### CONTRIBUTIONS OF THE SOCIAL ENVIRONMENT TO CENTRAL ADIPOSITY: RESULTS FROM ETIOLOGIC AND INTERVENTION STUDIES

by

Jamila L. Kwarteng

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Doctoral Committee:

Professor Amy J. Schulz, Chair Professor Barbara A. Israel Programmer Analyst Graciela B. Mentz Associate Professor Trina R. Shanks © Jamila L. Kwarteng 2014

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# List of Abbreviations

NHB(s)- Non-Hispanic Black

NHW(s)- Non-Hispanic White

# **List of Acronyms**

**BMI-Body Mass Index** 

CVD-Cardiovascular Disease

EDC- Epidemiologic Diabetic Complications

EHDIC-SWB-Exploring Health Disparities in Integrated Communities-Southwest

Baltimore

HEP- Healthy Environments Partnership

HPA Axis- Hypothalamic Pituitary Adrenal Axis

METs- Metabolic Equivalents

NHIS- National Health Interview Study

SES- Socioeconomic Status

SWAN- Study of Women Across the Nation

WYHH- Walk Your Heart to Health

#### ABSTRACT

Evidence is building regarding the relationship of the social environment to central adiposity risk. Proxies of the social environment include indicators of psychosocial stress, such as neighborhood poverty, perceived discrimination, and family stress. While some research has theorized that psychosocial stress influences central adiposity indirectly through poor health behaviors, other studies have theorized that psychosocial stress directly influences central adiposity via biophysiological pathways. However, these pathways have rarely been examined. The purpose of this dissertation is to examine both direct and indirect pathways that psychosocial stress may influence central adiposity. First, I examine several direct pathways that psychosocial stress (i.e. perceived discrimination, family stress, and cumulative stress index) may influence changes in central adiposity over time; and ways that neighborhood poverty, may influence changes in central adjointly through both direct and mediating pathways. Next, I examine indirect pathways that psychosocial stress may effect central adiposity. I examined a sample of 386 Non-Hispanic Black, Non-Hispanic White, and Hispanic adults of waves I and II of the Healthy Environments Partnership community surveys 2002-2003 and 2008, respectively. In addition, I analyzed an intervention study of 473 adults who were predominately NHB and Hispanic women. Results include 1) a positive association between perceived discrimination and central adiposity over time; 2) in general, a null association between multiple indicators of psychosocial stress and changes in central adiposity over time, with the exception of family stress; 3) a positive association between cumulative stress index (i.e. composite measure of psychosocial stress) at baseline changes in central adiposity over time; 4) neighborhood economic composition did not mediate the association between cumulative stress index and

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changes in central adiposity over time; and 5) a null association between perceived safety and participation in the physical-activity-intervention; 6) perceived safety modified the associations between participation and physical activity; however, perceived safety did not modify the association between physical activity and central adiposity. The understanding of these factors may help to elucidate the direct and indirect pathways between psychosocial stress and central adiposity, which may help public health professionals target interventions more effectively towards the factors that drives these changes.

#### Chapter 1

#### Introduction

Increasing prevalence of obesity is a growing public health concern. Although the prevalence of obesity in the United States has increased dramatically in the 20<sup>th</sup> century (Wang & Beydoun, 2007), recently, these trends have slowed and are starting to plateau at a very high level (Flegal, 2010; Ogden, 2010). This high level of obesity is a matter of concern because it is a precursor for several chronic diseases such as cardiovascular disease (CVD) (Truesdale et al., 2006); type II diabetes (Gregg et al., 2005); stroke (Caterson et al., 2004); breast cancer (Carmichael & Bates, 2004), endometrial cancer (Kaaks, Lukanova, & Kurzer, 2002), and ovarian cancer (Pan, Johnson, Ugnat, Wen, & Mao, 2004). Obesity can be assessed in several ways, including body mass index (BMI), waist circumference, and waist-to-hip ratio. BMI is a measure of overall obesity that does not measure where weight is distributed in the body (Azarbad & Gonder-Frederick, 2010). Some studies have found that measures that focus on central adiposity, such as waist circumference and waist-to-hip ratio, have stronger predictive power for various health outcomes than body mass index (BMI) (Després, 2012; Janssen, Katzmarzyk, & Ross, 2004). Particularly important in this issue is evidence suggesting that central adiposity, fat that accumulates in the mid-section, may be more harmful to health than fat that accumulates in other parts of the body (Després, 2012; Kumanyika et al., 2008). During the past 50 years, the prevalence of central adiposity has increased in the US (Beydoun & Wang, 2009; Okosun et al., 2004). Although the rate of increase has been similar for White, Black, and Mexican American men, obesity rates among Black and White women have

increased at a greater rate than among Mexican American women (Beydoun & Wang, 2009; Flegal, Carroll, Ogden, & Curtin, 2010; Okosun et al., 2004). The factors contributing to these differential increases in central adiposity are poorly understood. Understanding the processes that are associated with increases in central adiposity over time among adults can contribute to our understanding of the factors that contribute to excess risk for chronic disease and help public health professionals target interventions more effectively towards the factors that drive these changes.

Obesity interventions have generally focused on improving modifiable individual behaviors (Azarbad & Gonder-Frederick, 2010), such as diet and physical activity. While some of these interventions have been effective in achieving short-term results, few have been effective over the long-term (Kumanyika et al., 2008). Increasingly, researchers have called for strategies that address multiple levels of an ecological model (Kumanyika et al., 2008). These strategies have included efforts to address changes in the environment, which have been increasingly recognized in their role in shaping individual behaviors. One potential driver of changes in the neighborhood environment is neighborhood poverty, which has been associated with several health outcomes, including obesity (Amy J Schulz et al., 2013).

My conceptual model outlined below describes the theoretical pathways of neighborhood poverty and its potential influence on obesity. In particular, this model is a way of theorizing relationships between racial ideologies, racial segregation, and relationships between stress and health outcomes. It builds on literature describing relationships between racial ideologies, racial segregation, differential access to material resources and pathways to health (A Schulz &

Northridge, 2004; D. Williams & Collins, 2001; D.R. Williams & Williams-Morris, 2000); and research hypothesizing relationships between stress and central adiposity (P Björntorp, 1987; P. Björntorp, 1990, 1992, 1996, 1997, 2009; P. Björntorp, Holm, & Rosmond, 2001; B.S. McEwen & Seeman, 2006). As an adaptation of The Healthy Environments Partnership Conceptual Model (AJ Schulz et al., 2005), this model focuses specifically on organizing associations between these factors (i.e. neighborhood economic composition, everyday unfair treatment, perceived neighborhood physical environment, perceived neighborhood social environment, family stress, friend/neighbor stress, safety stress, and financial vulnerability) and central adiposity, as a key contributor to racial, ethnic, and socioeconomic disparities in cardiovascular disease (CVD).

Specifically, the conceptual model in Figure 1 suggests that the social environment is one pathway that may mediate the relationships between societal factors (i.e. racial ideologies, race-based residential segregation, and differential access to material resources) and health outcomes, namely central adiposity and physical activity, above and beyond the effects of individual health behaviors such as diet, smoking, and alcohol use. Potential mediating pathways in the social environment include, for example, everyday unfair treatment, perceptions of the neighborhood physical and social environment, family stress, friend/neighbor stress, safety stress and financial vulnerability as an indicator of psychosocial stress. The HEP conceptual model posits that associations between the fundamental and intermediate factors noted and health outcomes travel through multiple pathways, including health related behaviors and stress (Schulz et al 2005). In this adaptation of the model, pathways associated with psychosocial stress are specified,

influencing the distribution of fat in the abdominal area, above and beyond the effects of behavioral factors. The grayed out box in the model was not directly tested in this dissertation. The dashed arrows represent pathways that were not tested, but were used as controls. The italicization and underlining of Metabolic Equivalents (METs), indicate that this was an intermediate outcome for this study.

#### Societal Factors

The persistence of racial ideologies and the process of race-based residential segregation has led to a redistribution of economic resources and disinvestment in segregated areas (Darden, Rahbar, Jezierski, Li, & Velie, 2010; Landrine & Corral, 2009; Massey & Denton, 1989; D. Williams & Collins, 2001). Historically, negative racial ideologies about minority groups, particularly Blacks in the context of the United States, led to the creation of policies that restricted access to educational, employment, and residential opportunities for residents of predominantly Black communities, while access to such resources was not restricted for nonminority residents of segregated White communities. (Darden et al., 2010; Landrine & Corral, 2009; Massey & Denton, 1989; Oliver & Shapiro, 2006; D.R. Williams & Williams-Morris, 2000). This difference in access led to current differential access to material resources. For example, the federal government worked with the real estate and banking industry to establish and enforce housing policies like redlining to physically separate Blacks from Whites resulting in race-based residential segregation (Darden et al., 2010; Landrine & Corral, 2009; Massey & Denton, 1989; Shapiro & Oliver, 1995; D.R. Williams & Williams-Morris, 2000). In turn, the



Figure 1. Conceptual model of theorized pathways to obesity

process of race-based residential segregation led to differential access to material resources. Disinvestment has resulted in the proliferation of low-paying jobs and limited opportunities for the accumulation of wealth which has had long-term effects on socioeconomic status (Darden et al., 2010; Landrine & Corral, 2009; Shapiro & Oliver, 1995; D. Williams & Collins, 2001). These long-term effects on socioeconomic status include limitations to educational advancement, the accumulation of wealth, homeownership, employment that includes adequate health insurance and retirement benefits. Ultimately, this differential access to material resources truncates socioeconomic status in a way that hampers segregated minorities' ability to relocate, reinforcing residential segregation. In other words, although no laws exist that prevent individuals from relocating to other areas, the lack of wealth and the inequitable distribution of resources preclude many residents of segregated, low resource neighborhoods from moving to different neighborhoods (Darden et al., 2010; Landrine & Corral, 2009; D. Williams & Collins, 2001).

#### Neighborhood Poverty

Collectively, these societal factors drive the spatial concentration of the risks and resources related to obesity risk in many complex ways. One way is how societal factors shape the spatial concentration of poverty (i.e. neighborhood poverty). Racial minorities such as non-Hispanic Blacks and Hispanics are disproportionately more likely to reside in high-poverty neighborhoods (i.e. 40% or more residents in poverty) (Jargowsky, 2013). Residing in concentrated poverty has a myriad of effects on neighborhoods, from level of crime and violence, educational attainment, marital status, and labor force participation (Jargowsky, 2013;

William J Wilson, 1990; William Julius Wilson, 2012). These effects are correlated to health outcomes including obesity.

Evidence strongly suggests that residents who reside in poverty are more likely to be obese (Boardman, Onge, Rogers, & Denney, 2005; Diez Roux & Mair, 2010; Lovasi, Hutson, Guerra, & Neckerman, 2009). On one hand, neighborhood poverty as a source of psychosocial stress (AJ Schulz et al., 2008; Amy Schulz et al., 2008) may influence obesity directly, through the stress process. This stress process can lead to physiological responses that may cause metabolic changes that directly influence the distribution of fat in the body (M. F. Dallman et al., 2004), particularly in the internal, visceral adipose tissue regions (P Björntorp, 1987; P. Björntorp, 1997; Brydon, 2011; Koch, Sepa, & Ludvigsson, 2008; B.S. McEwen & Seeman, 2006; Wardle, Chida, Gibson, Whitaker, & Steptoe, 2011). In turn, excess visceral fat that accumulates in the abdominal region—called central adiposity<sup>1</sup>-- place individuals at higher risk for adverse health outcomes. On the other hand, neighborhood poverty may exert its influence indirectly through the deterioration of the physical environment (Zenk et al., 2013; Kwarteng et al., 2013; Schulz, 2005) and/or the social environment in ways that are described below.

#### Neighborhood Physical Environment

Researchers have become increasingly aware of the ways that neighborhood physical environments contribute to obesity, such as poor access to fresh fruits and vegetables, high fast food density, fewer recreational facilities, and deteriorating parks and open spaces (Black &

<sup>&</sup>lt;sup>1</sup> Also called central obesity, intra-abdominal fat, or abdominal fat. Proxy measures for central adiposity include a high-risk waist circumference (i.e  $\ge 120$  cm in men and  $\ge 80$  cm in women) and high-risk waist-to-hip ratio ( $\ge 1.0$  in men and  $\ge 0.85$  in women), (Ding et al., 2004)

Macinko, 2008; Diez Roux & Mair, 2010; Kumanyika et al., 2008). For example, a recent review of the built environment and obesity in disadvantaged populations showed that obesityrelated health disparities in the United States have been associated with similar factors such as lack of supermarket access, exercise facilities, and safety (Lovasi et al., 2009). Supermarket access is a major factor of the healthy food environment of neighborhoods since it is a major determinant of the availability of fresh fruits and vegetable consumption in a community. Zenk and colleagues (2006) found that fruit and vegetable access differs by neighborhood racial composition and neighborhood socioeconomic position, with neighborhoods that were predominately non-Hispanic Black and low income having less access to fresh fruit and vegetables than racially heterogeneous low and middle income neighborhoods (Zenk et al., 2006). These findings highlight one of the effects of race-residential segregation and how it contributes to differential access to healthy foods and places for physical activity, which, in turn, affect the development obesity.

#### Neighborhood Social Environment

On the other hand, researchers are less aware of the factors of the social environment that contribute to the development of obesity. The social environment has been defined as social, economic, and political relationships at the local level (Hynes & Lopez, 2009; Lopez, 2012; AJ Schulz et al., 2005). Inequities in the social environment have led to an imbalance of power that disproportionately adversely impacts minorities and low-socioeconomic groups (Hynes & Lopez, 2009; Lopez, 2012). Research suggests that the effects of living in an economically disadvantaged neighborhood are independently associated with obesity and obesity-related behaviors (Boardman et al., 2005; Mobley et al., 2006). For example, a cross-sectional study

by Robert and Reither (2004) found that when controlling for age, race, and individual SES, women living in economically disadvantaged neighborhoods had a higher BMI compared to women living in less disadvantaged neighborhoods(Robert & Reither, 2004).

#### The stress process

An area of growing interest that may account for some of the stratification of health is exposure to stress, as it may shape health disparities in mid to late life for populations of varying social statuses (e.g. race, gender, SES) (Pearlin et al., 2005). According to Pearlin (1999), the stress process includes three components: stressors, moderators, and outcomes. The first component, stressors, refer to any condition of threat, demands, or structural constraint that has the potential to upset balance or homeostasis, that requires the need for adaptation in order to restore balance (Glanz & Schwartz, 2008; L. I. Pearlin & Schooler, 1978; Wheaton, 1999). The second component, moderators, include coping, social support, and mastery. Coping refers to things individuals do to avoid or minimize the effect of stressors (L. I. Pearlin, 1999). Social support includes various types of assistance or emotional uplift individuals' receive from their networks (L. I. Pearlin, 1999). Mastery refers to the individuals' sense of control over demanding situations (L. I. Pearlin, 1999). The last component, outcomes, refer to health and well-being (L. I. Pearlin, 1999). Each component of the stress process is described in greater detail below.

There are three major types of stressors: daily hassles, life events, and chronic stressors (Thoits, 1995). Daily hassles are small discrete events which require slight behavioral readjustments during the course of a day (e.g. heavy traffic, unexpected visitors) (Thoits, 1995; Wheaton, 1999). A widely accepted criticism of daily hassles is the inclusion of some measures

that can be characterized as other concepts such as distress (e.g. being lonely) and chronic stress (e.g. problems with divorce) (Dohrenwend, 2006). Nonetheless, daily hassles represent a unique type of stressor that is generally not captured by other measures (e.g. planning meals, weather) (Wheaton, 1999). Life events are discrete occurrences that require major behavioral adjustment within a short period of time (e.g. death of a spouse, birth of first child) (Thoits et al., 1995). Chronic stressors are constant demands that require readjustments over lengthy periods of time (e.g. poverty, marital problems) (Thoits, 1995). Chronic stressors are embedded in social structures, roles, and relationships that persist over time (L. I. Pearlin, 1999). Three classes of chronic stressors have been identified: status strains, role strains, and contextual strains (L. I. Pearlin, 1999). Status strains refer to stressors that stem directly from one's location in a socially stratified system that has an unequal distribution of resources, opportunities and life chances, power, and prestige (e.g. race/ethnicity, gender) (L. I. Pearlin, 1999). Role strains are stressors that stems from socially accepted roles such as wife, employee, or caregiver (L. I. Pearlin, 1999). Contextual strains stem from hardships and problems that originate from one's proximal environments such as neighborhood and community (L. I. Pearlin, 1999).

Moderators refer to resources that lessen the negative effects of stress (L. I. Pearlin, 1999). This function is exercised in several ways including buffering and additive or main effects. Stress buffering provides protection against intense stressors through coping, social support, or mastery. For example, people with a high level of mastery are likely to have positive mental health irrespective of the stressors they experience (L. I. Pearlin, 1999). Resources can also function as mediators. Mediation occurs when stressors act indirectly through their effect on resources to affect health outcomes.

Outcomes refer to health outcomes. The stress process literature has focused mainly on mental health outcomes such as depression or distress (Elliott, 2000; L. I. Pearlin, 1999; Wheaton, 1999). Physical health outcomes, like central adiposity, have also been considered among the health outcomes researched (P. Björntorp, 2009).

Studies have shown that one pathway through which stress influences obesity is by contributing to maladaptive health behaviors. For example, Dallman and colleagues (2003) found that stress can lead to the consumption of energy-dense foods such as sweets and foods that are high in fat (M. Dallman et al., 2003). The consumption of these type of foods effect metabolism and may be the underlying cause of lower basal metabolism in some groups (M. Dallman et al., 2003). Relatedly, Jackson and colleagues (2010) tested whether certain populations have an addictive relationship to food by examining the physiological pathways of psychosocial stress and obesity through the hypothalamic-pituitary-adrenalcortical axis (HPA axis) (Jackson, Knight, & Rafferty, 2010). They found some evidence of a food addiction among African American women who may eat more comfort foods to reduce anxiety via the HPA axis resulting in higher rates of obesity (Jackson et al., 2010; Amy J. Schulz et al., 2006). These two studies are consistent with the theory of allostatic load, "the long-term effect of the physiologic response to stress", which purports a biological pathway of the mediating effect of chronic perceived stress between maladaptive behaviors like overconsumption of unhealthy foods and allostatic load (B. McEwen, 1998).

Another maladaptive behavior associated with stress is physical inactivity. Physical activity has an independent effect on obesity and may also be mediated by stress. For example, Schulz and colleagues (2008) hypothesized that chronic psychosocial stress (i.e. financial vulnerability) may lead to increased obesity risk in African Americans through an independent

effect on physical activity (AJ Schulz et al., 2008). Another study found that stressors in the environment, such as safety from crime, have been associated with greater physical activity (Casagrande, Whitt-Glover, & Lancaster, Odoms-Young, & Gary, 2009). Conversely, lack of safe and free recreational facilities and parks —factors that are common in minority neighborhoods with low SES—are associated with lower rates of leisure physical activity (Schulz et al., 2005). Unfortunately, oftentimes individual interventions that focus solely on behavioral modification place the onus totally on the individual without consideration of the context in which these behaviors occur. However, research has shown that individual behaviors alone do not account for all of the stratification in health (Boardman et al., 2005; Elliott, 2000).

#### Stress and central adiposity

Several studies have researched the relationship between stress and central adiposity. Evidence suggests that stress influences central adiposity by affecting the metabolism via the nervous system by changing hormone levels (e.g. leptin, cortisol) associated with appetite and fat deposition. For example, exposure to stress reduces sensitivity to leptin, a hormone that regulates energy balance by suppressing appetite and stimulating sympathetic nervous system activity(Brydon, 2011; Sandoval & Davis, 2003). Earlier research hypothesized that the release of cortisol in response to stress activated the HPA, causing an increase in abdominal and visceral fat deposition (P Björntorp, 1987; P. Björntorp, 1992) . Several subsequent studies by Bjorntorp and colleagues have extended their research on stress by examining how abdominal fat contributes to the development of chronic diseases such as cardiovascular disease and type II diabetes (P. Björntorp, 1990, 1992, 1996, 1997, 2009). Recent studies continue to implicate stress in the accumulation of abdominal fat. There is some evidence that stress-related cortisol secretion is positively related to the accumulation of abdominal fat in both women (Davis, Twamley, Hamilton, & Swan, 1999; Epel et al., 2000; Moyer et al., 1994) and men (Rosmond, Dallman, & Björntorp, 1998; Seidell et al., 1991). Taken together, these studies support the hypothesis that, stress induced cortisol secretion is associated with the accumulation of abdominal fat and that abdominal fat is linked to health outcomes.

While a physiological response to stress has been implicated in the accumulation of abdominal fat, less is known about what specific psychosocial stressors are related to central adiposity. Research involving the association between psychosocial stressors and abdominal fat has increased in recent years. This dissertation extends the research on the relationship between stress and central adiposity. It focuses on psychosocial stressors connected to both social and economic well-being which have been associated with central adiposity, such as status strains (Elliott, 2000; L. Pearlin, Schieman, Fazio, & Meersman, 2005; L. I. Pearlin, 1999). Low income urban populations are particularly vulnerable to status strains compared to higher income populations and populations with higher positions of status by race/ethnicity and gender (Pearlin, Schieman et al. 2005). The following paragraphs will review the literature examining these associations status strains and obesity.

#### Perceived discrimination and obesity

Studies examining the relationship between perceived discrimination and central adiposity have produced inconsistent results. A few cross-sectional studies have found an association between perceived discrimination and central adiposity that varied by race and gender (Hickson et al., 2012; H. Hunte & Williams, 2009; Lewis, Kravitz, Janssen, & Powell, 2011; Vines et al., 2007). One study found that perceived racism is negatively associated to

waist-to-hip ratio among African American women (Vines et al., 2007). Another study found both a null association between perceived discrimination and waist circumference and a positive association between perceived discrimination and visceral fat again, among African American and White women (Lewis et al., 2011; Vines et al., 2007). Among Black men, one study found a positive association for everyday unfair treatment and subcutaneous fat (Hickson et al., 2012); one study found a null association between everyday unfair treatment and visceral fat (Hickson et al., 2012); and one study found a null association between everyday unfair treatment and waist circumference (H. Hunte & Williams, 2009). Among White men, one study found a positive association between everyday unfair treatment and waist circumference for ethnic White men (e.g. Polish) (H. Hunte & Williams, 2009). Two longitudinal studies have examined the relationship between perceived discrimination and central adiposity in samples of African American women and Caucasian women and men and both found a positive association (Cozier, Wise, Palmer, & Rosenberg, 2009; H. E. R. Hunte, 2011). However, these studies did not test whether these associations were modified by race/ethnicity or gender. There is some evidence that suggests that psychosocial stressors (e.g. everyday unfair treatment) are positively associated with central adiposity, and that these associations may vary by demographic characteristics. For example, the above study by Hunte and Williams (2009) reported a positive association between everyday unfair treatment and central adiposity among ethnic Whites, but not African Americans (H. Hunte & Williams, 2009). In all, the findings show inconsistent associations in cross sectional studies, while longitudinal studies are more consistent, although limited in number.

#### Family stress sand obesity

At least one longitudinal study found that family stress is associated with obesity in adults. Block and colleagues (2009) found that perceived constraints in life and strain in

relations with family were associated with greater weight gain among women, and lack of skill discretion or decision authority at work was associated with greater weight gain among men (Block, He, Zaslavsky, Ding, & Ayanian, 2009). However, the majority of studies tend to focus on the association between family stress and obesity in children and adolescents (Gibson et al., 2007; Koch et al., 2008; Lohman, Stewart, Gundersen, Garasky, & Eisenmann, 2009). Collectively, these studies have found that family stress is positively associated with obesity. However, since obese children tend to have obese parents these studies may provide clues to how family stress affects adults as well as children.

#### Friend/Neighbor Stress/Safety Stress and obesity

Although no studies have examined the relationship between friend/neighbor stress on central adiposity, at least one study has examined the influence of friend/neighbor stress on self-rated health (Steptoe and Fieldman, 2001). This study showed that neighborhood problems (e.g. friend/neighbor stress, safety stress) were associated with poor self-rated health (Steptoe and Fieldman, 2001). Additionally, other studies on neighborhood safety and obesity have been conducted with children and adolescents. These studies show a positive correlation between safety stress and obesity, primarily through its adverse effect on physical activity (Cecil-Karb & Grogan-Kaylor, 2009; Fish, Ettner, Ang, & Brown, 2010; Molnar, Gortmaker, Bull, & Buka, 2004; Singh, Siahpush, & Kogan, 2010). A few studies have found a positive relationship between safety stress and obesity, measured as BMI in adults. For example, a study of women with young children found that obesity was more likely in women who perceived their neighborhoods to be unsafe (Burdette, Wadden, & Whitaker, 2012). Another study of adults found that individuals who perceived their neighborhood as unsafe had a higher BMI than those

who did not, after controlling for sociodemographic characteristics and neighborhood SES (Fish et al., 2010). These findings are consistent with a recent review of disadvantaged environments and obesity that found that safety was positively correlated with obesity(Lovasi et al., 2009). This dissertation will extend this research by examining whether neighborhood safety stress is associated with central adiposity over time.

#### *Economic well-being and obesity*

Psychosocial stressors connected to economic strain have been associated with central adiposity, including financial strain (Block et al., 2009; Fowler-Brown et al., 2009; Gerace & George, 1996), job-related demands (Block et al., 2009), and occupational stress (Israel, Baker, Goldenhar, & Heaney, 1996). A few cross-sectional studies have found a positive association between financial strain and obesity. Schulz and colleagues (2008) found that chronic psychosocial stress (i.e. financial vulnerability) is associated with increased obesity risk in Non-Hispanic Blacks, Non-Hispanic Whites, and Hispanics (AJ Schulz et al., 2008). Munster and colleagues (2009) found a positive association between over-indebtedness and obesity in a crosssectional study of German adults (Münster, Rüger, Ochsmann, Letzel, & Toschke, 2009). Vankim et al. (2012) found a positive association between financial strain and unhealthy weight control (Vankim & Laska, 2012). At least one prospective study found a positive association between financial strain and obesity (Robert E, William J, Stephane, & George A, 2002). Other prospective studies found no significant association between financial strain and weight gain (Fowler-Brown et al., 2009; Lohman et al., 2009). All together, the majority of these studies suggest a positive association between financial strain and obesity risk.

Findings from some prospective studies suggest that associations between psychosocial stress and central adiposity may also vary by gender. For instance, in a prospective study of the Epidemiologic Diabetic Complications (EDC) study of adults aged 18 and older with insulin dependent diabetes, Lloyd and colleagues (1996) found a positive association between perceived stress and waist-to-hip ratio among women but not men (Lloyd, Wing, & Orchard, 1996). In addition, a 9-year prospective study found that weight gain was positively associated with jobrelated demands, difficulty paying bills, depression or generalized anxiety disorder among men and women (Block et al., 2009). In sum, these studies of perceived discrimination, neighborhood physical environment, neighborhood social environment, family stress, friend/neighbor stress and financial vulnerability provide examples that are consistent with a hypothesis that posits that chronic stressors are associated with central adiposity. However, the inconsistencies in this literature suggest the need for additional studies that acknowledge the social context under which these associations operate in. Therefore, this dissertation will examine associations between neighborhood economic composition, multiple indicators of psychosocial stress, and obesity. This research focus extends studies that have focused solely on diet and physical activity important pathways to obesity—by examining the broader contexts within which these behaviors emerge. Questions remain about how stressors from the social environment contribute to risk for central adiposity. There is a need for further research to disentangle the multiple aspects of environmental characteristics that are correlated with central adiposity. A better understanding of these environmental characteristics can help public health professionals target interventions more effectively and advocate for policy change to reduce stressors in environments where populations are at high risk for becoming obese.

#### **Organization of Dissertation**

The purpose of this dissertation is to help disentangle these complex sets of the associations between stress and central adiposity among urban adults. Building on this extant literature, in the remainder of the dissertation, I will examine three main research questions: 1) Is perceived discrimination (as an indicator of psychosocial stress) at baseline associated with changes in central adiposity over time, above and beyond neighborhood economic composition? (Chapter 2); 2) Are multiple indicators of psychosocial stress at baseline associated with changes in central adjoint over time, above and beyond neighborhood economic composition? (Chapter 3); and 3) Do aspects of the social environment modify the effectiveness of an intervention designed to promote physical activity, an important correlate of obesity? (Chapter 4). The fifth and final chapter synthesizes the findings of the three analytical chapters and provide conclusions and recommendations for interventions. The analyses will extend previous research by examining the associations between stress at baseline and changes in central adiposity over time in a multiethnic urban sample, above and beyond the effects of health behaviors and neighborhood economic composition; testing whether stress may modify effectiveness of interventions, as well as testing the modifying relationships of race and gender on the association between stress and central adiposity in a multiethnic urban community. Together, these findings will help inform interventions that aim to ameliorate excess risk for central adiposity, and associated health outcomes, among urban adults. In each chapter, I describe in greater detail why these questions are important to public health and how they will inform future interventions.

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#### Chapter 2

# Neighborhood economic composition, perceived discrimination and central adiposity, among a multiethnic low-to-moderate income urban population: Findings from longitudinal analyses.

#### Introduction

While national-level studies have consistently demonstrated racial disparities in obesity among non-Hispanic Blacks (NHB) relative to non-Hispanic Whites (NHW) in the United States (Denney, Krueger, Rogers, & Boardman, 2004; Flegal et al., 2010; Mokdad et al., 2003), some community-level studies have found that disparities in obesity and other health outcomes (e.g., hypertension) are attenuated (Thorpe Jr, Brandon, & LaVeist, 2008) and eliminated (Bleich, Thorpe, Sharif-Harris, Fesahazion, & LaVeist, 2010) when considering NHBs and NHWs of similar incomes or within similar social contexts. Social context theory proposes that racial segregation has shaped racial differences in physical and social environments that drive disparate health outcomes (LaVeist, 2005a and b); in turn, individuals living in dissimilar neighborhoods experience health disparities (Bleich et al., 2010). For example, Bleich and colleagues (2010) compared racial disparities in obesity among NHB and NHW women in the National Health Interview Study (NHIS) to women in Southwest Baltimore, Maryland in the Exploring Health Disparities in Integrated Communities-SWB (EHDIC-SWB) study. Compared to the NHIS sample where Black women were more likely to be obese than whites, the EHDIC-SWB sample showed that Blacks and Whites were equally likely to be obese among low-income women living in the same social context. In addition, Boardman and colleagues (2005) examined race differentials in obesity among Blacks and Whites in the National Health Interview Survey while controlling for neighborhood economic composition, and found that racial disparities were attenuated (Boardman et al., 2005). To further explore this area of research, this chapter

examines the association between social context and obesity among a Detroit-community multiethnic sample.

#### Neighborhood economic composition and exposure to environmental factors

In Detroit, environmental factors are driven by neighborhood economic composition (Schulz et al., 2012; Schulz et al., 2000), which influences obesity through limited access to healthy foods (Zenk et al., 2013) and places that are not conducive to physical activity (Kwarteng, Schulz, Mentz, Zenk, & Opperman, 2013; AJ Schulz et al., 2005), among other factors. The influence of neighborhood economic composition and indicators of the physical environment on obesity is well supported (Boardman et al., 2005; Diez Roux & Mair, 2010; Lovasi et al., 2009; Amy J Schulz et al., 2013); however, research that focuses on the influence of neighborhood economic composition and the social environment on obesity is scarce. Neighborhood economic composition may influence obesity by shaping exposure to social environments that are conducive to psychosocial stress that is perceived as harmful, threatening, or bothersome (Schulz et al., 2012; Lazarus, 1984). This stress process can lead to physiological responses that may cause metabolic changes that directly influence the distribution of fat in the body (M. F. Dallman et al., 2004), particularly in the internal, visceral adipose tissue regions (P Björntorp, 1987; P. Björntorp, 1997; Brydon, 2011; Koch et al., 2008; B.S. McEwen & Seeman, 2006; Wardle et al., 2011). In turn, excess visceral fat that accumulates in the abdominal region—called central adiposity<sup>2</sup>-- place individuals at higher risk for obesity.

<sup>&</sup>lt;sup>2</sup> Also called central obesity, intra-abdominal fat, or abdominal fat. Proxy measures for central adiposity include a high-risk waist circumference (i.e  $\ge 120$  cm in men and  $\ge 80$  cm in women) and high-risk waist-to-hip ratio ( $\ge 1.0$  in men and  $\ge 0.85$  in women), (Ding et al., 2004)

Central adiposity is a form of obesity that appears to be particularly harmful by placing individuals at higher risk for CVD and diabetes (Ding et al., 2004), and has been strongly linked to psychosocial stress (P Björntorp, 1987; P. Björntorp, 1997; Brydon, 2011; Koch et al., 2008; B.S. McEwen & Seeman, 2006; Wardle et al., 2011). Although there is mounting evidence regarding the association between psychosocial stress and central adiposity, less is known about specific indicators of psychosocial stress that are harmful to health. One indicator of psychosocial stress that has been found to be particularly harmful to health is perceived discrimination, with linkages to several adverse physical and mental health outcomes, including hypertension, depression, and cardiovascular disease (Albert et al., 2010; Albert & Williams, 2011; Hickson et al., 2012; H. Hunte & Williams, 2009; H. E. R. Hunte, 2011; Lewis, Aiello, Leurgans, Kelly, & Barnes, 2010; Amy J. Schulz et al., 2006; D. Williams & Mohammed, 2009; David R. Williams, Neighbors, & Jackson, 2008).

Recently, perceived discrimination has been associated with central adiposity; however, findings from these studies have been mixed (i.e. positive, negative, and null) by race and gender among NHBs, NHWs, Hispanics, and Asian Americans (Cozier et al., 2009; Gee, Ro, Gavin, & Takeuchi, 2008; Hickson et al., 2012; H. Hunte & Williams, 2009; H. E. R. Hunte, 2011; Lewis et al., 2011). Whereas these mixed findings may be due in part to variation across studies in the measurement and analysis of both perceived discrimination and central adiposity, with some studies focusing on perceptions of racism, including racial discrimination (Vines et al., 2007 & Cozier et al., 2009), the variant associations, such as by race and gender, within studies are less clear.
A possible explanation for inconsistencies may be due to varying neighborhood economic composition and social environments, where some environments are more conducive to obesity than others. For example, in comparing two community samples from Chicago, one study focused on the southside of Chicago with similarities in SES and social environments and found similar associations between perceived discrimination and central adiposity, among NHBs and NHWs (Lewis, 2011). More specifically, a cross-sectional analyses of the Study of Women Across the Nation (SWAN), found a positive association between everyday unfair treatment and visceral fat among African American and Caucasian women irrespective of race among women of similar SES and social environments (Lewis et al., 2011). The other Chicago sample included a cross-sectional analysis of a wider geographical area among individuals of dissimilar SES and found disparate outcomes (Hunte & Williams, 2009). In particular, Hunte & Williams (2009) found a positive association between both racial/ethnic discrimination and nonracial/nonethnic discrimination and high-risk waist circumference among ethnic NHWs (e.g. Irish, Italian, Jewish, and Polish) but not NHBs, who had lower SES than NHWs (H. Hunte & Williams, 2009). One limitation of these studies is their cross-sectional design and inability to disentangle the longitudinal sequence of effects. In contrast, a longitudinal study that examined the relationship between perceived discrimination and discrimination among both NHBs and NHWs included a national sample of NHBs and NHWs of dissimilar SES and found differential associations between perceived discrimination and waist circumference by race and gender, with positive associations between increased racial discrimination and waist circumference among NHB women only (Cunningham et al., 2013). However, these studies did not include indicators of neighborhood characteristics that may influence exposure to varying social environments.

In sum, despite evidence that neighborhood factors are associated with both obesity and the stress process, studies on the association between perceived discrimination and obesity have not considered this evidence. Therefore, this chapter builds on, and substantially extends, the extant literature by examining multilevel associations between perceived discrimination and increased obesity within the context of neighborhood economic composition.

# **Research Questions:**

2.1) Do rates of central adiposity at baseline vary among Non-Hispanic Blacks, Non-HispanicWhites, and Hispanics in a Detroit community sample?

2.2) Is neighborhood economic composition at baseline associated with change in central adiposity over time?

2.3) Is perceived discrimination at baseline associated with change in central adiposity over time, after controlling for neighborhood economic composition?

# **Methodology:**

### <u>Sample</u>

This chapter used a prospective 6-year follow up drawing upon two data sources: The Healthy Environments Partnership (HEP) Wave I 2002-2003 and Wave II 2008, community surveys.

The HEP Wave 1 Community Survey was conducted in 2002-2003 with a stratified two-stage probability sample of occupied housing units in Detroit. The survey was designed for 1000 completed interviews of NHB, NHW, and Hispanic adults aged  $\geq$ 25 years residing in Detroit. At each household unit, a listing was completed of eligible residents, and one eligible adult was

selected randomly for inclusion in the study. Of the 2517 housing units in the initial sample, 1297 were invalid (e.g. vacant), unable to be screened after repeated attempts (i.e.12+ attempts), or contained no eligible respondent. The final sample consisted of 919 face-to-face interviews: interviews were completed with 75% of households in which an eligible respondent was identified (919 of 1220), 55% of households with a known or potential respondent (919 of 1663), and 90% of households in which an eligible respondent was contacted (919 of 1027) (Schulz, 2005).

The 2008 HEP Wave II community survey followed up on the 2002 survey and included re-interviews with 219 of the 2002 survey respondents, as well as new residents of housing units included in the 2002 sample (n = 241). This chapter analyzed this sample of 460 face-to-face interviews.

# <u>Measures</u>

The *dependent variable* was a continuous measure of waist circumference in centimeters for 2002 and 2008.

*Individual level independent variables*. The time-varying *independent variable* was a continuous measure of everyday unfair treatment (Williams et al., 1997), constructed as a mean scale of five items from 1-5 (e.g. how often are you "treated with less courtesy or respect than other people," in the previous 12 months) (range 1=never, 5= always) (Cronbach's alpha 0.77).

*Individual level control variables.* **Behavioral control variables** included alcohol intake (Block et al., 1994), which was constructed by mean daily frequency intake of alcoholic beverages reported on the modified Block 98 questionnaire: beer, red wine, wine, and liquor. For the four alcoholic beverages, reported intake frequencies, ranging from never to everyday, were converted into the number of drinks per month ranging from 0 to 300. This value was then converted to a binary variable that represents individuals with less than 1 drink per month=0 and individuals with 1 or more drinks per month=1. Current smoker (Frazier et al., 1992) (e.g. "Do you currently smoke cigarettes") was constructed by using the self-report of whether the individual never smoked, currently smoked, or formerly smoked (1=current, 0=never smoked, or formerly smoked). The healthy eating index (Kennedy et al., 1994) was constructed by taking the sum of mean daily frequency of intake of foods that consist of grains, meat, milk, vegetables, fruit, fat, saturated fat, sodium, and cholesterol reported on the modified block 98 semiquantitative food frequency questionnaire. For the ten food categories, reported intake frequencies, ranging from never to six or more times per day, were converted to daily frequencies using the following weights: "never or less than once a month" =0, "1-3 times a month" = 0.1, "4-6 times a month"=5/7, "1 time every day" = 1, "2-3 times every day"=3, "4-5 times every day"=5, and "6 or more times every day"=6. The study used a modified version of the HEI and included a value of the ten items was summed. The final modified-HEI ranged from 0-100, with a higher number representing healthier consumption of foods. Metabolic minutes (METs) was constructed as a continuous measure of minutes the individual reported being engaged in physical activity. Physical activity was captured by asking how many days and the amount of time an individual reported moderate-intensity activities (vacuuming, gardening, or anything else that causes small increases in breathing or heart rate) or vigorous activities (such as fast walking, running, dancing, or participating in strenuous sports that cause large increases in breathing or heart rate) in a usual week for at least 10 minutes at a time (Ainsworth et al., 2003).

*Time-varying controls* consisted of a dummy variable representing time (0=2002, 1=2008), age (years), and BMI ( $\geq 30$ ). *Time-invariant controls* consisted of gender (1=female,

0=male); self-reported race/ethnicity; education (<12 years, 12 years,  $\geq$ 12 years=referent); the poverty-to-income categorization (U.S. Census Bureau, 2000) was calculated by dividing the household income by the federal poverty threshold for the related family size (Poverty-to-income categorization $\leq$ 1, 1=Below poverty, 0=Above poverty); marital status (1=married, 0=single, widowed, or divorced); car (1= owns or leases car, 0= no car); and home ownership (1=owns home, 0= does not own home).

*Neighborhood level independent variables*. The *time-invariant independent variable* neighborhood economic composition (i.e. percent poverty) was derived from the 2000 census and was a continuous measure of the percent of households with incomes below the poverty line.

# <u>Analysis</u>

Weighted 3-level hierarchical linear regression models for a continuous outcome were estimated to account for both the longitudinal and nested structure of the data. Individuals who reported that they were pregnant or breastfeeding (n=23), and those missing a measure for waist circumference (n=60) were removed from the analysis. In addition, since HLM cannot handle unbalanced data for the time varying measures, individual (level-2) and neighborhood (level-3) levels with missing data were removed from the analysis (n= 5). The final models included the remaining 157 repeated measures (level 1), nested in 386 individuals (level 2), and 64 census block groups (level 3).

To examine the first research question, whether rates of central adiposity varied among NHBs, NHWs, and Hispanics at baseline in a Detroit community sample, chi-squared analysis was conducted to examine racial differences. Given that NHB women typically have the highest rates of central adiposity, additional analyses were conducted to examine differences in waist

circumferences by race and gender. I then examined whether racial differences were apparent after accounting for other demographic variables by running a linear multilevel regression model for a continuous outcome, controlling for time, age, gender, education, poverty to income ratio, marital status, car ownership, and home ownership (Model 2.1). Next, I adjusted the variable for BMI in order to differentiate between racial differences due to BMI and those due to waist circumference (Model 2.2).

I then examined whether neighborhood percent poverty was significantly associated with waist circumference. The neighborhood percent poverty measure was added to the level-3 intercept and adjusted for time, age, gender, race, education, poverty-to-income categorization, marital status, car ownership, home ownership, and (Model 2.3).

Next I added everyday unfair treatment to the model to examine whether everyday unfair treatment was significantly associated with waist circumference, above and beyond neighborhood economic composition. Everyday unfair treatment was added to the level-2 intercept controlling for time, age, gender, race, education, poverty-to-income categorization, marital status, car ownership, home ownership, and BMI . Last, I added health-related behaviors (i.e. alcohol, smoking, HEI, and METs) to examine whether the association of everyday unfair treatment on waist circumference remained robust after accounting for differences in alcohol intake, current smoking, HEI, and METs (Model 2.4).

Model 2.1: WCIR=  $\gamma_{000}$ +  $\gamma_{100}$ TIME1+  $\gamma_{200}$ (AGE<sub>tij</sub>-AGE...)+ $\gamma_{010}$  FEMALE<sub>ij</sub>+  $\gamma_{020}$ HISPANIC+  $\gamma_{030}$ WHITE<sub>ij</sub> +  $\gamma_{040}$ OTHER<sub>ij</sub>+  $\gamma_{050}$ LESS12+  $\gamma_{060}$ YEARS12+ $\gamma_{070}$ (PIR<sub>ij</sub>-PIR..)+  $\gamma_{080}$ MARRIED +  $\gamma_{090}$ CAR OWNERSHIP+ $\gamma_{0100}$ HOMEOWNERSHIP +  $\gamma_{0110}$ ALCOHOL+  $\gamma_{0120}$ SMOKING+  $\gamma_{0130}$ HEI+  $\gamma_{0140}$ METs+ e

# Results

Data on waist circumference were examined for 386 participants. Descriptive characteristics of the sample are presented in Table 2.1. The mean waist circumference was 98.9 cm. The mean level of everyday unfair treatment was 1.7.

Table 2.2 shows results of the chi-squared analyses. The results indicate no significant differences between mean high-risk waist circumference (i.e.  $\geq$ 88 cm for women,  $\geq$ 102cm for men) between NHBs and NHWs (X<sup>2</sup>=0.09) and NHBs and Hispanics (X<sup>2</sup>=0.08). To account for differences by gender, additional analyses were examined by race and gender. There were no significant differences between NHB women and NHW women (X<sup>2</sup>=0.08), NHB women and Hispanic women (X<sup>2</sup>=0.09), NHB men and NHW men (X<sup>2</sup>=0.87), or NHB men and Hispanic men (X<sup>2</sup>=0.75).

To assess the patterns described above, visible in the unadjusted data, I ran multiple regression models to adjust for relevant demographic characteristics. Results are presented in Table 2.3 and showed no difference between NHBs, NHWs, and Hispanics after controlling for age, time, gender, marital status, education, income, car ownership, homeownership. However, after adjusting for BMI, there was a significant difference between NHBs and Hispanics.

Table 2.3, Model 2.3 shows results for the second research question, "Is neighborhood economic composition at baseline associated with change in central adiposity over time?" I found that when controlling for age, gender, race, education, poverty-to-income categorization, marital status, car ownership, home ownership, and BMI, neighborhood economic composition was positively associated with changes in waist circumference over time ( $\beta$ = 0.1, p=0.03). Specifically, neighborhoods with higher levels of neighborhood percent poverty showed greater increases in obesity, compared to those with lower concentrations of poverty.

Table 2.3, Model 2.4 show findings for the last research question, "Is perceived discrimination associated with changes in waist circumference, after controlling for neighborhood economic composition?" The results show that everyday unfair treatment at baseline is associated with central adiposity independent of neighborhood economic composition. Everyday unfair treatment at baseline is positively associated with changes in central adiposity over time ( $\beta$ = 1.9, p=.01) above and beyond the effect of neighborhood economic categorization, marital status, car ownership, home ownership, BMI, alcohol intake, current smoking, METs, and HEI (Model 2.4).

Additional models were tested to adjust for time-varying covariates, such as everyday unfair treatment, METs, smoking, and alcohol intake at baseline and follow-up; these covariates were not statistically significant in the models. Moreover, their inclusion did not affect the fit of the models (results not shown). Data were not available to measure change in HEI.

# Discussion

This analysis yielded three main findings. The first is that central adiposity is not significantly different between NHBs, NHWs, and Hispanics after accounting for time, age, gender, education, poverty-to-income categorization, marital status, car ownership, and home ownership. After adjusting for BMI, significant differences in central adiposity between NHBs and Hispanics were found. Second, there is evidence to support that neighborhood economic composition is associated with increases in central adiposity over time. Third, everyday unfair

treatment is associated with change in central adiposity, independent of neighborhood economic composition. I discuss each of these findings in greater detail below.

### Neighborhood economic composition and obesity

The finding that neighborhood economic composition is positively associated with increases in central adiposity is partially consistent with other studies (Kahn et al., 1998). For example, Kahn and colleagues (1998) examined the association between income inequality and increases in central adiposity among men and women in geographic regions with high income inequality, and found that men from states with high income inequality experienced greater increase in central adiposity (Kahn et al., 1998). My finding suggests that neighborhood economic composition plays an important role in increased risk for central adiposity over time. Others have reported associations between neighborhood economic composition and access to healthy foods (Zenk et al., 2008.) and places that are not conducive to physical activity (Kwarteng et al., 2013; Schulz, 2005). Furthermore, there is some evidence that neighborhood economic composition is associated with exposure to social environments that are conducive to psychosocial stress that is perceived as harmful, threatening, or bothersome (Schulz et al., 2012; Lazarus, 1984).

# *Everyday unfair treatment and central adiposity*

My study supports the theory that social environment shapes obesity risk through the finding of an association between everyday unfair treatment and increased central adiposity in a multiethnic sample, above and beyond the effect of neighborhood economic composition. These results join findings reported by two other longitudinal studies that found a positive association

between perceived discrimination and obesity (Cozier et al. 2009; Hunte, 2012). However, my findings are only partially consistent with Cunningham and colleagues (2013), who examined the relationship between racial discrimination and increased central adiposity in a national cohort, and found a positive association for racial discrimination and increased central adiposity among Black women, but not for Black men, or White women and men. Such findings are likely due to the exclusion of measures of social context that are correlated with central adiposity (Cunningham, 2013).

# Limitations/Conclusions

This chapter has several limitations. First, this study only includes two measures of the social environment (i.e. neighborhood economic composition and everyday unfair treatment). Therefore I may have unmeasured aspects of the social environment that were not accounted for. Further, given that income may also be associated with physical characteristics of the environment, such as access to healthy foods, I may have overlapping constructs of the neighborhood physical and social environment that were not accounted for. Third, a large proportion of my sample was overweight and obese at baseline, which may have biased my findings, given that obese individuals may be more likely to increase in weight over their lifecourse than normal weight individuals. To minimize this bias, I controlled for BMI at baseline and follow-up and still found evidence that both perceived discrimination and neighborhood poverty level were associated with increases in central adiposity. Moreover, since a third of my sample was obese at baseline, it was important that I was able to disentangle the influence of BMI on waist circumference and the influence of weight-based discrimination on increased waist circumference. Lastly, this study did not include other indicators of psychosocial

stress (such as family stress and perceptions of neighborhood physical and social environment) that may be associated with obesity in my population. The next chapter will examine the influence of other indicators of psychosocial stress and increases in central adiposity among this sample.

In conclusion, I found that neighborhood economic composition is positively associated with increased central adiposity, with individuals who live in areas with the greatest percentage of poverty having the greatest increases over time. This finding may show that poverty is an indicator of psychosocial stress that contributes to excess central adiposity. Similarly, after controlling for neighborhood economic composition, I found that perceived discrimination was positively associated with increases in central adiposity among NHBs, NHWs, and Hispanics, which shows that perceived discrimination influences individuals who live in neighborhoods of varying percent poverty. This study's finding that increases in central adiposity are not significantly different between NHBs and NHWs underscores the importance of considering social context when designing interventions to address obesity. Understanding ways in which social context creates or eliminates disparate outcomes can inform interventions on ways to create effective interventions in urban populations.

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(Thompson et al., 2003)

Individual (Levels 1 and 2)	Mean	Percent	Range
Age	47.6±0.9		25.0-96.0
Female		52%	
White		17%	
Black		55%	
Hispanic		26%	
Less than High School		40%	
High School		24%	
Beyond High School		35%	
Below poverty		38%	
Married		27%	
Car ownership		66%	
Home ownership		51%	
Alcohol use		47%	
Currently Smoking		39%	
Healthy Eating Index	63.9±0.7		0.0-90.0
METs (n=314)	$1.0\pm0.1$		0.0-4.2
Waist Circumference	$98.9 \pm 0.9$		64.0-153.8
Body Mass Index	31.2±7.3		
Everyday Unfair Treatment	1.7±0.0		0.0-4.2
Block Group (Level 3)			
Percent Poverty	31.9±11.2		8.0-54.0

# Table 2.1 Weighted Descriptive Characteristics

	NHB	NHW	Hispanic		$\mathbf{X}^2$	
Sample Size No.	219	71	90	NHB vs	NHB vs	
				NHW	Н	
High WC (%, SE)	64% (.05)	52% (.07)	53% (.07)	0.09	0.08	
Obese: High Risk						
Waist Circumference						
Men (≥102cm)	48%	46%	44%	0.87	0.75	
Women (≥88 cm)	76%	60%	64%	0.08	0.09	

Table 2.2 Chi-squared analysis of high risk waist circumference by race and gender

NHB: Non-Hispanic Black; NHW: Non-Hispanic White

# Table 2.3 Waist Circumference regressed on race, neighborhood economic composition and everyday unfair treatment

		<u>Model 2.1</u> <u>Model 2.2</u>			<u>Model 2.3</u>		Model 2.4		<u>Model 2.5</u>		
	N=386	В	SE	В	SE	β	SE	В	SE	β	SE
				106.8**							
	Intercept	101.8***	2.6	*	1.8	106.7***	1.7	106.5***	1.7	106.0***	1.7
Level 2 (BG)	Percent Poverty					0.1**	0.0	0.1**	0.0	0.1**	0.04
				-							
Level 1 (individual)	Time	-3.4***	0.7	3.09***	0.6	-3.1***	0.6	-3.0***	0.6	-3.1***	0.6
Baseline and follow-up	Age	0.1	0.1	0.15***	0.0	0.2***	0.0	0.2***	0.0	0.2***	0.04
	BMI			1.80***	0.1	1.8***	0.1	1.8***	0.1	1.8***	0.08
Baseline Only				-							
	Female	-1.7	1.8	6.99***	1.1	-6.9***	1.1	-6.7***	1.1	-6.5***	1.0
	Hispanic	-3.0	1.9	-2.59**	1.3	-2.7**	1.2	-2.8**	1.2	-2.7**	1.3
	White	-2.7	2.2	-1.0	1.6	-0.9	1.6	-1.0	1.6	-1.2	1.7
	Less than HS	2.0	2.2	0.8	1.2	0.7	1.2	0.7	1.2	0.6	1.2
	High School	2.1	2.8	1.1	1.5	1.0	1.4	1.1	1.4	1.0	1.4
	PIR	-1.3**	0.6	-0.6	0.4	-0.6	0.4	-0.6	0.4	-0.5	0.4
	Married	-0.3	2.1	-1.3	1.5	-1.1	1.5	-1.1	1.4	-0.7	1.4
	Car Owner	2.8	2.1	-0.1	1.2	-0.0	1.2	-0.1	1.2	0.4	1.3
	Homeowner	-0.2	2.5	1.2	1.4	1.2	1.4	1.4	1.4	1.5	1.5
	Everyday Unfair										
	Treatment							1.5*	0.8	1.6	0.8
	Alcohol use									-1.0	0.9
	Currently Smoking									1.2	1.1
	Healthy Eating Index									-1.0^	0.1
	METs									-0.19	0.72
	sigma square	2	22.93726	18	3.10056		18.08086	18	3.10216	18.037	
	tau pi	23	33.05157	65	5.14689		65.52835	65	5.10673	64.112	
	tau beta		0.07897	4	.01535		2.83003	2	2.17285	1.6155	

^<u><0.10</u>,\*0.05,\*\*<0.05, \*\*\*<u><</u>0.01

# **Chapter 3**

# The influence of multiple indicators of stress on obesity: Does exposure to multiple indicators of psychosocial stress account for changes in central adiposity over time among residents of Detroit?

Non-Hispanic Blacks (NHBs), Hispanics, and low-income non-Hispanic Whites (NHWs) in the United States are disproportionately at risk of cardiovascular disease (CVD), diabetes, and some cancers (Kumanyika et al., 2008). While research suggests that this disproportionate risk is driven, at least in part, by higher rates of obesity among these racial groups (Kumanyika et al., 2008), the rates of obesity often vary by neighborhood characteristics, such as neighborhood economic composition. For example, neighborhoods with a higher percentage of poverty often have the highest rates of obesity (Boardman et al., 2005). Evidence is building that neighborhood economic composition contributes to obesity through, for example, shaping access to healthy foods (Zenk et al, 2012) and places that are conducive to physical activity (Kwarteng et al., 2013; Schulz, 2005). However, these indicators of the physical environment do not fully explain excess risk in obesity by race and income, which has provoked interest in factors of the social environment that may influence obesity.

Neighborhood economic composition may influence obesity by patterning exposure to social environments that are conducive to psychosocial factors that are perceived as harmful, threatening, or bothersome, resulting in stress (Schulz et al., 2012; Lazarus, 1984). This stress process can lead to physiological responses that may cause metabolic changes that directly

influence the distribution of fat in the body (M. F. Dallman et al., 2004), particularly in the internal, visceral adipose tissue regions (P Björntorp, 1987; P. Björntorp, 1997; Brydon, 2011; Koch et al., 2008; B.S. McEwen & Seeman, 2006; Wardle et al., 2011). In turn, excess visceral fat that accumulates in the abdominal region—called central adiposity<sup>3</sup>-- places individuals at higher risk for obesity.

In Chapter 2, I found that everyday unfair treatment is positively associated with increases in central adiposity over time, independent of neighborhood economic composition. This begs the question of whether disadvantaged groups are at greater exposure to other indicators of psychosocial stress have a greater influence on the patterning of obesity. It has been hypothesized that the response to psychosocial stress may be shaped by one's social environment (L. I. Pearlin, 1989; D.R. Williams & House, 1991; D.R. Williams, Spencer, & Jackson, 1999). Psychosocial stress is embedded in social structures, roles, and relationships that persist over time (L. I. Pearlin, 1999). As described in Chapter 1, three classes of chronic stressors have been identified: status strains, role strains, and contextual strains (L. I. Pearlin, 1999). This chapter will focus on contextual strains such as neighborhood physical environment, neighborhood social environment, family stress, friend/neighbor stress, safety stress, and financial vulnerability because these stressors are common among my sample population.

Relatively few studies have examined the effect of multiple indicators of psychosocial stress on obesity. Three prospective studies have examined the relationship between stress and

<sup>&</sup>lt;sup>3</sup> Also called central obesity, intra-abdominal fat, or abdominal fat. Proxy measures for central adiposity include a high-risk waist circumference (i.e  $\ge 120$  cm in men and  $\ge 80$  cm in women) and high waist-to-hip ratio ( $\ge 1.0$  in men and  $\ge 0.85$  in women), (Ding et al., 2004).

weight change. In a prospective study, Lloyd and colleagues (1996) found a positive cross sectional association at baseline between stress and waist-to-hip ratio among women, but found no association between stress and change in waist-to-hip ratio over a two year period in either men or women when controlling for age, education, and BMI. In contrast, a thirteen year prospective study by Fowler-Brown and colleagues (2009) in a sample of Black women found that when controlling for age, stress at baseline, smoking, education, occupation, and financial strain, higher levels of perceived stress at baseline predicted a higher percentage increase in BMI over time (Fowler-Brown et al. 2009). Similarly, a prospective study of non-White Hispanic, White Hispanic<sup>4</sup>, and Black non-Hispanic male fire fighters and paramedics found a positive association between financial insecurity at baseline and weight change over a 7-year period, when controlling for age, race, smoking, speed of eating, marital status, and percentage of ideal weight (Gerace & George, 1996). Finally, a 9-year prospective study that included both Black, White, Hispanic, and Other women and men found that weight gain was associated with jobrelated demands, perceived constraints in life, strain in relations with family, and difficulty paying bills among women (Block et al., 2009). In this sample, job-related demands, lack of skill discretion, lack of decision authority, and difficulty paying bills were associated with weight gain among men (Block et al., 2009). Together, these findings generally suggest that exposure to stress at baseline is associated with increases in obesity over time. However, none of these studies have examined the influence of a cumulative effect of multiple indicators of pscychosocial stress on central adiposity over time, in order to test the influence of chronic stress on central adiposity. Further, none of these studies considered the mediating effect of neighborhood economic composition and cumulative stress on central adiposity.

<sup>&</sup>lt;sup>4</sup> The racial and ethnic categories reported here are those reported in the original manuscript.

To address this gap in the literature, this chapter examines the influence of neighborhood economic composition and both multiple indicators of psychosocial stress and cumulative stress on increases in obesity over time. Given that individuals who live in urban environments are often exposed to multiple indicators of psychosocial stress (Geronimus, 2000, Schulz & Northridge, 2004), and are at disproportionate risk of chronic conditions associated with obesity (Wang et al., 2011), these sources of stress may shape trends in central adiposity over time. This chapter will help me to examine pathways and factors that may be associated with obesity risk in addition to commonly known behavioral indicators (i.e. diet, physical activity). Knowledge of these factors can help to identify multiple indicators of psychosocial stress that drive excess risk in central adiposity among NHBs, Hispanics, and low-income NHWs. This research may help facilitate public health advocacy for more equitable environments that decrease stress exposure that stem from inequitable environments and lessen the risk of obesity in urban populations. Moreover, these findings can identify those most vulnerable to the adverse effects of stress on central adiposity, which can be used to develop and tailor interventions to lessen disproportionate risk among vulnerable populations.

# **Research Questions:**

3.1) Are multiple indicators of psychosocial stress associated with increases in central adiposity over time?

3.2) Is the chronic stress index associated with increases in central adiposity over time?3.3) Do multiple indicators of psychosocial stress mediate the relationship between neighborhood economic composition and increases in central adiposity over time?

3.4) Does the cumulative stress index mediate the relationship between neighborhood economic composition and increases in central adiposity over time?

### **Methodology**

# <u>Sample</u>

This chapter used a prospective 6-year follow up drawing upon two data sources: The Healthy Environments Partnership (HEP) Wave I 2002-2003 and Wave II 2008, community surveys.

The Healthy Environments Partnership (HEP) Wave 1 Community Survey was conducted in 2002-2003 with a stratified two-stage probability sample of occupied housing units in Detroit.

The HEP community survey was designed for 1000 completed interviews of NHB, Hispanic and NHW adults aged  $\geq 25$  years residing in Detroit. At each household unit, a listing was completed of eligible residents and one eligible adult was selected randomly for inclusion in the study. Of the 2517 housing units in the initial sample, 1297 were invalid (e.g. vacant), unable to be screened after repeated attempts (i.e.12+ attempts) or contained no eligible respondent. The final sample consisted of 919 face-to-face interviews: interviews were completed with 75% of households in which an eligible respondent was identified (919 of 1220), 55% of households with a known or potential respondent (919 of 1663) and 90% of households in which an eligible respondent was contacted (919 of 1027) (Schulz, 2005. The 2008 HEP community survey followed up on a 2002 and included 2002 survey respondents (n = 219) as well as new residents of housing units included in the 2002 sample (n = 241). This chapter analyzed this subsample of 460 face-to-face interviews.

### <u>Measures</u>

The *dependent variable* is waist circumference in centimeters in 2002 and 2008. Independent variables include measures from 2002: Neighborhood physical environment was the mean scale of seven items assessing agreement with the following statements: houses in my neighborhood are generally well maintained (reverse coded); there is heavy car or truck traffic in my neighborhood; my neighborhood has a lot of vacant lots or vacant houses; there is air pollution like diesel from trucks or pollution from factories or incinerators in my neighborhood; streets, sidewalks, and vacant lots in my neighborhood are kept clean of litter and dumping (reverse coded); there is a lot of noise from cars, motorcycles, music, neighbors, or airplanes in my neighborhood; and there is contaminated land in my neighborhood. Response categories ranged from 1=strongly agree to 5=strongly disagree, with a high score indicating a more positive ranking of neighborhood environment (Cronbach's alpha =0.69). Neighborhood social environment was the mean scale of six items assessing the frequency with which the respondent indicated that each of the following were a problem in their neighborhood: Gang activity; drug dealing or drug dealers; gunfire or shootings; prostitution; loitering or hanging around; theft, vandalism, or arson. Response categories ranged from 5= never to 1= always, with a high score indicating a more positive ranking of the neighborhood social environment (Cronbach's alpha=0.83). Family stress is mean scale of three items (e.g. Did problems experienced by a parent or other relative put extra burden on you (Cronbach's alpha=0.63) (A. Schulz, Parker, Israel, & Fisher, 2001). Friend/neighbor stress is a single item asking "Did you have problems in relationships with friends and neighbors?" Safety stress is a mean scale of three items asking the respondent whether they were worried about personal safety in their home or neighborhood (e.g. "How often did you worry about your safety in your home?"). An index of financial

vulnerability was assessed using two items assessing financial strain (e.g. "Difficulty in meeting the family's monthly payments") (mean= 3.1, range 1=less financial vulnerability, 5=more financial vulnerability) (James et al., 1992). In addition, since economically-disadvantaged individuals are often exposed to more than one type of stress simultaneously, a composite measure of all of these stressors was created as an index of cumulative stress. The index of cumulative stress was created by dichotomizing the scores of each indicator of psychosocial stress into 0 for low values and 1 for high values, and then the scores were summed for a composite measure.

Behavioral control variables include measures from 2002: alcohol use was constructed by mean daily frequency intake of alcoholic beverages reported on the modified Block 98 questionnaire: beer, red wine, wine, and liquor (Block et al., 1994). For the four alcoholic beverages, reported intake frequencies, ranging from never to everyday, were converted into the number of drinks per month ranging from 0 to 300. Due to the skewed nature of this variable, with the majority of participants reporting 0 or 1, this value was then converted to a binary variable that represents individual with less than 1 drink per month =0 and individuals with 1 or more drinks per month=1. Current smoker (e.g. "Do you currently smoke cigarettes") was constructed by using the self-report of whether the individual never smoked, currently smoked, or formerly smoked (1=current) (Frazier et al., 1992). The healthy eating index was constructed by taking the sum of mean daily frequency of intake of foods that consist of grains, meat, milk, vegetables, fruit, fat, saturated fat, sodium, and cholesterol reported on the modified block 98 semi-quantitative food frequency questionnaire (Kennedy et al., 1994). For the ten food categories, reported intake frequencies, ranging from never to six or more times per day, were converted to daily frequencies using the following weights: "never or less than once a month"

=0, "1-3 times a month" = 0.1, "4-6 times a month"=5/7, "1 time every day" = 1, "2-3 times every day"=3, "4-5 times every day"=5, and "6 or more times every day"=6. The value of the ten items was summed. The final HEI ranged from 0-100, with a higher number representing greater consumption of healthy foods. Metabolic minutes (henceforth MET), constructed as a continuous measure of minutes the individual reported being engaged in physical activity. Physical activity was captured by asking how many days and the amount of time an individual reported moderate-intensity activities (vacuuming, gardening, or anything else that causes small increases in breathing or heart rate) or vigorous activities (such as fast walking, running, dancing, or participating in strenuous sports that cause large increases in breathing or heart rate) in a usual week for at least 10 minutes at a time (Ainsworth et al., 2003).

*Time-varying controls* consisted of age (years) and BMI ( $\geq$ 30). *Time-invariant controls* consisted of gender (1=female); self-reported race/ethnicity; marital status (1=married); education (<12 years,  $\geq$ 12 years); the poverty-to-income categorization (U.S. Census Bureau, 2000) was calculated by dividing the household income by the federal poverty threshold for the related family size (Poverty-to-income categorization<1); car (1= owns or leases car); and home ownership (1=owns home).

*Neighborhood level independent variables.* The time-invariant *independent variable* neighborhood economic composition (i.e. percent poverty) was derived from the 2000 census and was a continuous measure of the percent of households with incomes below the poverty line.

#### Analysis

Weighted 3-level hierarchical linear regression models for a continuous outcome were estimated to account for both the longitudinal and nested structure of the data. Individuals who reported that they were pregnant or breastfeeding (n=23), and those missing a measure for waist circumference (n=60) were removed from the analysis. In addition, since HLM can only handle unbalanced data for the time varying measures, individuals (level-2) and neighborhood (level-3) levels with missing data were removed from the analysis (n= 5). The final models included the remaining 157 repeated measures (level 1), nested in 386 individuals (level 2); and 64 census block groups (level 3).

To examine the first research question, whether multiple indicators of psychosocial stress (i.e. neighborhood physical environment, neighborhood social environment, family stress, friend/neighbor stress, safety stress, and financial vulnerability) are associated with increases in central adiposity over time, each individual indicator of psychosocial stress was added to the level 2 intercept. Next, an indicator for time was added to the level-1 intercept to account for change in central adiposity over time, controlling for age, gender, race, marital status, education, poverty-to-income categorization, car ownership, home ownership (Model 3.1).

Models 3.1-3.7 tested the hypothesis that each indicator of psychosocial stress will be independently associated with central adiposity over time: neighborhood physical environment (NePhEn), neighborhood social environment (NeSoEn), family stress (FAMILY),

friend/neighbor stress (FRIEND), safety stress (SAFETY), and financial vulnerability (FiVul).

Model 3.1: WCIR=  $\gamma_{000}$ +  $\gamma_{100}$ (POVPER<sub>j</sub> - POVPER ...)+TIME +  $\gamma_{100}$ (AGE<sub>tij</sub>-AGE...)+ (BMI<sub>tij</sub>-BMI...)+ $\gamma_{010}$  FEMALE<sub>ij</sub>+  $\gamma_{020}$ HISPANIC+  $\gamma_{030}$ WHITE<sub>ij</sub>+  $\gamma_{040}$ OTHER<sub>ij</sub>+  $\gamma_{070}$ MARRIED+  $\gamma_{060}$ EDUCATION+  $\gamma_{050}$ RATIO OF HOUSEHOLD INCOME TO POVERTY+  $\gamma_{090}$ CAR +  $\gamma_{080}$ HOMEOWNER + e

Model 3.2: Model 3.1+ NePhEn Model 3.3: Model 3.1+NeSoEn Model 3.4: Model 3.1+ FAMILY Model 3.5: Model 3.1+FRIEND Model 3.6: Model 3.1+SAFETY Model 3.7: Model 3.1+FiVul Model 3.8: Model 3.1 + CUMULATIVE STRESS Model 3.8 tested the hypothesis that an index of cumulative stress (CUMULATIVE

STRESS) in 2002 is associated with an increase in waist circumference in 2008, above and beyond basic demographics (i.e. age, gender, race, marital status, income, education, car ownership, homeownership, BMI, physical activity, diet, smoking, and alcohol use.

Model 3.8: WAIST CIRCUMFERENCE=  $\beta_0 + \beta_1 AGE + \beta_2 GENDER + \beta_3 WHITE + \beta_4 HISPANIC + \beta_5 OTHER + \beta_6 RATIO OF HOUSEHOLD INCOME TO POVERTY + \beta_7 EDUCATION + \beta_8 HOMEOWNER + \beta_9 CAR + \beta_{10} MARRIED + \beta_{11} PHYSICAL ACTIVITY + \beta_{12} HEI + \beta_{13} SMOKING + \beta_{14} ALCOHOL + \beta_{15} CUMULATIVE STRESS + e$ 

Model 3.10: Model 3.15

Finally, to analyze the mediating effect of these indicators of psychosocial stress on the association between neighborhood percent poverty and central adiposity, the Freedman and Schatzkin (1992), was used to test the difference in point estimates for neighborhood percent poverty with and without the mediator. The formula can be written as follows:

$$t_{N-2} = \frac{c - c'}{\sqrt{\sigma_c^2 - \sigma_c^2 - 2\sigma_c \sigma_{c'\sqrt{1 - \rho_{xm}}}}}$$

where  $\rho_{xm}$  refers to the correlation between the independent variable X and the mediator M.

# **Results**

The sample demographics are illustrated in Table 3.1. The mean age was 47.6 years, mean cumulative stress was 2.0 (on a scale of 1-4.5), and mean waist circumference was 98.9 centimeters at baseline.

Are independent indicators of psychosocial stress associated with increases in central adiposity over time?

Table 3.2 shows results for the first research question on whether there are independent associations between independent indicators of psychosocial stress (i.e. neighborhood physical environment, neighborhood social environment, family stress, friend/neighbor stress, safety stress, and financial vulnerability) and central adiposity, above and beyond sociodemographics and neighborhood economic composition. In chapter 2, I found that neighborhood economic composition was positively associated with central adiposity over time (Chapter 2, Model 2.3). I found that neighborhood physical environment ( $\beta$ =2.76, p=0.14), neighborhood social environment ( $\beta$ =1.14, p=0.59), friend/neighbor stress ( $\beta$ =1.50, p=0.39), safety stress ( $\beta$ =2.61, p=0.23), and financial vulnerability ( $\beta$ =-0.48, p=0.20). were not significantly associated with central adiposity, except for family stress ( $\beta$ =1.03, p=0.05). Models were adjusted for behavioral controls (i.e. alcohol use, smoking, HEI, and METs) and yielded similar results

# Is cumulative stress index associated with central adiposity over time, after controlling for neighborhood economic composition?

Table 3.4 shows results for the second research question on whether multiple sources of cumulative stress are associated with central adiposity over time, above and beyond sociodemographics and neighborhood economic composition. I found that my cumulative stress index at baseline was associated with an increase in central adiposity, after controlling for behavioral characteristics ( $\beta$ =0.28, p=0.04).

Do multiple indicators of psychosocial stress mediate the association between neighborhood economic composition and central adiposity over time?

I did not find evidence to suggest that any of my indicators of psychosocial stress (i.e. neighborhood physical environment, neighborhood social environment, family stress, friend/neighbor stress, safety stress, and financial vulnerability) mediate the association between neighborhood economic composition and central adiposity over time (Table 3.3).

# Do multiple indicators of psychosocial stress mediate the association between neighborhood economic composition and central adiposity over time?

I did not find evidence to suggest that any of my indicators of cumulative stress index mediates the association between neighborhood economic composition and central adiposity over time (Table 3.4).

# Discussion

There are two main findings of this study. The first is that after controlling for age, gender, race, marital status, education, poverty-to-income categorization, car ownership, and home ownership, neighborhood physical environment, neighborhood social environment, friend/neighbor stress, safety stress, and financial vulnerability at baseline are not associated with an increase in central adiposity. Moreover, additional models were analyzed that controlled for health behaviors (i.e. alcohol use, smoking, HEI, and METs), and did not yield significant results for these indicators of psychosocial stress. However, family stress at baseline is associated with an increase in central adiposity over time. This finding remained robust after the addition of behavior controls to the model. Second, my index of cumulative stress was not significantly associated with change in central adiposity over time in a model accounting for neighborhood poverty and individual level demographic characteristics. However, when individual health-

related behaviors were included in the model, the index of cumulative stress was positively associated with central adiposity over time. I did not find evidence to support that neighborhood physical environment, neighborhood social environment, friend/neighbor stress, safety stress, and financial vulnerability at baseline are independently associated with central adiposity over time. These findings are inconsistent with other longitudinal studies that found a positive association between indicators of psychosocial stress, namely financial vulnerability, and increased weight over time (Gerace & George, 1996, Fowler-Brown, 2009). These differential findings may be due the use of different dependent variables. These studies used measures of overall obesity (i.e. weight gain and BMI), while this dissertation examines central adiposity. Further, previously reported studies did not include measures of the perceived physical environment and social environment, friend/neighbor stress, or safety stress. In contrast, I did find evidence to support that family stress at baseline is positively associated with central adiposity over time. This finding is partially consistent with Block and colleagues (2009), which found that family stress is positively associated with an increase in weight over time among NHB, NHW, and Hispanic women. My findings join studies that suggest that family stress is a potential indicator of psychosocial stress that may have harmful effects on central adiposity. Moreover, this is the first study that shows the harmful effects of family stress on central adiposity over time among adults.

The finding that my index of cumulative stress at baseline is positively associated with an increase in central adiposity over time supports my research hypothesis. This finding suggests that while neighborhood physical environment, neighborhood social environment, friend/neighbor stress, safety stress, and financial vulnerability do not have independent associations on central adiposity, they may have a cumulative effect on central

adiposity. However, for some of the indicators of psychosocial stress, the coefficients and pvalues for poverty became more significant, and the beta increases, suggesting that there may be a suppression effect, with the effects of poverty on central adiposity suppressed until after I have accounted for these respective stressors. In contrast, when I added the cumulative stress index, the coefficient for percent poverty becomes marginally significant, while the size of the coefficient is reduced.

Finally, I did not find evidence to support mediating effects of indicators of psychosocial stress, or cumulative stress index, on the association between neighborhood economic composition and central adiposity. This is the first study that I are aware of that examines whether psychosocial stress mediates the pathway between neighborhood composition and increased central adiposity over time. I did not find studies that tested whether indicators of psychosocial stress mediated the association between neighborhood economic composition and central adiposity. These findings may show that over time, individuals who are exposed to cumulative stress index are more at risk for increases in central adiposity than those with less exposure to cumulative stress index. My findings builds on a body of research that shows that greater exposure to cumulative stress is associated with a myriad of physical health outcomes (Wang et al., 2011(Williams Shanks, 2013 #414)(Bennett, 2004 #146)(Block, 2009 #322)(Glanz, 2008 #328)(Koch, 2008 #332)).

# Limitations and Strengths

The main limitation of this study is the relatively modest sample size. Future studies with larger samples should examine whether multiple indicators of psychosocial stress at baseline are associated with central adiposity over time.

Despite this limitation, a key strength of this chapter is the examination of central adiposity and a cumulative stress index, which included multiple indicators of psychosocial stress. Reviews of the literature on the relationship between stress and obesity, have recommended the inclusion of a wide range indicators of psychosocial stress. My findings may provide insight on the influence of cumulative stress index on excess central adiposity risk.

# Conclusion

In conclusion, my study implies that family stress and cumulative stress index at baseline are associated with an increase in central adiposity over time. These findings suggest that individuals who are exposed to cumulative stress are at greater risk of increased central adiposity over time than those who are exposed to less cumulative stress. Public health advocacy for more equitable environments that decrease stress exposure that stem from inequitable environments may lessen the risk of central adiposity in urban populations.
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	Mean	Percent	Range
Individual (Levels 1 and			
2)			25.0.000
Age	47.6±0.9		25.0-96.0
Female		52%	
White		17%	
Black		55%	
Hispanic		26%	
Married		27%	
Less than High School		40%	
High School		24%	
Beyond High School		35%	
Below poverty		38%	
Car ownership		66%	
Home ownership		51%	
Alcohol use		47%	
Currently Smoking		39%	
Healthy Eating Index	63.9±0.70		0.0-90.0
METs (n=314)	$1.0\pm0.10$		0.0-4.2
Waist Circumference	98.9±0.90		64.0-153.8
Body Mass Index	31.2±7.30		
Neighborhood Physical			
Environment	3.1±0.05		
Neighborhood Social	07.010		
Environment	$2.7\pm0.10$		
Family Stress	2.0±0.03		
Safety Stress	$2.5\pm0.05$		
Financial Vulnerability	$3.1\pm0.10$		1045
Cumulative Stress Index	$2.0\pm0.00$		1.0-4.5
Block Group (Level 3)			
Percent Poverty	31.9±11.20		8.0-54.0

 Table 3.1. Weighted Descriptive Characteristics

		Model 3	3.1	Model 3.2 Model 3.3		Model 3	Model 3.4 Model 3.5		Model 3.6		Model 3.7				
N=386		β	SE	β	SE	β	SE	β	SE	В	SE	β	SE	β	SE
	Intercept	106.0***	1.7	104.7***	1.8	105.7***	1.7	105.6***	1.7	105.8***	1.8	105.6***	1.8	107.9***	2.2
Level 2 (BG)	Percent Poverty	0.1**	0.0	0.1	0.0	0.1**	0.0	0.1**	0.0	0.1**	0.0	0.1**	0.0	0.1**	0.0
Level 1 (individual)	Time	-3.1***	0.6	-3.1***	0.6	-3.1***	0.6	-3.1***	0.6	-3.1***	0.6	-3.1***	0.6	-3.1***	0.6
<b>Baseline and</b>	Age	0.1***	0.0	0.1***	0.0	0.1***	0.0	0.1***	0.0	0.1***	0.0	0.1***	0.0	0.1***	0.0
Follow-up	BMI	1.8***	0.1	1.8***	0.1	1.8***	0.1	1.8***	0.1	$1.8^{***}$	0.1	1.8***	0.1	1.8***	0.1
Baseline Only	Female	-6.7***	1.0	-6.8***	1.0	-6.7***	1.0	-6.8***	1.0	-6.6***	1.0	-6.8***	1.0	-6.8***	1.0
	Hispanic	-2.6**	1.2	-2.7**	1.2	-2.7**	1.2	-2.7**	1.2	-2.6**	1.2	-3.3**	1.3	-2.3^	1.3
	White	-1.1	1.7	-1.4	1.7	-1.1	1.7	-1.2	1.6	-1.1	1.7	-1.1	1.6	-1.3	1.7
	Other	0.7	2.0	0.5	1.9	0.4	2.1	-0.2	2.3	0.3	2.2	0.8	2.1	1.2	2.2
	Married	-0.7	1.4	-0.7	1.4	-0.6	1.4	-0.8	1.4	-0.8	1.4	-0.7	1.4	-0.7	1.4
	Less than HS	0.7	1.2	0.5	1.2	0.7	1.3	0.9	1.2	0.8	1.2	0.5	1.3	0.8	1.2
	High School	0.8	1.5	0.9	1.5	0.9	1.4	1.0	1.5	0.9	1.5	0.9	1.5	0.7	1.5
	PIR	-0.5	0.4	-0.5	0.3	-0.5	0.4	-0.5	0.4	-0.5	0.4	-0.5	0.3	-0.6	0.4
	Car Owner	0.4	1.3	0.3	1.3	0.3	1.3	0.4	1.3	0.5	1.3	0.5	1.3	0.2	1.3
	Homeowner	1.4	1.5	1.6	1.5	1.4	1.5	1.4	1.5	1.4	1.5	1.5	1.5	1.1	1.4
	Alcohol use	-0.8	0.9	-0.9	0.9	-0.9	0.9	-0.9	0.9	-0.9	0.9	-0.9	0.9	-0.9	0.9
	Currently Smoking	1.3	1.1	1.4	1.1	1.4	1.1	1.3	1.1	1.3	1.1	1.2	1.1	1.4	1.2
	Healthy Eating Index	-0.1	0.1	-0.1	0.1	-0.1	0.1	-0.1	0.1	-0.1	0.1	-0.1	0.1	-0.1	0.1
	METs	-0.2	0.8	-0.3	0.7	-0.2	0.7	-0.2	0.7	-0.2	0.7	-0.3	0.7	-0.3	0.8
	NePhEn			3.3	1.9										
	NeSoEn					1.4	2.0								
	Family Stress							1.1**	0.5						
	Friend/Neighbor Stress									2.0	1.8				
	Safety Stress											2.7	2.1		
	Financial vulnerability													-0.5	0.4
	sigma square	18.04332		18.0333		18.08938		18.06051		18.04594		18.07258		18.00726	
	tau pi	64.45673		64.135		64.30356		64.13824		64.56856		63.76313		64.30574	
	tau beta	2.33184		1.968		2.14506		1.84567		1.99171		2.04478		2.04283	

 Table 3.2 Waist circumference regressed on multiple indicators of psychosocial stress, above and beyond health behaviors

^<0.10,\*0.05,\*\*<0.05, \*\*\*<0.01

N=386	B	SE	р
Intercept	106.20	1.74	0.00
Level 1 (individual)			
<b>Baseline and follow-up</b>			
Time	-3.10	0.60	0.00
Age	0.15	0.04	0.00
BMI	1.80	0.08	0.00
Baseline Only			
Female	-6.71	1.00	0.00
Hispanic	-3.03	1.23	0.01
White	-1.29	1.65	0.44
Married	-0.61	1.38	0.66
Less than HS	0.50	1.25	0.69
High School	0.98	1.46	0.50
PIR	-0.49	0.34	0.16
Car Owner	0.33	1.30	0.80
Homeowner	1.60	1.51	0.29
Alcohol use	-1.01	0.89	0.26
Currently Smoking	1.34	1.11	0.23
Healthy Eating Index	-0.09	0.06	0.10
METs	-0.24	0.73	0.74
Cumulative Stress Index	0.26	0.12	0.03
Level 2 (BG)			
Percent Poverty	0.08	0.04	0.07
sigma square	18.11445	5	
tau pi	63.7291	1	
tau beta	1.81895		

Table 3.3 Waist circumference regressed on neighborhood economic composition, cumulative stress index

Table 3.4 Results from test of mediation of neighborhood physical environment, neighborhood social environment, family stress, friend/neighbor stress, safety stress, financial vulnerability, and cumulative stress index on the association between neighborhood percent poverty and waist circumference.

										c-c'/	p-
Antecedent	Outcome	Mediator	С	c'	Σ	σ'	2σσ'	<b>(1-ρ)</b>	<b>σ(c-c'</b> )	sd(c-c')	value
	Waist										
%Poverty	Circumference										
		NePhEn	0.10	0.08	0.04	0.05	0.00	0.71	0.06	0.31	0.38
		NeSoEn	0.10	0.09	0.04	0.04	0.00	0.71	0.06	0.18	0.43
		Family									
		Stress	0.10	0.10	0.04	0.04	0.00	0.71	0.06	0.00	0.50
		Friend									
		Stress	0.10	0.10	0.04	0.04	0.00	0.71	0.06	0.00	0.50
		Safety Stress	0.10	0.10	0.04	0.04	0.00	0.71	0.06	0.00	0.50
		FiVul	0.10	0.09	0.04	0.04	0.00	0.71	0.06	0.18	0.43
		Cumulative									
		Stress Index	0.10	0.08	0.04	0.04	0.00	0.71	0.06	0.35	0.36

#### Chapter 4

### Do perceptions of neighborhood safety influence the effectiveness of a walking-group intervention designed to promote physical activity?

Insufficient levels of physical activity are associated with many indicators of cardiovascular risk, including hypertension, cholesterol, blood glucose, and central adiposity (Thompson et al., 2003). Although research has demonstrated that physical activity is beneficial to health, many in the U.S. do not meet the recommended requirements for daily physical activity (Haskell et al., 2007). Social ecological models suggest that physical activity is influenced by individual, social, and environmental factors (Sallis et al. 2012). Understanding why many do not meet physical activity requirements is a public health priority and can inform future interventions focused on reducing cardiovascular risk. Walking is the most popular way that individuals engage in physical activity levels as they provide social support for behavior change (Rothman, 2000). However, less is known about the effectiveness of these interventions in the context of the real world.

A few studies have found that behavior change interventions to promote physical activity are less effective in less supportive environments (Kerr et al., 2010; King et al., 2006; Sallis, King, Sirard, & Albright, 2007). For example, one study examined whether their interventions were equally effective in neighborhoods that were more and less walkable and found that walkability (i.e. objective measures of residential density, land use mix, and street connectivity), moderated the relationship between a three component intervention that included an interactive computer program, web-based activities, and tip sheets, to increase physical activity in men; men living in less walkable neighborhoods benefitted more from the intervention that those who lived in more walkable neighborhoods (Kerr et al., 2010). Two other studies that investigated the moderating effect of the social environment on a physical activity intervention found that individuals who reported safer neighborhoods had greater increases in physical activity (King et al., 2006; Sallis et al., 2007). However, all of these studies were individual-level interventions that used self-reported measures of physical activity and focused on increasing individual physical activity. This chapter will build on prior research by focusing on the moderating effect of the social environment on the association between a group-level intervention aimed at increasing individual-level physical activity.

Specifically, the first objective of this chapter is to assess the moderating effect of perceived safety on the relationship between walking-group interventions and objectively measured walking. It examines whether perceived stress modifies the effectiveness of a walking group intervention designed to increase physical activity. In particular, I examined whether perceptions of neighborhood safety affect the effectiveness of walking groups by reducing physical activity (e.g., reducing the number of steps walked during intervention activities).

The second objective is to examine the effect of the social environment on the relationship between physical activity and central adiposity. Physical activity has several positive benefits on central adiposity. For example, one study found that physical activity reduced abdominal fat without a reduction in weight (Janiszewski & Ross, 2007). Another study showed that physical activity increased metabolism and decreased hormones that contribute to fat deposition. In contrast to mechanisms driven by physical activity, stress does the exact opposite by decreasing the body's metabolism, increasing hormones that increase fat deposition in the abdominal area leading to central adiposity (Bruce S McEwen, 2007). Therefore, this chapter examines whether perceived stress modifies the association between increased physical activity and reduced waist circumference.

To examine these objectives, I tested two hypotheses: 1) Low perceived safety will weaken the effect of participation on physical activity over time, for example, participants who attend walking group meetings and who have lower perceptions of safety may take fewer steps during the walking groups compared to those who perceived the route as more safe.; and 2) Participants in physical activity who perceived the walking route as less safe will experience less of an effect on central adiposity. Said differently, participants who increase the number of steps over time, with low perceptions of safety, will have less reduction in waist circumference over time than those who perceive the area as more safe. This is feasible if the influence of stress on central adiposity is stronger than the influence of physical activity on waist circumference as suggested by the stress process theory. These hypotheses relate to chapters two and three by continuing to examine the association between stress and central adiposity. Therefore, this chapter asked the following questions:

#### **Research Questions:**

4.1) Does perceived safety modify associations between participation and changes in physical activity over time?

4.2) Does perceived safety modify associations between physical activity and changes in central adiposity over time?

#### Methodology

#### <u>Sample</u>

Description of the intervention and study design

This chapter drew on data from The Walk Your Heart to Health (WYHH) community intervention aimed to promote physical activity and other heart health behaviors through an emphasis on structured opportunities and social support for physical activity. Walking groups were hosted by community-based organizations and faith based organizations located in Detroit neighborhoods, and facilitated by Community Health Promoters, community residents who received training, support and supervision in group facilitation and physical activity promotion. Community Health Promoters were residents of Detroit, and matched the racial, ethnic, socioeconomic and linguistic characteristics of the study communities. Each Walk Your Heart to Health walking group lasted for 32-weeks. The intervention had an intervention and lagged intervention design. Eligibility of individuals who expressed an interest in participating in the walking group intervention was assessed via a pre-screening questionnaire: Physical Activity Readiness Questionnaire (PAR-Q). This process was used to identify any potential health concerns that might preclude participation, or warrant recommendation for a conversation with a health care provider prior to participation in the study. Following completion of the PAR-Q, eligible individuals reviewed an informed consent document which provided detailed information about the study protocol, requirements for participation, described potential risks, and benefits and rights (e.g. the right to withdraw from the study at any point). Those who agreed to participate in the study following review of the informed consent document were randomized into two groups, an intervention group and a lagged intervention group. Individuals who chose to join with one or more friends or family members were randomized as clusters (e.g., a cluster of three family members who joined together could be randomized as a group to assure that they were in the same walking group). All wore a pieso-electric pedometer for the next week, and returned to the study center to upload baseline steps prior to initiating the walking

groups. Those randomized into the intervention group began walking the following week, while the lagged intervention group began after 8 weeks, providing a short term comparison group to assess changes in walking behavior between the two groups. Five cohorts of walking groups were held beginning in 2009 and ending in 2012, with sites based in eastside, northwest and southwest Detroit.

Walk Your Heart to Health groups met three times per week for 1.5 hours during the 32 week intervention period. At each walking group session, attendance was taken, icebreakers and group discussion on a health topic were provided, the group help set the route for the day, warmed up, walked, and cooled down. Pedometers were issued to each participant at the beginning of the intervention period, and participants were encouraged to wear pedometers daily: steps were uploaded at each walking group session. These data were used to provide participants with weekly graphs showing their steps, allowing them to track their own progress over time and track changes in physical activity among participants. During the initial 8 weeks, the CHP was present at each walking group members. After 8 weeks, CHPS gradually began to reduce their leadership/facilitation role, encouraging other members of the group to take on leadership roles within the group, toward the end of building capacity for sustained leadership and group cohesion following the end of the formal intervention period.

Baseline data were collected prior to initiation of the walking groups, and consisted of psychosocial, self-reported health and health related behaviors, and anthropometric measures, including: self-reported physical activity, height (inches), weight (in pounds), resting systolic and diastolic blood pressure (micrograms mercury), waist circumference (cm), and HDL, LDL, TC, glucose and triglycerides based on a fasting finger stick. For the lagged intervention group,

which began 8 weeks after the first intervention group, a second baseline assessment was conducted prior to initiation of the walking group intervention. Both groups were surveyed at baseline, 8 weeks, and 32 weeks, and completed a short survey focused on group dynamics at 4 weeks. In addition to attendance and uploading steps at each walking group session, CHPS completed session summaries describing challenges, facilitating factors and basic structure of each session, and also recorded adverse events experienced by any walking group member.

This study analyzed cohorts 2-5 which included 473 participants ≥18 years old participated in the study: 283 (60%) identified as non-Hispanic Black or African American, 153 (35%) as Hispanic or Latino, and 13 (3%) White. I excluded cohort 1 because of missing measures for cohort 1. Walking groups were hosted by 7 faith-based organizations and 6 community-based organizations. The mean age of walking group participants were 48 years old.

#### <u>Measures</u>

There were three *dependent variables*, one for each of the three hypotheses. They included: consistent participation (i.e. the number of weeks participants attended at least one walking group session), physical activity (e.g. mean steps per day on days when walking in group) and waist circumference (i.e. a continuous measure of waist circumference in centimeters) as a proxy for central adiposity. This study did not analyze number of steps walked alone, because I did not have information on whether participants walked in their own neighborhoods when they walked alone. My measure of perceived safety that pertained to walking without the group, asked about how safe individuals felt in their neighborhoods. Therefore, this study only examined perceived safety on the walking route and steps walked with the group. The *independent variable* was a measure of perceived safety (i.e. "I feel safe walking

on the routes") that ranged from whether the participant strongly agree, somewhat agree, neither agree nor disagree, somewhat disagree, or strongly disagree, that they felt safe the participant on the walking route based on participants responses ( to 'When you think about the physical activities you do in your walking group sessions, please think about walking, stretching, aerobics, or any other type of physical activities you do in your sessions. *Demographic control variables* consisted of age (years); and self-reported race/ethnicity.

#### <u>Analysis</u>

I used multivariate statistical models such as generalized linear models (GLM) with the generalized estimation equations (GEE) approach to test my hypotheses (Liang & Zeger, 1986). The advantage of a GEE model is that I can control for repeated measures within an individual. Secondly, GEE models permit the calculation of robust estimates for the standard errors of the regression coefficients (Burton et al, 1998). In this analysis, I used GEE to account for the clustering of observations of the same individual over time. Model 4.1 tested the hypothesis that perceptions of safety are associated with participation, above and beyond age and race.

Previous analyses of WYHH showed a main effect of participation on increased physical activity and reductions in waist circumference (Schulz et al., 2014, in press). In addition, I conducted a preliminary analyses that examined whether the main effects of perceived safety on participation and found no association. Similarly, I found that perceived safety is not significantly associated with central adiposity. The purpose of this chapter was to test two modifying pathways of perceived stress on central adiposity. The first is to examine whether perceived safety modifies the effect of participation on physical activity. Reductions in physical activity would then lead to less of a reduction in waist circumference. The second pathway was

to examine whether perceived safety modified the effect of physical activity and waist circumference. Given that the physical activity is associated with lower reactivity of the sympathetic nervous system and the HPA axis to psychological stress (Rimmele et al., 2008) that can lead to reductions in waist circumference and stress is associated with higher reactivity of the sympathetic nervous system that can lead to increases in waist circumference. This study examined this pathway to examine whether perceived stress offsets the effect of physical activity on central adiposity over time.

To test these hypotheses, I regressed physical activity on an interaction between participation and perceived safety.

# Model 4.1: PHYSICAL ACTIVITY= $\beta_0 + \beta_1 AGE + \beta_2 WHITE + \beta_3 HISPANIC + \beta_4 PARTICIPATION + \beta_5 PERCEIVED SAFETY + \beta_6 PARTICIPATION * PERCEIVED SAFETY + e$

Next I added waist circumference to the model as the dependent variable and regressed it on an interaction between physical activity and perceived safety, to test whether perceived safety modified the association between physical activity and waist circumference (Model 4.2).

### 

#### Results

The sample included the following characteristics: mean age was 43 years; 90% were female, 60% Non-Hispanic Black, 36% Hispanic, and 4% other. The mean waist circumference was 100.9 cm; mean participation was 7.6 weeks with at least once attendance; mean number of steps with the group were 7,615.6 steps, and mean perceived safety was 1.9.

#### Does perceived safety modify the relationship between participation and physical activity?

Table 4.1 shows results for the second research question on whether perceptions of safety on the route modifies the relationship between consistent participation and the number of steps taken when walking with the group. Table 4.2 Model 3 shows that perceptions of safety in their neighborhood modified the association between participation and number of steps when walking alone ( $\beta$ =-78.5, p=0.01), with individuals who felt the route was safe walking slightly more than those who did not.

## Does perceived safety modify the relationship between physical activity and waist circumference?

Table 4.2 shows results for the last research question on whether perceived safety modifies the relationship between physical activity and waist circumference. Based on results reported in Table 4.2, Model 4.2, I am unable to rule out the null hypothesis that perceived safety does not modify the association between mean steps walked with the group and waist circumference ( $\beta$ = -0.0, p=0.17) (Table 4.2, Model4.2).

#### Discussion

There are two main findings of this study. The first is that after controlling for age, race, and gender, perceived safety modified the association between participation and physical activity (i.e. number of steps), with individuals who perceived the route as safe reporting more steps ( $\beta$ =-78.5, p=0.01) than those who did not find the route as safe. Second, I found no evidence to support the hypothesis that perceived safety modified the relationship between physical activity and central adiposity (i.e. waist circumference).

My finding that perceived safety modifies the association between participation and physical activity is consistent with other studies. Although my study is the first to examine this association for a walking-group intervention, individually-based interventions have found similar results. King and colleagues (2006) found that participants who strongly agreed that their neighborhoods were generally safe showed more minutes per week of 24-month moderate or vigorous physical activity relative to participants who reported their neighborhood as less safe. Collectively, these findings suggest that lower levels of perceived safety may weaken the impact of a physical activity intervention on physical activity.

The finding that perceived safety does not modify the relationship between physical activity in a walking group intervention and waist circumference is the first study to show this result. These findings may indicate that stress does not weaken the positive association between increasing physical activity has on reductions waist circumference. This finding does not support my hypothesis that the physiological response to the stress process (i.e. lowering metabolism, increasing fat deposition in the abdominal area) modifies the effect of physical activity has a role in increasing metabolism and regulating hormones that increase fat deposition, above and beyond the effects of perceived safety.

#### **Limitations and Strengths**

There are some limitations to this study. The first is that the measure of safety was a general measure that did not allow me to assess what specific forms of safety (e.g. safety from traffic, safety from crime) may hinder physical activity. Future studies should aim to distinguish which specific forms of safety may weaken physical activity interventions. Second, I did not

assess whether perceptions of safety were nested within neighborhoods with specific characteristics, or were solely an individual level phenomenon. Thus I cannot disentangle whether this reflected reporting of neighborhood conditions or simply individual variations in stress. Further, this study lacked objective measures of safety that may also influence physical activity. Future studies should include both objective and perceived measures of physical activity. Lastly, this study was not able to disentangle the cross-sectional effects from the longitudinal effects due to limitations of my measure for time. Future studies should collect data at more points in time to allow the disentangling of cross-sectional from longitudinal effects. Despite these limitations, this study had a number of strength. These included an objective measure of physical activity (i.e. accelerometer data), a modest sample size with repeated measures, an intervention/lagged intervention design.

#### Conclusion

In conclusion, my study examined two potential ways that perceived safety might have influenced the effectiveness of the walking group –associations between participation and physical activity, and associations between physical activity and obesity. I found evidence that it did indeed, influence one of these pathways – that is, among those who participated, the positive effects of participation were significantly dampened for those who saw the neighborhoods as less safe, as they took significantly fewer steps compared to those at the same level of participation who felt more safe; however, they still increased overall physical activity. This outcome suggest us that while walking groups can be effective among individuals with lower perceived safety, they appear to be less effective in improving physical activity among those who perceive their

environments as less safe. Neighborhood investments that promote safe walking routes can enhance the effectiveness of interventions to promote physical activity in urban environments.

There are several public health implications of these findings: These investments and other increased resources focused on reducing health inequities, among NHBs and Hispanics (who are more likely to live in neighborhoods with higher rates of crime and other factors that influence safety) should be the focus of future interventions and policies. For example, resources that focus on reducing violence and physical disorder, increasing police presence can improve access to safe areas that are conducive to physical activity. Finally, these increased efforts to improve safety in conjunction with interventions focused on increasing physical activity can work towards improving physical activity for NHBs and Hispanics which can lead a myriad of improved health outcomes including reduced central adiposity and improved cardiovascular health.

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(Thompson et al., 2003)

		Model 4.1			
Variable	β	S.E.	р		
Intercept	4723.3	352.0	< 0.01		
Age	-33.2	10.5	< 0.01		
Hispanic	353.6	283.3	0.21		
Participation	280.3	26.6	< 0.01		
Perceived safety	1469.7	369.4	< 0.01		
Participation*Perceived safety	-78.5	29.2	0.01		

Table 4.1 Physical activity regressed on participation and perceived safety

		<b>Model 4.2</b>			
Variable	β	S.E.	Р		
Intercept	104.8	1.8	< 0.01		
Age	-0.10	0.10	0.12		
Hispanic	-5.8	1.8	< 0.01		
Physical Activity	-0.0	0.0	0.10		
Perceived Safety	2.0	1.5	0.18		
Physical Activity*Perceived Safety	-0.0	0.0	0.18		

Table 4.2 Waist Circumference regressed on the modifying effects of perceived safety on the association between physical activity and waist circumference

#### Chapter 5 Findings from etiologic and intervention studies on the association between the social environment and central adiposity

#### **Study rationale**

The purpose of this dissertation was to examine factors in the social environment associated with increases in central adiposity over time in urban populations. This dissertation built on literature describing relationships between racial ideologies, racial segregation, differential access to material resources and pathways to health (A Schulz & Northridge, 2004; D. Williams & Collins, 2001; D.R. Williams & Williams-Morris, 2000); and research hypothesizing relationships between stress and central adiposity (P Björntorp, 1987; P. Björntorp, 1990, 1992, 1996, 1997, 2009; P. Björntorp et al., 2001). As an adaptation of The Healthy Environments Partnership Conceptual Model (AJ Schulz et al., 2005), the conceptual model of this dissertation (Figure 1) focused specifically on organizing associations between indicators of psychosocial stress (neighborhood economic composition, everyday unfair treatment, perceived neighborhood environment, family stress, friend/neighbor stress, safety stress, and financial vulnerability) and central adiposity, as a key contributor to racial, ethnic, and socioeconomic disparities in cardiovascular disease (CVD).

#### **Chapter summaries**

In chapter two, I examined associations between neighborhood economic composition, perceived discrimination, and central adiposity over time. I found that central adiposity did not differ for NHBs and NHWs, but did differ between NHBs and Hispanics. I also found that neighborhood economic composition is associated with increased central adiposity over time, with individuals who live in areas with the greatest percentage of poverty having the greatest increases. This finding suggests that poverty is an indicator of psychosocial stress that contributes to excess central adiposity. Similarly, after controlling for neighborhood economic composition, I found

that perceived discrimination was positively associated with increases in central adiposity over time among NHBs, NHWs, and Hispanics, independent of the effects of neighborhood poverty. This suggests that perceived discrimination influences individuals who live in neighborhoods of varying percent poverty.

Chapter three expanded on chapter 2 and examined the mediating effect of multiple sources of stress on the association between neighborhood economic composition and increased central adiposity over time. I found that most of the indicators of psychosocial stress (i.e. neighborhood physical environment, neighborhood social environment, friend/neighbor stress, safety stress, and financial vulnerability) were not associated with increases in central adiposity over time. Family stress was associated with increases in central adiposity over time, such that those reporting higher levels of family stress over time experienced larger increases in central adiposity over time. An index of cumulative stress was positively associated with central adiposity over time, above and beyond the effects of neighborhood poverty, individual sociodemographic characteristics, and health related behaviors. I also examined whether multiple indicators of psychosocial stress mediated the relationship between neighborhood economic composition and central adiposity over time, and I did not find evidence to support this hypothesis. These findings suggest that family stress and cumulative stress are positively associated with increased central adiposity over time, independent of neighborhood economic composition.

Chapter 4 builds on 2 and 3, by examining the theorized pathways of stress within a walking-group intervention. In particular, I examined whether aspects of the social environment modified the effectiveness of an intervention designed to promote physical activity, an important correlate of obesity. Focusing on one indicator of perceived stress, namely, perceived safety, I

examined whether it: 1) modified associations between participation and physical activity or 2) modified associations between physical activity and central adiposity. I found evidence to support the first pathway; that is, among those who participated, the positive effects of participation were significantly dampened for those who saw the neighborhoods as less safe, as they took significantly fewer steps compared to those at the same level of participation who felt more safe. I did not find evidence to support that perceived safety modified the effect of physical activity on central adiposity.

#### **Contributions to the literature**

These findings contribute to the literature by addressing four key gaps: 1) examining racial differences in central adiposity among NHBs, NHWs, and Hispanics of similar SES, controlling for neighborhood economic composition; 2) examining associations between perceived discrimination and central adiposity considering the context of neighborhood economic composition; 3) examining whether psychosocial stress mediated the pathway between neighborhood economic composition and central adiposity; and 4) examining whether perceived safety modified the effect of a walking-group intervention. Each gap and contribution to the literature is described below.

### Examining racial differences in central adiposity among NHBs, NHWs, and Hispanics of similar SES, controlling for neighborhood economic composition

This dissertation examines racial disparities in central adiposity among NHBs, NHWs, and Hispanics, controlling for neighborhood economic composition and individual SES. Social context theory proposes that racial segregation has shaped racial differences in physical and

social environments that drive disparate health outcomes (LaVeist, 2005a and b). In urban areas, racial segregation has driven differential access to material resources, which has resulted in NHBs and Hispanics disproportionately residing in neighborhoods below the poverty line (Schulz et al., 2012, Schulz et al. 2013). However, there is a dearth of research on whether race influences central adiposity, when minorities and non-minorities have similar incomes and live in similarly disadvantaged environments. Therefore, chapter 2 sought to address this question, by examining racial difference in central adiposity among NHBs, NHWs, and Hispanics living in relatively disadvantaged neighborhoods. I refer to disadvantaged as living in communities where with a high percentage of residents are living below the poverty line. According to the Census 2010, Detroit had a high proportion of households living below the poverty level (32%), which was twice the state of Michigan (33% vs 16%) (Census, 2010).

I found no difference between rates of central adiposity between NHBs and NHWs, which is consistent with Bleich and colleagues (2013). These consistent findings may be due to the study's relatively equal distribution of income between NHBs and NHWs, and controlling for neighborhood economic composition. My findings contribute to this body of literature by suggesting that NHBs and NHWs can have similar central adiposity outcomes, when they reside in communities, with similar neighborhood economic composition.

In contrast, I found different central adiposity outcomes among NHBs and Hispanics. Differences in central adiposity rates between NHBs and Hispanics did exist in the final models and may be due to differences in the neighborhood environment between the predominately NHB eastside of Detroit and the predominately Hispanic southwest side of Detroit that were not accounted for in this study. For example, Zenk and colleagues (2013), tested the relationship between food shopping behaviors and perceived discrimination in the first wave of this study, and found that NHBs relative to Hispanics were more likely to live farther from food stores and that shopping farther from home increased the likelihood of being exposed to discrimination (Zenk et al., 2013). Similarly, Zenk and colleagues (2013) found that residing in neighborhoods with a large grocery store within a 0.5 mile radius was positively associated with fruit and vegetable consumption, and that individual race/ethnicity moderated this relationship between NHBs and Hispanics, with Hispanics consuming 2.2 more servings of fruits/vegetables than NHBs, however it did not modify this association between NHBs and NHWs (Zenk et al. 2013). These findings warrant additional studies between NHBs and Hispanics to better understand racial differences in central adiposity.

# *Examine associations between perceived discrimination and central adiposity considering the context of neighborhood economic composition*

While several studies have examined the association between perceived discrimination and central adiposity, none of these studies considered neighborhood economic composition (Gee, Ro et al. 2008; Cozier, Wise et al. 2009; Hunte and Williams 2009; Hunte 2011; Lewis, Kravitz et al. 2011; Hickson, Lewis et al. 2012). Few of these studies made comparisons across race, however only one of these studies considered the confounding effects of class on race. The other two studies that ignored the confounding effects of class on race found differential outcomes by race, which may have been due to the higher number of NHBs in the study having lower mean income than NHWs and neighborhood percent poverty. Evidence suggest that neighborhood percent poverty influences obesity, through limited access to healthy foods (Zenk et al., 2013) and lack of places that are conducive to physical activity (Kwarteng et al., 2013; Schulz, 2005).

physical environment is well supported, research on its influence on the social environment and obesity is scarce. Therefore, this dissertation presents findings on the influence of the social environment, while keeping in mind the influence of neighborhood economic composition on differential obesity outcomes between NHBs and NHWs. Findings from this analysis indicate a positive association between perceived discrimination and central adiposity, above and beyond the effects of neighborhood poverty, among NHBs and NHWs. My findings are consistent with a cross-sectional analysis of the SWAN study, which found a positive association between perceived discrimination and central adiposity among NHB and NHW women in southwest Chicago (Lewis et al., 2012) of similar income. However, I have substantially extended these findings, by showing a longitudinal association in a multiethnic sample of women and men of similar income, above and beyond the effects of neighborhood percent poverty on central adiposity, above and beyond health behaviors.

# Examine whether psychosocial stress mediates the pathway between neighborhood economic composition and central adiposity

This is the first study to examine whether psychosocial stress mediates the pathway between neighborhood composition and increased central adiposity over time of which I am aware. Moreover, I did not find evidence to support the hypothesis that indicators of psychosocial stress mediate the association between neighborhood economic composition and increased central adiposity over time. Further, results reported here did not support the hypothesis that an index of cumulative stress mediated the positive association between neighborhood economic

composition and increased central adiposity over time. This finding shows that exposure to family stress and cumulative stress at baseline are longitudinally associated with increases in central adiposity over time, among NHB, NHW, and Hispanics, after controlling for demographics, BMI, and health behaviors. Although this dissertation does not satisfy all of the requirements to suggest causality (Oates, 2004), the findings reported here provide some insights into the sequence of effects.

#### Examining whether perceived safety modified the effect of a walking-group intervention

Chapter 4 suggests that there were two potential ways that perceived safety might have influenced the effectiveness of the walking group –associations between participation and physical activity, and associations between physical activity and obesity. I found evidence that it did indeed, influence one of these pathways – that is, among those who participated, the positive effects of participation were significantly dampened for those who saw the neighborhoods as less safe, as they took significantly fewer steps compared to those at the same level of participation who felt more safe. Future studies that include measures of the neighborhood environment, will help us to better understand the ways in which perceived safety are association with indicators of safety in the environment.

#### **Limitations and Strengths**

This dissertation has several limitations. First, the relatively modest size of the sample used for the study in chapters 2 and 3 may have lessened my ability to examine racial differences in obesity and should be re-examined in a larger sample to further assess NHBs, NHWs, and

Hispanics living in similar contexts to further understand the influence of social contexts on racial disparities in central adiposity. In addition, future studies with a larger variation of neighborhood economic composition that compares NHBs, NHWs, and Hispanics of similar household and neighborhood income, would help us understand the effect of racial disparities among higher SES racial groups as well. Third, while the purpose of this dissertation was to examine the influence of the social environment on central adiposity, controlling for measures of the physical environments (e.g. food environment) might have allowed me to more accurately measure my findings.

Similarly in chapter 4, there were several limitations. The first is that the measure of safety was a general measure that did not allow me to assess what specific forms of safety (e.g. safety from traffic, safety from crime) may hinder physical activity. Future studies should aim to distinguish which specific forms of safety may weaken physical activity interventions. Second, I did not assess whether perceptions of safety were nested within neighborhoods with specific characteristics, or were solely an individual-level phenomenon. Thus I could not disentangle whether these perceptions reflected reporting of neighborhood conditions or simply individual variations in stress, as measured in chapters 2 and 3. Further, this study lacked objective measures of safety that may have influenced physical activity and central adiposity. Future studies should include both objective and perceived measures of safety. Lastly, chapter 4 was not able to disentangle the cross-sectional effects from the longitudinal effects due to limitations of my measure for time. Future studies should collect data at more points in time to allow the disentangling of cross-sectional from longitudinal effects.

Despite these limitations, this dissertation has several strengths. I examined the independent and joint associations of a broad range of indicators of psychosocial stress and its

effect on central adiposity, which has been called for in the literature, particularly among individuals of lower SES. This has allowed elucidation of processes through which the social environment may influence central adiposity, thereby contributing to excess risk among urban populations of predominately low-to-moderate income neighborhoods. In addition, this study used an objective measure of waist circumference to measure central adiposity, rather than a self-reported measure, which may reduce inaccurate measures of waist circumference. Further, this is the first study that I are aware of that used an objective measure of central adiposity and examined indicators of psychosocial stress at baseline and increases in central adiposity over a 6-year follow-up, among a low-to-moderate income multiethnic urban population, above and beyond the effects of neighborhood economic composition. Finally, this is the first study that examined the modifying effect of perceived safety on the association between participation in a walking-group intervention and objectively measured physical activity and the association between physical activity and central adiposity among NHB and Hispanic women in an urban population.

#### **Public Health Implications**

This dissertation has several findings that can inform public health interventions that focus on obesity among urban populations of low-to-moderate income. My findings suggest the importance of considering social context when designing interventions to address obesity. These interventions can use the multilevel models (e.g. social ecological model) to design complex interventions that can work to address both the structural (e.g. neighborhood poverty) and individual (e.g. physical activity) influences on obesity described in chapter 1. Promising strategies to reduce the negative effects of differential access to material resources and

disinvestment in communities include asset building programs for communities and individuals. These strategies include alternative financial services within communities that will loan money to individuals to start business or home mortgages for individuals that would typically be denied at a bank (Weber & Smith, 2003); and improved educational opportunities for residents of poor communities. For individuals, the development of savings accounts so that low-income individuals can save to buy a home or start a business can help these individuals secure wealth that would normally be inaccessible to them (Weber & Smith, 2003; Williams Shanks, Boddie, & Rice, 2010). Collective efforts that build the capacity of both communities and individuals by making neighborhoods more appealing for residents with the improved financial means to relocate, can promote neighborhood revitalization and reductions in neighborhood poverty. Provided that individuals with improved financial means choose to stay in their neighborhoods, asset building will reverse the negative effects of disinvestment and promote neighborhood revitalization (Weber & Smith, 2003; Williams Shanks et al., 2010). Neighborhood revitalization is more likely if asset building is complemented by efforts to improve the environment such as providing a stronger police presence to reduce crime/violence, offering more employment/job training opportunities, providing mixed income housing, improving sidewalks, roads, and recreational facilities (Lovasi et al., 2009; Weber & Smith, 2003; Williams Shanks et al., 2010). Such improvements that work to fix deteriorating physical and social environments, that are caused by neighborhood poverty, can positively influence the development of supportive environments that facilitate reductions in obesity.
**Future Studies** 

These investments and other increased resources focused on reducing health inequities, particularly among NHBs and Hispanics (who are more likely to live in neighborhoods with higher rates of crime and other factors that influence safety) should be the focus of future interventions and policies. Future researchers should examine the effectiveness of interventions that offer supportive environments, through the strategies discussed above, coupled by behaviorchange strategies to address obesity. For example, multi-level interventions that work to revitalize community centers, where they have been abandoned or de-funded, can provide spaces for health education activities that address modifiable behaviors such as exercise and healthy eating can help to reduce obesity. In addition, the revitalization of community centers can create opportunities for community residents to build informal networks and improve social connectedness that can help in the development of more supportive environments to prevent and reduce obesity.

## Conclusions

The findings presented here build evidence on understudied factors of the social environment that contribute to central adiposity, above and beyond the effects of health behaviors. In particular, it has described ways in which differential access to material resources (i.e. neighborhood economic composition), has contributed to excess rates of central adiposity. In particular, this dissertation may explain some of the excess rates of central adiposity among NHBs and Hispanics, who disproportionately live in neighborhoods below poverty line. However, this dissertation also shows that NHWs who reside in neighborhoods below the poverty line in Detroit also experience increases in central adiposity over time.

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Finally, by understanding how specific indicators of psychosocial stress influence increases in central adiposity, the field of public health is better equipped to design interventions that aim to reduce indicators of psychosocial stress, which may reduce increases in central adiposity over time. Finally, among urban populations of predominately low-to-moderate income, interventions that improve access to material resources may greatly reduce indicators of psychosocial stress that drive excess rates of central adiposity.

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