sample obtained at an annual meeting of community foundations. In addition, several of the communities did not have FIPS codes assigned, as they were geographically diverse. The lack of available FIPS codes for all communities may have biased the findings. In regards to sample size, only 27 out of a possible 41 communities were eligible for inclusion, thus reducing both possible variance and power. Current suggestions in the literature indicated that having a sample size of 25 – 30 at level-2 is the lower limit to obtain confidence in results (Kreft & de Leeuw, 1998). Conclusions drawn from the subsample employed in the study might have been quite different than if the whole dataset were utilized - the inclusion of more communities may yield different results.

Variable Selection and Measurement

Additionally, the selection and measurement of the variables may have constrained the capacity of this study to support the hypotheses. Variables were selected based upon theoretical considerations and informed by previous literature. However, their use was restricted by their measurement, which was not defined by the researcher. Each dataset used introduced limitations to this study. In regards to BRFSS data, there may have been threats to the validity of multiple variables selected. For example, there was some suggestion that the use of BMI as indicator of obesity may have limited validity if the muscle mass if

the individual is not considered (National Heart, Lung, & Blood Institute, 2005). Also, several of the original sociodemographic variables were not in continuous form, but rather already in ordinal-level categories (e.g., income), which limited their utility. Additionally, the outcomes (e.g., CVD or hypertension) were assessed with only single item measures and although had demonstrated good reliability and validity, self-report measures still might have introduced bias from sources including recall, resulting in underestimation of poor health. In addition, as reliability and validity of the scores measuring the constructs are sample specific, conclusions drawn from this study must consider the possibility that the variables measured did not represent the same level of reliability and validity that have been shown in previous studies utilizing the same data.

Measures of social capital were selected based upon availability. The selected indices were created by the authors of the Social Capital Community Benchmark Survey and reflected mean social trust, informal social engagement, organizational activism, and mutual aid. It might have been more suitable to use individual items or a subset of questions. Unfortunately, there were inadequate data on bridging social capital, the correlate of reciprocity, and no index available assessing global social capital within a community. Currently, there have been no studies in public health literature investigating the influence of multiple dimensions of social capital on specific health outcomes. Future studies should include selection of certain items and create additional groupings/indices. This study was just an initial step in examining whether social capital dimensions

previously identified in the literature had direct or indirect effects on self-rated health outcomes.

The lack of support in this study for a significant association between income inequality and general health status was in contrast to previous findings in the social epidemiology literature. There are several reasons for this discrepancy – all methodological in nature. The small sample at level 2 and the restricted range of gini coefficients (0.4 – 0.49) may have resulted in no relationship being detected. In addition, communities themselves were highly geographically heterogeneous within the sample – comparisons were made between individual counties, cluster of contiguous counties, and lightly populated states. There may have been more variance within a community than between them. Due to the design and the need to link secondary data sets, the sample *was intended* to represent the same population, but the data sources did reflect different levels of aggregation.

The social structural variables, income inequality and poverty, may have been constrained not by measurement alone, but also by their restricted opportunity for variance. For example, due to a narrow range of Gini values, the data selected may not demonstrate the true direct effect of income inequality on both health behaviors and health outcomes. In addition, the use of too broad of an indicator with this type of data may have limited validity and usefulness (e.g., using the community-level proportion of those living at or below 200% FPL may have been too broad an indicator and actually washed out possible effects;

moreover, several of the communities included in the dataset were intra-geographically heterogeneous). Therefore, the explanatory value of poverty in elucidating disparities in chronic disease and self-reported health status might have been better assessed by a more sensitive indicator. The lack of evidence regarding social structural inequalities influence on health might have been due to the lack of important characteristics included in the models, such as residential segregation and/or political environment. In addition, Macintyre and Ellaway (2003) suggest that as aspects of place shape individual characteristics, and visa versa, (e.g., historical shifts in industry creates context-specific opportunities for individual occupation and hence, income – and supply of trained individuals impacts demand/local labor market), measuring structural effects while controlling for individual level factors may result in a “partialling” fallacy whereby overcontrol of characteristics of the individual may result in insignificant findings where a relationship may exist; variance is concealed by correlates (Macintyre & Ellaway, 2003).

Lastly, a limitation of this study that had both measurement and design implications pertained to stability and fluidity of the composition of each community. The role of population density and transience might have biased the assessment of the “true” value of community constructs (e.g., individuals residing in communities in 1999 might have been different from individuals residing in those communities in 2001). For example, gross migration rates (including into and out of the state) from 1995 – 2000 ranged from 111.6/1000 in Michigan to

303.3/1000 in Colorado (Census 2000 Special Report, 2003). Generally, the Southern region witnessed more overall migration, with the Northeast experiencing the least. This limitation might have influenced both social structure and social context, thereby influencing exposure (both time and type) to the detrimental aspects of the social environment under study.

Design Issues

There were several limitations in the design of this study. Concerns included the role of time and exposure in addition to the absence of moderators. The cross-sectional nature of the data did not permit the design to examine temporal effects on the outcomes; length of exposure to the detrimental influence of social structural and social contextual inequalities could not be considered. The result was that it could not be demonstrated in this study that the level of poverty (or dose, as it were) influencing the outcomes was cumulative in nature or an instantaneous effect. Only a longitudinal design would have permitted a more reliable assessment of exposure.

Because there was no one nationally available dataset that includes information on multiple levels of the environment, data had to be linked in order for this investigation to be conducted. This issue restricted both the type of design as well as the type of inferences that can be made. Due to the limitations of linking data, the social trust, for instance, that was being assessed in the

community by the SCCBS may not have represented the same group of individuals represented in the BRFSS. Therefore, conclusions drawn regarding the influence of social trust in the community in which one resided on the odds of an individual rating his/her general health as poor must be considered in light of this limitation.

Threats to external validity of findings include those related to the lack of randomization with which the communities in the SCCBS were selected. Selection and setting bias may be operating (Cook & Campbell, 1979) – whereby unmeasured attributes of those residents, or their communities, who responded to the 2000 Social Capital survey differ from individuals (and areas) responding to the 2001 BRFSS questionnaires. These characteristics may have impacted the generalizability of findings, alternatively known as population, ecological, and temporal validity (Onwuegbuzie, 2003).

Specifically, individuals residing in communities in 1999 might have been different than individuals residing in those communities in 2001. In addition, the transience of residents might have influenced the social structure and social context of the community, as discussed earlier, as well as the physical characteristics (e.g., land use, zoning, development or dilapidation) of the areas. Another limitation was that the design did not examine the possible cross-level interactions among predictors or the role of moderators. For example, the moderating influence of individual sociodemographic characteristics (i.e., gender) on the influence of poverty on behavior or mental distress was not included.

Eliminating this path may have led to erroneous results. The lack of consistent findings might not be due to no relationship existing, but rather to relationships not measured in the models. Perhaps poverty's negative influence on self-reported mental distress is significant only for young men of color or perhaps it shapes activity levels only for older women. This design did not allow for these considerations. Finally, the design may have reflected a fully mediated relationship by a variable not included in the model.

Contributions of this Study and Implications for Public Health

This study contributed to public health by adding to the knowledge base in three areas where major gaps existed in knowledge related to the role of the social environment in health. The first contribution was that it added to the current empirical data regarding studies on the influence of social structure on specific outcomes. Most of what we know has come from studies on morbidity/mortality rates, life expectancy, and general health. Another advantage was that this study investigated hypertension, general health status, and mental distress outcomes using data that was expressly collected to study the effects of social capital. Finally, this study provided additional knowledge on the structural and contextual influences in which, specifically, risk behavior occurred.

Given the theoretical rationale and previous empirical studies, a greater number of significant findings supporting the hypotheses were anticipated. Although findings from this study were more limited and failed to conclusively demonstrate the extent to which context affects behavioral and self-rated health outcomes, the possibility that behavior only partially mediates the relationship between context and disease remains. This investigation pointed to the need for further examination of the effects of social context on the initiation and maintenance of health behaviors in order to broaden our understanding as well as to incorporate relevant findings into public health policy and practice. Moreover, this investigation added to the current literature by demonstrating that, despite methodological limitations, there is empirical evidence to support the influence of broader factors on health disparities. For example, poor social trust in the community in which one resided was associated with the resident participating in limited, if any, physical activity. If residents of a community do not trust their neighbors or environment, they may be less willing to spend time outside engaging in activity, such as walking. This study's findings support the continued study of macro and meso determinants of the social environment and their influence on the public health burden of chronic disease. By doing so, this study extended the understanding of the multilevel nature of health disparities and the need for multilevel interventions to reduce them (e.g., state-level policies targeting improved funding for well-lit sidewalks, community advocacy to obtain funds and implement changes to promote community cohesion and/or establish

new norms related to physical activity, individual-level behavioral change strategies).

Future implications of this study for public health included contributions to both research and practice. In a more general sense, limited evidence demonstrated that interventions focusing on disparities in multiple health outcomes should simultaneously address social and behavioral factors to inform service delivery and health policy. Several practical implications derived from this study include those related to design and data.

One suggestion that has received attention more recently is the need for quasi or experimental studies to relieve the reliance on observational investigations (Berkman, 2004). Randomized community trials that permit causal inferences to be made compel the scientist to clearly and precisely identify exposure (Oakes, 2004). For example, testing contextual influence on health through evaluation of housing policies, to which families have been randomly assigned to programs (Kaufman et al., 2003). Other possibilities include community intervention trials aimed at health promotion (e.g., targeted zoning of affordable fruit/vegetable grocery stores). In order to implement many of these studies, improved data is needed.

In regards to its influence on future studies, evidence from this work may promote the needed restructuring of large national surveillance systems to include contextual data. For example, assessment of physical and social characteristics of local environments through “ecometrics” incorporating

qualitative methods of systematic social observation (Raudenbush & Sampson, 1999; Raudenbush, 2003) would enrich available data and add to definitional clarity of constructs most commonly used in public health. Use of qualitative data, such as that collected via focus groups and open-ended interviews regarding notions of community, social trust, racial relations, and poverty, would augment currently available survey data. In addition to these issues, implications include the need to examine certain types of outcome data in the study of neighborhoods and health – such as measures of variance as well as knowledge suggested by measures of association (Merlo et al., 2005a,b). Also, the availability of longitudinal data that includes important biomarkers (e.g., cortisol, fibrinogen levels, blood pressure, norepinephrine and epinephrine levels) is imperative. By broadening influential public health surveillance systems to include these types of data, the knowledge base from which interventions are developed and conducted for diverse populations regarding common behavioral risk factors for chronic disease (e.g., smoking, physical activity, dietary practices, substance use) is expanded.

Specifically in regards to hypertension, this study provided a significant contribution to understanding the relationships between social structural, contextual, and behavioral aspects of self-reported hypertension. Results from this study provided further evidence that if the social context within which behavior occurs is not considered, interventions targeting behavior change as a prevention strategy will have limited effectiveness. Educating individuals

regarding the benefits of regular exercise may not be as valuable in changing behavior if the individual lives in an unsafe community, with no clean areas in which to walk, and in addition, without community support to shift current practices to new, more healthy norms of behavior.

In addition, this work may inform an expansion of social and structural changes. For example, to reduce the disparate burden of chronic disease, intervention targets might include: instituting regulatory changes in political and economic policy which currently shape market influences which produce and perpetuate social inequalities (Kaplan & Lynch, 1999; Terris, 1999); strengthening social capital within communities (Kawachi, 1999) or perhaps directing prevention efforts towards developing community capacity (Elliott et al., 1998); developing models that are aimed at shaping local public agendas to include community-level CVD prevention (Finnegan, Viswanath, & Hertog, 1999; Schmid, Pratt, & Howze, 1995). In light of some of the results of this study (e.g., income inequality and poverty's negative association with social trust), structural changes based on intervening at the policy-level include institution of a living wage in lieu of the inadequate "minimum wage" as currently legislated. Further studies are needed to examine if reducing the experience of poverty (both individually and community-wide) and/or income inequality would result in a commensurate reduction in isolation and disconnection. In a similar vein, improved affordable housing and medical care may reduce the disparate health burden of chronic disease currently plaguing the less fortunate in our country.

Moreover, this study added to the literature pertaining to self-reported general health and mental distress. It was unique in that it was the only investigation that examined the influence of broader, more fundamental social determinants on general health status *and* mental distress in a multilevel design, while accounting for individual attributes and behaviors. Previous research had pointed to the need for studies examining multiple dimensions of Health Related Quality of Life indicators in the pursuit of Healthy People 2010 goals of improving quality of life and reducing health disparities (Zack, Moriarty, Stroup, Ford, & Mokdad, 2004). By providing additional data assessing the burden and indicators of mental distress in a geographically diverse sample in addition to expanding possible intervention targets to improve self-reported health, this study added to the advancement of knowledge in the field of population health.

This study sought to contribute to filling some of the gaps present in the social epidemiology literature. This work empirically examined the role of social structure on specific health outcomes. Another innovation was the use of data that is expressly collected to study the effects of social capital. This work expanded the knowledge base of structural and contextual influences in which risk behavior occurs.

Public health implications of these contributions include practice, policy, and theoretical benefits. In regards to public health practice, findings from this study indicated a need to improve service delivery (e.g., by contextualizing health education programs). There were many possible policy implications of this

investigation. Firstly, national surveys might include assessment of social environmental factors as part of standard surveillance procedures. This improved surveillance might include both the linking capacity of publicly available datasets as well as broadening the inclusion of contextual factors in behavioral surveys.

By finding some supporting evidence of the negative influence of social structural and social contextual inequalities on health behavior and self-rated health despite methodological limitations, this study may inform not just assessment of public health, but also intervention strategies by adding to the growing data on the need to target wider, more fundamental, levels of the social environment, such as local, state, and federal law. Recognition that public health policy begins with the economic and is driven by the political is a critical step in order to then envision changes in political and economic policy which shape market influences which produce and perpetuate social inequalities in health (Kaplan & Lynch, 1999; Terris, 1999). At the national level, possible policy interventions might include: instituting a living wage in order to both attenuate absolute deprivation as well as narrow the range of income inequality; improving education funding to prevent early drop-out among at-risk youth; restructuring public housing to reduce residential segregation, promote safety and encourage a sense of community; nationalizing health care to provide for a more equitable distribution of benefits; access to opportunities to participate in the democratic process through legislative and other governmental initiatives.

Finally, this study contributed to the need in present public health literature to explicitly use theory to inform investigations. This study extended present empirical testing and applied use of a newer theoretical orientation, the ecosocial perspective. The work also demonstrated the benefits of combining complementary perspectives (ecosocial and political economy) to inform empirical investigations in social epidemiology. The limitations of each were reduced through employment of both.

In regards to the application and empirical testing of these theoretical positions, there were several strengths and weaknesses evident in both perspectives. Specifically in regards to political economy, the strength of its application in this study is rooted in its concentration on material conditions and power relations in addition to the notion of nonspecific mortality – getting rid of one disease is ineffective in dealing with public health, because health effects of social inequalities are not manifested in a specific disease per se, but rather are reflected in many diseases (sick individuals because a sick society). The use of self-rated health is aligned with this premise of general susceptibility. In addition, it is a formalized and coherent theory. It is integrated and yet relatively parsimonious. However, the application of political economy necessitated an ecological design, due to its explicit focus on broader aspects of the social and economic environment. Whereas it does explain reality in one sense, it fails to account for the influence of individual differences and within group variation. This inherent weakness is more evident when studying the multilevel nature of

the social determinants of health. Therefore, it was complemented by the use of the ecosocial framework.

Overall, the ecosocial approach was a better framework for this study. The social production of disease viewpoint does not include a place in its theoretical framework for neither individual agency nor intraindividual (i.e., biological or psychological) influences.

The ecosocial perspective better explained the nested nature of the phenomena under study by combining the social production of disease with biology and ecology in a dynamic process. Its focus includes the physiological pathogenic responses to social structural conditions (Krieger, 1994, 2001). Through its application, however, several limitations were found. Several tenets were difficult to operationalize and therefore unable to be tested empirically (e.g., pathways of embodiment and the cumulative interplay between exposure, susceptibility, and resistance). This weakness was not necessarily the result of inadequate theory development. It might better reflect the state of (inadequate) surveillance in public health in that there is a complex data requirement to test this framework. Utilizing this perspective necessitates improved surveillance and availability of multilevel and/or linkable datasets, in order to include aspects from the structural world to the biological system. With improved data, the relevance of the ecosocial framework for public health will surely expand.

Recommendations for Future Research

There are multiple recommendations from this study for future research, including theoretical, conceptual and methodological areas. At present, research in social epidemiology does not adequately focus on expansion of theory development in the field. The development *and* use of cohesive, predictable, parsimonious theoretical frameworks would assist in advancing this area of scientific inquiry. Moreover, studies are needed which explicitly examine the *relative* theoretical contribution of differential explanations for inequalities (e.g., material conditions versus psychosocial factors versus genetic or biological risk). Some pertinent rationales may be mutually exclusive whereas some may provide a complementary framework from which to conceptualize future studies. At this time, it is not known.

Further, potential directions of study include topics that have both methodological and conceptual implications. Studies are needed to improve assessment of the broader social determinants of health. To do so, changes need to be made regarding not just what kind of data that is collected, but also how it is obtained. As biomedical bias shapes research agendas – in addition to the questions posed and studies that are funded, national surveillance systems need to be restructured (through lobbying and engaging of policymakers) to accommodate qualitative data on context, such as the aforementioned

systematic social observation. Collection of complementary forms of data would permit improved investigations utilizing secondary data.

In addition to newer forms of data, an expanded use of design types is required. For example, there is a need for longitudinal studies using multilevel data, which may capture the lag effects of social structural and social contextual inequalities on health as these may not be immediate. Considerations include issues related to lifespan and length of exposure at structural and contextual levels for child, adolescent, and adult outcomes. There may be critical periods and transitions in development across the lifespan which may buffer or exacerbate the negative influence of inequalities. Conceptual and methodological advances such as studies utilizing a longitudinal design combining developmental theory and an ecosocial approach may generate important hypotheses related to the role of the life-cycle.

Longitudinal studies would also allow pertinent questions taking account of the level of variable most appropriate (state, community, neighborhood, block) for what type of timing, for example, temporal (e.g., cohort) effects versus role of (e.g., individual) development. Probability sampling in these studies would also permit causality to be examined and perhaps established. These studies would also utilize improved measurement and availability of characteristics of the environment (such as integrating local and national *qualitative* data on social context with enhanced quantitative assessment), perhaps using data easily linked (by common geographical identifier) to GIS or other federal, state, and

local data. Moreover, there is a need to clarify and consistently refine the operationalization of the constructs we examine in the social epidemiology literature.

In regards to methodological innovation and to advance the field, cross-level analyses would be a critical component of a future study, as this would enable contextual effects to be isolated from compositional effects. It would also permit a more refined examination of the subtleties of level-1 and level-2 interactions (e.g., testing whether individuals with a low SES living in a community characterized by high income inequality and low social capital reported mental distress more than individuals with a low SES living in a community characterized by low income inequality and high social capital or whether living in a community characterized by high poverty and disparate levels of social capital differently affected whether one engaged in risk behavior and reported hypertension). This analysis process may also tease apart the differential effects of types of social structural inequalities and correlates of social capital in explaining risk behavior and self-reported health for disparate populations (e.g., racial/ethnic groups, men vs. women, etc.).

In regards specifically to social contextual variables, separate analyses might be conducted for both composite measures as well as a global indicator of social capital. This method has been suggested, as it may provide both summary information about the relationships between the variables as well as elucidate the possible distinct effects of individual items (Putnam, 2004).

Additional considerations regarding future studies of social capital include investigations of the comparative contribution of each dimension in explaining disparities in specific health outcomes in addition to general susceptibility of subpopulations; a follow-up study to the Social Capital Community Benchmark Study; examinations of the role of density and transience of the population on the social capital of a community and its possible health effects

Conclusion

Two overarching themes emerge from this research. One theme that surfaces relates to the need to include both physical and mental aspects of health when studying chronic disease. Isolating the two hemispheres of experience serves to limit our understanding of pathology and the role of self-rated health in chronic disease.

Further, harkening back to Rose (1992) and Wilkinson (1996), although the stated focus in public health research is on population health outcomes, there is still a dearth of evidence on the ways in which population-based multilevel studies of health disparities in chronic disease can not just acknowledge, but rather impact broader social environmental influences. Understanding precedes advocacy; it is critical to make this form of research germane through increasing the role social epidemiological research plays in development of health policy.

Ultimately the value of public health research is in its relevance and ability to improve the public's health. The application of knowledge is a critical component of the research process whereby findings should translate into effective policy initiatives. Knowledge of the issues is insufficient. Heymann and Fischer (2003) ask the question "Will any of this research make a difference in public policy and practice?" Scientific study for the sake of research does little to improve public health without generating possible policy propositions; societal problems necessitate societal solutions (Heymann, 2000).

In regards to the first matter, population-based strategies may be limited if they rely on high-risk population *behavioral* change, to the exclusion of the effects of structural and contextual inequalities. Despite mounting evidence that this approach has been limited in reducing the overall burden of disease, present public health interventions continue to focus on individual-level risk factors. Although the lack of support for structural and contextual influences may be interpreted as evidence for compositional and/or individual risk factor explanations for health disparities, I conceptualize a different interpretation of the significant findings (or lack thereof) of this study. I must be conceded that it is possible that there is no direct relationship between fundamental or broader factors of the social environment and specific health outcomes. However, I interpret the limited results of this study as not indicative of support for the "composition" argument or perspective (focus on individuals rather than contextual), but rather point to the need for improved measurement and data

issues. The lack of evidence from this study to support the contextual influence of place points to several conditions of this investigation that may have shaped conclusions found – the restricted range of variables (e.g., income inequality), sample size (e.g., only 27 communities with available data), inability to incorporate temporal effect (e.g., role of transience within communities, length of exposure to environment), and additional issues related to the use of cross-sectional secondary data (e.g., incompatibility of communities between linked datasets and external validity compromises). Despite these concerns, this study has added limited support to the growing evidence base that there are macro-level structural and contextual influences on population health that cannot be reduced to individual or compositional effects. Therefore, public health goals, such as Healthy People 2010 twin goals of increasing quality of life while decreasing health disparities, will fail to be met without due consideration to fundamental factors which serve to perpetuate and maintain disparities in health.

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Appendix A1: Sample Distribution Comparison

SPONSOR: C.F. of Greater Birmingham (AL)

STATE/Counties: Alabama/Jefferson, Shelby

	BRFSS	SCCBS	Census
Population	496	500	805340
Sex			
Male	36.7%	46.9%	47.4%
Female	63.3%	53.1%	52.6%
Race			
White	65.4%	65.4%	64.4%
Black	31.7%	30.2%	34.0%
Hispanic	1.0%	3.1%	1.7%
Other	2.8%	4.4%	2.5%
Education			
0 – 12 (no diploma)	10.9%	9.2%	18.0%
12	30.3%	28.3%	27.0%
13-15	29.2%	39.6%	28.2%
≥ 16	29.5%	22.9%	26.8%
Age			
20 – 34	26.4%	28.1%	29.1%
35 – 44	20.0%	22.3%	22.2%
45 – 64	33.6%	31.0%	31.1%
65 +	20.1%	18.6%	17.6%
Income			
<\$20,000	26.4%	15.0%	24.5%
\$20,000 – \$50,000	41.5%	44.4%	35.7%
\$50,000 - \$75,000	15.2%	21.0%	18.2%
>\$75,000	16.8%	19.6%	21.4%
Marital Status			
Married	50.9%	57.8%	52.9%
Separated/Widowed/Divorced	31.0%	22.3%	20.8%
Never Married	18.1%	19.9%	26.2%

Appendix A2: Sample Distribution Comparison

SPONSOR: Arizona Community Foundation

STATE/County: Arizona/Maricopa

	BRFSS	SCCBS	Census
Population	856	501	3072149
Sex			
Male	42.5%	49.5%	50.0%
Female	57.5%	50.5%	50.0%
Race			
White	82.4%	77.3%	79.8%
Black	4.1%	4.4%	4.3%
Hispanic	13.8%	20.2%	24.8%
Other	13.6%	18.4%	19.0%
Education			
0 – 12 (no diploma)	8.9%	19.2%	17.5%
12	25.5%	20.2%	23.1%
13-15	33.3%	31.3%	33.6%
≥ 16	32.3%	29.3%	25.9%
Age			
20 – 34	26.8%	30.8%	33.1%
35 – 44	21.7%	23.5%	22.1%
45 – 64	32.1%	29.2%	28.2%
65 +	19.5%	16.5%	16.7%
Income			
<\$20,000	13.2%	17.1%	24.5%
\$20,000 – \$50,000	45.8%	44.1%	35.7%
\$50,000 - \$75,000	17.5%	17.3%	18.4%
>\$75,000	23.5%	21.5%	21.4%
Marital Status			
Married	54.4%	60.7%	54.9%
Separated/Widowed/Divorced	27.7%	15.3%	26.8%
Never Married	17.9%	24.0%	18.4%

Appendix A3: Sample Distribution Comparison

SPONSOR: California C.F.

STATE/County: California/Los Angeles

	BRFSS	SCCBS	Census
Population	1002	515	9519338
Sex			
Male	43.1%	48.4%	49.4%
Female	56.9%	51.6%	50.6%
Race			
White	75.8%	50.4%	52.8%
Black	11.4%	10.2%	10.5%
Hispanic	34.4%	39.7%	44.6%
Other	12.8%	39.3%	42.0%
Education			
0 – 12 (no diploma)	17.0%	27.5%	30.0%
12	21.4%	14.2%	18.8%
13-15	26.5%	27.9%	26.2%
≥ 16	35.2%	30.4%	24.9%
Age			
20 – 34	31.7%	32.6%	34.7%
35 – 44	22.4%	25.0%	23.1%
45 – 64	32.0%	28.0%	28.1%
65 +	13.9%	14.4%	14.1%
Income			
<\$20,000	27.7%	23.2%	23.3%
\$20,000 – \$50,000	31.5%	35.5%	33.6%
\$50,000 - \$75,000	16.5%	15.8%	17.8%
>\$75,000	24.4%	25.6%	25.3%
Marital Status			
Married	46.0%	49.1%	48.8%
Separated/Widowed/Divorced	28.2%	20.2%	17.1%
Never Married	25.8%	30.7%	34.1%

Appendix A4: Sample Distribution Comparison

SPONSOR: The San Diego Foundation

STATE/County: California/San Diego

	BRFSS	SCCBS	Census
Population	346	504	2813833
Sex			
Male	41.3%	50.1%	50.3%
Female	57.7%	49.9%	49.7%
Race			
White	87.8%	65.9%	70.3%
Black	4.1%	6.3%	6.6%
Hispanic	22.3%	18.8%	26.7%
Other	8.1%	27.8%	28.1%
Education			
0 – 12 (no diploma)	9.3%	18.5%	17.4%
12	24.4%	17.7%	19.9%
13-15	28.1%	30.5%	33.2%
≥ 16	38.3%	33.4%	29.6%
Age			
20 – 34	22.7%	29.8%	33.7%
35 – 44	22.6%	26.1%	22.8%
45 – 64	30.1%	29.2%	27.8%
65 +	19.6%	14.9%	15.7%
Income			
<\$20,000	21.3%	15.2%	18.2%
\$20,000 – \$50,000	33.0%	43.1%	34.3%
\$50,000 - \$75,000	20.6%	15.2%	20.2%
>\$75,000	25.1%	26.5%	27.3%
Marital Status			
Married	56.9%	53.2%	52.0%
Separated/Widowed/Divorced	24.7%	21.9%	17.8%
Never Married	18.4%	24.9%	30.2%

Appendix A5: Sample Distribution Comparison

SPONSOR: Walter & Elise Haas Fund

STATE/County: California/San Francisco

	BRFSS	SCCBS	Census
Population	95	500	776733
Sex			
Male	48.4%	49.8%	50.8%
Female	51.6%	50.2%	49.2%
Race			
White	74.7%	52.2%	53.0%
Black	7.4%	4.7%	8.6%
Hispanic	14.7%	20.1%	14.1%
Other	17.9%	43.1%	43.1%
Education			
0 – 12 (no diploma)	6.3%	8.3%	18.8%
12	9.5%	14.2%	13.9%
13-15	24.2%	37.5%	22.4%
≥ 16	60.0%	40.0%	45.0%
Age			
20 – 34	34.0%	44.9%	36.4%
35 – 44	18.1%	23.1%	20.6%
45 – 64	33.0%	24.0%	26.6%
65 +	14.9%	8.0%	16.3%
Income			
<\$20,000	19.8%	12.5%	19.0%
\$20,000 – \$50,000	25.3%	31.1%	26.6%
\$50,000 - \$75,000	16.5%	15.4%	17.7%
>\$75,000	38.5%	41.0%	36.7%
Marital Status			
Married	33.8%	37.7%	38.7%
Separated/Widowed/Divorced	30.0%	13.1%	16.6%
Never Married	36.3%	49.3%	44.8%

Appendix A6: Sample Distribution Comparison

SPONSOR: C.F. Serving Boulder County

STATE/County: Colorado/Boulder

	BRFSS	SCCBS	Census
Population	124	500	291288
Sex			
Male	44.4%	46.8%	50.6%
Female	55.6%	53.3%	49.4%
Race			
White	90.2%	89.9%	90.5%
Black	0.8%	0.8%	1.2%
Hispanic	7.3%	8.3%	10.5%
Other	9.0%	9.3%	10.6%
Education			
0 – 12 (no diploma)	3.2%	6.2%	7.2%
12	12.1%	15.2%	15.1%
13-15	25.8%	36.6%	25.3%
≥ 16	58.9%	42.1%	52.4%
Age			
20 – 34	30.8%	29.3%	34.9%
35 – 44	25.8%	27.3%	24.0%
45 – 64	29.2%	31.3%	30.0%
65 +	14.2%	12.1%	10.6%
Income			
<\$20,000	14.2%	11.9%	15.0%
\$20,000 – \$50,000	40.7%	32.0%	29.2%
\$50,000 - \$75,000	16.8%	22.7%	20.5%
>\$75,000	28.3%	33.4%	35.5%
Marital Status			
Married	57.4%	54.7%	52.1%
Separated/Widowed/Divorced	15.7%	13.5%	15.0%
Never Married	27.0%	31.7%	32.9%

Appendix A7: Sample Distribution Comparison

SPONSOR: Denver Foundation/Rose C.F./Piton Foundation

STATE/County: Colorado/Denver

	BRFSS	SCCBS	Census
Population	228	501	554636
Sex			
Male	39.9%	46.1%	50.5%
Female	60.1%	53.9%	49.5%
Race			
White	76.1%	66.9%	68.3%
Black	12.0%	11.6%	12.1%
Hispanic	22.8%	28.7%	31.7%
Other	12.0%	21.6%	23.5%
Education			
0 – 12 (no diploma)	17.1%	17.0%	21.1%
12	18.0%	18.7%	20.0%
13-15	21.1%	28.1%	24.4%
≥ 16	43.9%	36.1%	34.5%
Age			
20 – 34	33.5%	40.5%	38.0%
35 – 44	19.4%	23.2%	20.6%
45 – 64	31.8%	19.7%	26.5%
65 +	14.3%	16.5%	14.9%
Income			
<\$20,000	24.2%	13.0%	22.6%
\$20,000 – \$50,000	40.6%	48.3%	38.5%
\$50,000 - \$75,000	16.4%	18.0%	18.3%
>\$75,000	18.8%	20.7%	20.6%
Marital Status			
Married	39.5%	49.0%	43.2%
Separated/Widowed/Divorced	29.4%	18.4%	20.8%
Never Married	31.2%	32.6%	35.9%

Appendix A8: Sample Distribution Comparison

SPONSOR: Delaware Division of State Service Centers/Delaware C.F.

STATE: Delaware

	BRFSS	SCCBS	Census
Population	3514	1379	783600
Sex			
Male	38.7%	48.0%	48.6%
Female	61.3%	52.0%	51.4%
Race			
White	81.0%	75.2%	75.9%
Black	14.6%	18.5%	20.1%
Hispanic	2.8%	5.4%	4.8%
Other	4.5%	6.3%	5.9%
Education			
0 – 12 (no diploma)	9.3%	17.3%	17.4%
12	34.5%	28.4%	31.4%
13-15	25.7%	31.1%	26.1%
≥ 16	30.5%	23.2%	25.0%
Age			
20 – 34	23.3%	27.1%	28.4%
35 – 44	21.8%	24.7%	22.6%
45 – 64	33.3%	30.0%	31.0%
65 +	21.6%	18.2%	18.0%
Income			
<\$20,000	20.1%	13.9%	17.7%
\$20,000 – \$50,000	40.4%	40.6%	35.0%
\$50,000 - \$75,000	17.9%	20.3%	21.3%
>\$75,000	21.6%	25.3%	26.0%
Marital Status			
Married	53.5%	59.2%	54.0%
Separated/Widowed/Divorced	27.7%	18.4%	18.7%
Never Married	18.8%	22.4%	27.2%

Appendix A9: Sample Distribution Comparison

Sponsor: C.F. For Greater Atlanta

State/Counties: Georgia/ DeKalb, Fulton, Cobb, Rockdale, Henry

	BRFSS	SCCBS	Census
Population	646	510	2279074
Sex			
Male	40.3%	46.9%	49.1%
Female	59.8%	53.1%	50.9%
Race			
White	59.3%	60.5%	54.7%
Black	34.1%	31.9%	38.9%
Hispanic	2.0%	4.0%	6.8%
Other	6.6%	7.6%	8.2%
Education			
0 – 12 (no diploma)	7.0%	8.8%	14.4%
12	17.5%	23.8%	21.1%
13-15	22.5%	35.3%	26.7%
≥ 16	53.1%	32.1%	37.8%
Age			
20 – 34	28.5%	29.9%	35.9%
35 – 44	27.6%	25.5%	24.3%
45 – 64	31.6%	31.2%	28.8%
65 +	12.2%	13.4%	11.0%
Income			
<\$20,000	11.5%	9.6%	16.0%
\$20,000 – \$50,000	36.2%	36.1%	32.0%
\$50,000 - \$75,000	16.1%	21.5%	20.5%
> \$75,000	36.3%	32.7%	31.5%
Marital Status			
Married	49.5%	56.4%	48.6%
Separated/Widowed/Divorced	22.2%	19.1%	17.9%
Never Married	28.4%	24.6%	33.5%

Appendix A10: Sample Distribution Comparison

SPONSOR: Indiana Grantmakers Alliance

STATE: Indiana

	BRFSS	SCCBS	Census
Population	3993	1001	6080485
Sex			
Male	40.4%	48.3%	49.0%
Female	59.6%	51.7%	51.0%
Race			
White	90.7%	88.5%	88.6%
Black	6.2%	6.3%	8.8%
Hispanic	2.9%	5.3%	3.5%
Other	3.0%	5.3%	3.9%
Education			
0 – 12 (no diploma)	10.4%	18.2%	17.9%
12	38.7%	28.2%	37.2%
13-15	24.1%	28.5%	25.5%
≥ 16	26.8%	25.1%	19.4%
Age			
20 – 34	26.4%	25.5%	29.1%
35 – 44	21.4%	25.4%	22.3%
45 – 64	33.1%	32.7%	31.2%
65 +	19.1%	16.4%	17.4%
Income			
<\$20,000	18.7%	16.3%	20.8%
\$20,000 – \$50,000	47.0%	43.2%	38.5%
\$50,000 - \$75,000	18.7%	22.2%	21.4%
>\$75,000	15.6%	18.3%	19.5%
Marital Status			
Married	56.7%	60.1%	56.3%
Separated/Widowed/Divorced	28.1%	17.4%	18.8%
Never Married	15.3%	22.5%	24.8%

Appendix A11: Sample Distribution Comparison

SPONSOR: Forum 35/Baton Rouge Area Foundation

STATE/County: Louisiana/East Baton Rouge Parish

	BRFSS	SCCBS	Census
Population	461	500	412852
Sex			
Male	39.9%	46.1%	47.9%
Female	60.1%	53.9%	52.1%
Race			
White	63.8%	59.1%	56.9%
Black	32.1%	36.3%	40.5%
Hispanic	1.7%	2.7%	1.8%
Other	4.0%	4.6%	3.7%
Education			
0 – 12 (no diploma)	7.6%	8.3%	16.1%
12	24.6%	21.8%	26.4%
13-15	28.1%	41.4%	26.7%
≥ 16	39.7%	28.5%	30.8%
Age			
20 – 34	36.3%	30.0%	34.4%
35 – 44	16.8%	23.8%	21.3%
45 – 64	32.7%	30.7%	30.0%
65 +	14.3%	15.5%	14.3%
Income			
<\$20,000	20.1%	19.8%	27.7%
\$20,000 – \$50,000	41.7%	37.1%	34.6%
\$50,000 - \$75,000	16.6%	19.4%	17.1%
>\$75,000	21.6%	23.7%	20.7%
Marital Status			
Married	48.1%	52.2%	47.4%
Separated/Widowed/Divorced	25.2%	18.2%	18.3%
Never Married	26.7%	29.6%	34.2%

Appendix A12: Sample Distribution Comparison

SPONSOR: Kalamazoo C.F.

STATE/County: Michigan/Kalamazoo

	BRFSS	SCCBS	Census
Population	89	500	238603
Sex			
Male	44.9%	50.0%	48.4%
Female	55.1%	50.0%	51.6%
Race			
White	85.4%	86.0%	86.5%
Black	9.0%	9.4%	10.8%
Hispanic	2.3%	2.2%	2.6%
Other	5.6%	4.6%	5.3%
Education			
0 – 12 (no diploma)	12.4%	5.6%	11.3%
12	23.6%	23.3%	25.9%
13-15	22.5%	43.4%	31.6%
≥ 16	41.6%	27.7%	31.1%
Age			
20 – 34	36.1%	30.3%	33.8%
35 – 44	15.1%	22.6%	20.6%
45 – 64	30.2%	30.6%	29.6%
65 +	18.6%	16.6%	16.0%
Income			
<\$20,000	27.6%	12.4%	22.1%
\$20,000 – \$50,000	38.2%	41.2%	36.5%
\$50,000 - \$75,000	6.6%	21.3%	20.0%
>\$75,000	27.6%	25.1%	21.5%
Marital Status			
Married	54.7%	54.9%	51.6%
Separated/Widowed/Divorced	23.3%	16.6%	16.5%
Never Married	22.1%	28.5%	31.9%

Appendix A13: Sample Distribution Comparison

SPONSOR: C.F. for Southeastern Michigan

STATE/Counties: MICHIGAN/Wayne, Oakland, Macomb, St.Clair,
Washtenaw, Monroe, Livingston

	BRFSS	SCCBS	Census
Population	1554	501	4833493
Sex			
Male	38.2%	50.0%	48.7%
Female	61.8%	50.0%	51.3%
Race			
White	72.8%	69.5%	73.8%
Black	20.5%	21.7%	22.5%
Hispanic	3.2%	3.1%	2.8%
Other	6.7%	8.8%	5.9%
Education			
0 – 12 (no diploma)	8.5%	9.9%	17.2%
12	27.7%	28.4%	28.3%
13-15	30.5%	41.2%	29.8%
≥ 16	33.4%	20.5%	24.7%
Age			
20 – 34	24.7%	27.8%	29.2%
35 – 44	23.9%	22.9%	23.1%
45 – 64	34.1%	31.4%	31.2%
65 +	17.4%	17.8%	16.5%
Income			
<\$20,000	14.7%	9.9%	18.5%
\$20,000 – \$50,000	39.6%	37.7%	31.7%
\$50,000 - \$75,000	18.8%	23.7%	20.4%
>\$75,000	26.9%	28.7%	29.6%
Marital Status			
Married	50.7%	54.8%	51.3%
Separated/Widowed/Divorced	27.8%	19.5%	18.9%
Never Married	21.6%	25.7%	29.8%

Appendix A14: Sample Distribution Comparison

Sponsor: The St. Paul Foundation

State/Counties: Minnesota/Dakota, Ramsey, Washington

	BRFSS	SCCBS	Census
Population	844	503	1068069
Sex			
Male	41.2%	46.9%	48.9%
Female	58.8%	53.1%	51.1%
Race			
White	91.7%	88.6%	86.9%
Black	4.4%	1.9%	5.6%
Hispanic	1.9%	4.1%	3.9%
Other	3.9%	9.5%	9.9%
Education			
0 – 12 (no diploma)	5.1%	7.9%	9.3%
12	21.1%	17.7%	24.8%
13-15	32.0%	25.8%	31.4%
≥ 16	41.8%	48.6%	34.4%
Age			
20 – 34	26.2%	35.8%	30.6%
35 – 44	27.0%	26.4%	25.2%
45 – 64	31.0%	25.7%	30.6%
65 +	15.8%	12.1%	13.5%
Income			
<\$20,000	8.4%	6.1%	13.2%
\$20,000 – \$50,000	36.7%	37.9%	33.4%
\$50,000 - \$75,000	20.7%	25.2%	23.2%
>\$75,000	34.2%	30.8%	32.2%
Marital Status			
Married	55.5%	62.0%	55.6%
Separated/Widowed/Divorced	25.1%	12.0%	15.1%
Never Married	19.5%	26.0%	29.2%

Appendix A15: Sample Distribution Comparison

SPONSOR: Montana C.F.

STATE: Montana

	BRFSS	SCCBS	Census
Population	3338	502	902195
Sex			
Male	42.6%	49.1%	49.8%
Female	57.4%	50.9%	50.2%
Race			
White	86.4%	89.4%	92.2%
Black	0.2%	0.6%	0.5%
Hispanic	2.5%	3.9%	2.0%
Other	13.5%	10.0%	9.2%
Education			
0 – 12 (no diploma)	10.9%	13.9%	12.9%
12	34.1%	29.2%	31.3%
13-15	29.1%	29.6%	31.5%
≥ 16	25.9%	27.3%	24.4%
Age			
20 – 34	19.8%	27.6%	25.1%
35 – 44	20.1%	24.8%	22.0%
45 – 64	37.6%	31.7%	34.2%
65 +	22.5%	15.9%	18.8%
Income			
<\$20,000	25.5%	22.8%	28.8%
\$20,000 – \$50,000	52.7%	46.9%	42.2%
\$50,000 - \$75,000	12.5%	18.8%	17.2%
>\$75,000	9.3%	11.5%	11.9%
Marital Status			
Married	56.3%	63.9%	57.3%
Separated/Widowed/Divorced	30.4%	17.8%	18.7%
Never Married	13.3%	18.3%	24.0%

Appendix A16: Sample Distribution Comparison
SPONSOR: New Hampshire Charitable Foundation
STATE: New Hampshire

	BRFSS	SCCBS	Census
Population	4068	711	1235786
Sex			
Male	42.5%	49.6%	49.2%
Female	57.5%	50.4%	50.8%
Race			
White	96.0%	94.9%	97.0%
Black	0.4%	0.9%	1.0%
Hispanic	1.6%	1.6%	1.7%
Other	3.6%	4.2%	3.2%
Education			
0 – 12 (no diploma)	7.4%	7.0%	12.6%
12	29.8%	31.4%	30.1%
13-15	26.5%	37.4%	28.7%
≥ 16	36.4%	24.3%	28.7%
Age			
20 – 34	22.0%	25.2%	25.7%
35 – 44	25.5%	24.2%	24.8%
45 – 64	35.3%	32.4%	32.9%
65 +	17.2%	18.2%	16.6%
Income			
<\$20,000	12.3%	13.4%	16.0%
\$20,000 – \$50,000	40.4%	39.7%	34.6%
\$50,000 - \$75,000	21.6%	24.5%	23.1%
>\$75,000	25.7%	22.4%	26.5%
Marital Status			
Married	59.4%	57.5%	57.3%
Separated/Widowed/Divorced	25.2%	19.6%	17.8%
Never Married	15.4%	22.9%	24.9%

Appendix A17: Sample Distribution Comparison

SPONSOR: Central New York C.F.

STATE/County: New York/Onondaga

	BRFSS	SCCBS	Census
Population	106	541	458336
Sex			
Male	37.7%	45.7%	47.8%
Female	62.3%	54.3%	52.2%
Race			
White	93.3%	82.4%	86.4%
Black	4.8%	9.2%	10.3%
Hispanic	2.8%	2.7%	2.4%
Other	1.9%	8.4%	5.3%
Education			
0 – 12 (no diploma)	7.7%	5.0%	14.3%
12	26.0%	29.5%	29.1%
13-15	27.9%	40.4%	28.1%
≥ 16	38.5%	25.1%	28.4%
Age			
20 – 34	20.0%	27.8%	27.0%
35 – 44	23.0%	22.3%	22.6%
45 – 64	34.0%	30.3%	31.0%
65 +	23.0%	19.7%	19.4%
Income			
<\$20,000	19.8%	16.7%	23.9%
\$20,000 – \$50,000	34.1%	39.9%	35.4%
\$50,000 - \$75,000	22.0%	20.7%	19.4%
>\$75,000	24.2%	22.8%	21.3%
Marital Status			
Married	51.5%	53.1%	50.9%
Separated/Widowed/Divorced	30.1%	19.1%	18.6%
Never Married	18.5%	27.8%	30.4%

Appendix A18: Sample Distribution Comparison

SPONSOR: Rochester Area C.F.

STATE/Counties: NEW YORK/Monroe, Wayne, Ontario,
Livingston, Genesee, Orleans

	BRFSS	SCCBS	Census
Population	164	988	1098201
Sex			
Male	34.2%	45.7%	48.6%
Female	65.9%	54.3%	51.4%
Race			
White	84.7%	84.4%	85.3%
Black	9.2%	9.4%	11.1%
Hispanic	4.3%	2.7%	4.3%
Other	6.1%	6.3%	5.4%
Education			
0 – 12 (no diploma)	5.5%	7.2%	15.7%
12	26.2%	30.7%	29.1%
13-15	28.1%	38.9%	28.1%
≥ 16	40.2%	23.3%	27.1%
Age			
20 – 34	26.0%	27.8%	26.9%
35 – 44	24.1%	22.4%	23.0%
45 – 64	32.9%	31.3%	32.0%
65 +	17.1%	18.5%	18.1%
Income			
<\$20,000	16.3%	13.2%	20.6%
\$20,000 – \$50,000	38.8%	45.1%	35.6%
\$50,000 - \$75,000	15.0%	22.3%	20.9%
>\$75,000	29.9%	19.4%	22.9%
Marital Status			
Married	50.9%	54.7%	52.6%
Separated/Widowed/Divorced	25.8%	21.6%	18.6%
Never Married	23.3%	23.7%	28.8%

Appendix A19: Sample Distribution Comparison

SPONSOR: Winston-Salem Foundation

STATE/County: North Carolina/Forsyth

	BRFSS	SCCBS	Census
Population	454	750	306067
Sex			
Male	38.8%	48.0%	47.8%
Female	61.2%	52.0%	52.2%
Race			
White	70.7%	71.8%	69.5%
Black	26.9%	23.8%	26.2%
Hispanic	1.8%	5.3%	6.4%
Other	2.5%	4.4%	5.7%
Education			
0 – 12 (no diploma)	13.5%	11.7%	18.0%
12	27.0%	28.1%	27.0%
13-15	24.6%	35.4%	26.4%
≥ 16	35.0%	24.9%	28.7%
Age			
20 – 34	22.3%	27.4%	29.6%
35 – 44	19.8%	22.8%	22.1%
45 – 64	33.5%	31.6%	31.1%
65 +	24.5%	18.1%	17.2%
Income			
<\$20,000	20.8%	16.0%	21.4%
\$20,000 – \$50,000	46.8%	41.0%	36.6%
\$50,000 - \$75,000	17.8%	21.7%	20.4%
>\$75,000	14.6%	21.3%	21.5%
Marital Status			
Married	49.9%	58.5%	54.9%
Separated/Widowed/Divorced	31.2%	21.6%	19.4%
Never Married	18.9%	19.9%	25.7%

Appendix A20: Sample Distribution Comparison

SPONSOR: C.F. of Greater Greensboro

STATE/County: North Carolina/Guilford

	BRFSS	SCCBS	Census
Population	413	750	421048
Sex			
Male	35.6%	48.0%	47.9%
Female	64.4%	52.0%	52.1%
Race			
White	68.7%	68.5%	65.5%
Black	26.7%	25.6%	29.9%
Hispanic	2.4%	3.1%	3.8%
Other	4.7%	5.9%	6.1%
Education			
0 – 12 (no diploma)	11.4%	8.3%	17.1%
12	25.6%	27.8%	25.1%
13-15	26.3%	38.1%	27.6%
≥ 16	36.7%	25.8%	30.3%
Age			
20 – 34	24.6%	27.4%	31.7%
35 – 44	22.6%	23.1%	21.8%
45 – 64	31.8%	32.1%	30.3%
65 +	20.9%	17.5%	16.1%
Income			
<\$20,000	20.8%	14.6%	20.1%
\$20,000 – \$50,000	45.2%	41.5%	37.5%
\$50,000 - \$75,000	16.0%	21.7%	19.9%
>\$75,000	18.1%	22.2%	22.4%
Marital Status			
Married	46.4%	57.6%	52.9%
Separated/Widowed/Divorced	31.2%	17.9%	18.6%
Never Married	22.4%	24.5%	28.5%

Appendix A21: Sample Distribution Comparison

SPONSOR: Cleveland Foundation

STATE/County: Ohio/Cuyahoga

	BRFSS	SCCBS	Census
Population	459	1100	1393978
Sex			
Male	37.7%	46.6%	47.2%
Female	62.3%	53.4%	52.8%
Race			
White	73.7%	68.5%	68.7%
Black	22.6%	25.6%	28.2%
Hispanic	3.5%	0.7%	3.4%
Other	3.8%	5.9%	5.0%
Education			
0 – 12 (no diploma)	8.3%	7.7%	18.4%
12	30.1%	33.2%	30.0%
13-15	28.2%	37.6%	26.5%
≥ 16	33.4%	21.6%	25.2%
Age			
20 – 34	21.7%	24.2%	26.3%
35 – 44	23.9%	21.8%	21.7%
45 – 64	33.6%	31.7%	30.6%
65 +	20.8%	22.4%	21.4%
Income			
<\$20,000	16.3%	15.8%	24.9%
\$20,000 – \$50,000	49.0%	45.4%	36.5%
\$50,000 - \$75,000	14.5%	21.1%	18.4%
>\$75,000	20.2%	17.7%	20.4%
Marital Status			
Married	46.8%	51.1%	49.4%
Separated/Widowed/Divorced	29.2%	21.4%	21.9%
Never Married	24.1%	27.6%	30.6%

Appendix A22: Sample Distribution Comparison

SPONSOR: Greater Cincinnati Foundation

STATE/Counties: OHIO/Butler, Clermont, Hamilton, Warren

STATE/Counties: Kentucky/Boone, Campbell, Kenton

STATE/Counties: Dearborn

	BRFSS	SCCBS	Census
Population	1038	1001	1886650
Sex			
Male	40.9%	46.4%	48.5%
Female	59.1%	53.6%	51.5%
Race			
White	87.5%	84.5%	85.6%
Black	10.5%	11.8%	12.7%
Hispanic	1.8%	2.2%	1.1%
Other	2.1%	3.7%	2.9%
Education			
0 – 12 (no diploma)	10.6%	10.8%	17.0%
12	32.6%	34.5%	31.1%
13-15	26.3%	34.1%	26.2%
≥ 16	30.5%	20.7%	25.8%
Age			
20 – 34	28.6%	26.7%	29.3%
35 – 44	21.2%	23.2%	23.4%
45 – 64	30.8%	31.6%	30.7%
65 +	19.4%	18.5%	16.6%
Income			
<\$20,000	17.3%	13.3%	19.5%
\$20,000 – \$50,000	42.6%	40.5%	34.9%
\$50,000 - \$75,000	17.8%	23.4%	21.0%
>\$75,000	22.3%	22.8%	24.6%
Marital Status			
Married	52.3%	59.9%	54.5%
Separated/Widowed/Divorced	27.0%	20.2%	18.4%
Never Married	20.7%	19.9%	27.2%

Appendix A23: Sample Distribution Comparison
SPONSOR: Northwest Area Foundation
STATE/County: OREGON/Crook, Deschutes, Jefferson

	BRFSS	SCCBS	Census
Population	99	500	153558
Sex			
Male	47.5%	48.9%	49.8%
Female	52.5%	51.2%	50.2%
Race			
White	96.9%	90.5%	93.3%
Black	0.0%	1.7%	0.4%
Hispanic	5.1%	5.0%	5.7%
Other	3.1%	7.8%	8.5%
Education			
0 – 12 (no diploma)	3.0%	10.1%	14.1%
12	38.4%	34.8%	29.2%
13-15	34.3%	37.3%	34.7%
≥ 16	24.2%	17.8%	22.0%
Age			
20 – 34	29.8%	22.1%	24.9%
35 – 44	17.0%	22.3%	21.5%
45 – 64	34.0%	33.4%	35.3%
65 +	19.2%	22.2%	18.4%
Income			
<\$20,000	12.4%	15.1%	20.2%
\$20,000 – \$50,000	55.1%	47.1%	41.8%
\$50,000 - \$75,000	16.9%	20.0%	20.3%
>\$75,000	15.7%	17.9%	17.8%
Marital Status			
Married	62.0%	67.9%	61.6%
Separated/Widowed/Divorced	22.8%	15.5%	18.2%
Never Married	15.2%	16.7%	20.1%

Appendix A24: Sample Distribution Comparison

SPONSOR: York Foundation

STATE/County: Pennsylvania/York

	BRFSS	SCCBS	Census
Population	127	500	381751
Sex			
Male	40.9%	46.6%	49.2%
Female	59.1%	53.5%	50.8%
Race			
White	92.9%	93.5%	93.7%
Black	4.0%	1.9%	4.2%
Hispanic	2.4%	1.8%	3.0%
Other	3.2%	4.6%	3.2%
Education			
0 – 12 (no diploma)	8.7%	11.0%	19.3%
12	40.9%	42.7%	41.6%
13-15	23.6%	32.5%	20.7%
≥ 16	26.8%	13.8%	18.4%
Age			
20 – 34	30.9%	24.8%	25.0%
35 – 44	15.5%	22.9%	23.6%
45 – 64	40.7%	32.3%	33.0%
65 +	13.0%	19.9%	18.5%
Income			
<\$20,000	16.4%	13.7%	16.7%
\$20,000 – \$50,000	50.9%	40.9%	38.9%
\$50,000 - \$75,000	23.3%	24.2%	24.8%
>\$75,000	9.5%	21.2%	19.5%
Marital Status			
Married	61.8%	67.0%	60.3%
Separated/Widowed/Divorced	23.6%	16.7%	17.4%
Never Married	14.6%	16.4%	22.3%

Appendix A25: Sample Distribution Comparison

SPONSOR: Greater Houston C.F.

STATE/County: Texas/Harris

	BRFSS	SCCBS	Census
Population	802	500	3400578
Sex			
Male	41.0%	47.8%	49.8%
Female	59.0%	52.2%	50.2%
Race			
White	68.7%	63.7%	61.2%
Black	16.0%	19.6%	19.0%
Hispanic	24.9%	26.9%	32.9%
Other	15.3%	16.8%	22.9%
Education			
0 – 12 (no diploma)	15.7%	18.5%	25.4%
12	22.0%	22.3%	21.6%
13-15	23.7%	31.5%	26.0%
≥ 16	38.7%	27.7%	26.9%
Age			
20 – 34	31.5%	30.8%	35.7%
35 – 44	25.8%	26.1%	24.3%
45 – 64	30.7%	30.4%	29.1%
65 +	12.0%	12.7%	10.9%
Income			
<\$20,000	19.7%	20.4%	20.9%
\$20,000 – \$50,000	38.5%	39.9%	35.9%
\$50,000 - \$75,000	17.2%	15.7%	18.4%
>\$75,000	24.6%	24.0%	24.6%
Marital Status			
Married	51.7%	54.7%	53.8%
Separated/Widowed/Divorced	26.7%	21.2%	17.4%
Never Married	21.6%	24.2%	28.7%

Appendix A26: Sample Distribution Comparison

SPONSOR: Northwest Area Foundation

STATE/County: Washington/Yakima

	BRFSS	SCCBS	Census
Population	119	500	222581
Sex			
Male	39.5%	46.9%	49.9%
Female	60.5%	53.1%	50.1%
Race			
White	93.2%	77.4%	68.6%
Black	0.9%	2.4%	1.4%
Hispanic	14.3%	30.1%	35.9%
Other	5.9%	20.2%	33.7%
Education			
0 – 12 (no diploma)	17.7%	23.5%	31.4%
12	35.3%	30.3%	27.4%
13-15	26.9%	32.1%	26.0%
≥ 16	20.2%	14.1%	15.3%
Age			
20 – 34	21.7%	26.2%	30.8%
35 – 44	18.3%	21.7%	21.7%
45 – 64	27.8%	31.6%	30.3%
65 +	32.2%	20.5%	17.2%
Income			
<\$20,000	25.2%	23.0%	26.8%
\$20,000 – \$50,000	48.5%	49.0%	40.6%
\$50,000 - \$75,000	17.5%	17.7%	18.4%
>\$75,000	8.7%	10.4%	14.5%
Marital Status			
Married	58.0%	62.2%	56.9%
Separated/Widowed/Divorced	26.9%	20.6%	17.5%
Never Married	15.1%	17.3%	25.7%

Appendix A27: Sample Distribution Comparison

SPONSOR: Greater Kanawha Valley Foundation

STATE/Counties: WEST VIRGINIA/Kanawha, Putnam, Boone

	BRFSS	SCCBS	Census
Population	497	500	277197
Sex			
Male	40.4%	46.1%	48.0%
Female	59.6%	53.9%	52.0%
Race			
White	93.5%	90.3%	93.6%
Black	3.8%	5.0%	5.7%
Hispanic	1.0%	2.3%	0.6%
Other	2.6%	4.8%	1.8%
Education			
0 – 12 (no diploma)	14.5%	11.2%	21.2%
12	36.9%	35.7%	37.1%
13-15	26.4%	37.6%	22.9%
≥ 16	22.1%	15.5%	19.2%
Age			
20 – 34	22.5%	22.6%	24.8%
35 – 44	21.1%	21.8%	20.9%
45 – 64	36.4%	34.3%	34.0%
65 +	20.0%	21.3%	20.2%
Income			
<\$20,000	22.9%	19.7%	29.1%
\$20,000 – \$50,000	47.0%	44.6%	38.0%
\$50,000 - \$75,000	16.8%	22.4%	17.4%
>\$75,000	13.4%	13.3%	15.7%
Marital Status			
Married	57.1%	64.0%	56.8%
Separated/Widowed/Divorced	27.7%	18.1%	21.9%
Never Married	15.3%	17.9%	21.4%

About the Author

Caroline Mae McKay received her B.A. in English from University of Miami. She attended Florida State University School of Social Work, where she graduated with honors with a master's degree in clinical social work. After practicing in Miami with the homeless mentally-ill veteran population, she came to the doctoral program in the Department of Community and Family Health, University of South Florida College of Public Health. Focus areas of her work include social structural and contextual influences on health and health behavior, theoretical development in Public Health, and multilevel modeling.