9-6-2015

Coping with Racial Discrimination: The Impact of Substance Use, Emotional Eating, and Emotional Support on Mental and Physical Health

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This Doctoral Dissertation is submitted to The Graduate School at the University of Missouri-St. Louis in partial fulfillment of the requirements for a Doctoral degree in Clinical Psychology with an Emphasis in Behavioral Medicine.

August 2015

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Abstract

Racial discrimination is a likely contributor to disparate health outcomes for African-American and Hispanic-Americans. The current study elaborates on previous research by assessing the impact of three coping variables, substance use, emotional eating, and social support, which may explain the relationship between racial discrimination and poorer perceptions physical functioning and three health maladies, including hypertension, diabetes, and cardiovascular disease. Furthermore, the coping responses were hypothesized to serve as protective factors against the development of depression and anger symptoms.

A total of 424 Hispanic, African-American participants between the ages of 18 and 65 completed an online survey. A six variable model was tested using structural equation modeling. Within this model the roles of the coping variables as response to racial discrimination were assessed in conjunction with their subsequent impact on mental and physical health. Differences between African-American and Hispanic participants in the structural model were found, as well as gender-based differences. Meditated logistic regression methods were applied to test the coping variables as mediators in the relationship between racial discrimination and each health malady. Exposure to racial discrimination was associated with increase anger and depressive symptoms, and poorer perceptions of physical health. Substance use and emotional eating, but not social support partially explained the link between discrimination and physical health, while strengthening the relationship between discrimination and mental health. Racial discrimination predicted increased likelihood of reporting high blood pressure and cardiovascular disease, but not diabetes. Emotional eating was the only coping variable that partially explained the relationship between racial discrimination and the report of a diagnosis of hypertension and cardiovascular disease.

Keywords: Racial discrimination, health, substance use, eating, and social support

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Racial and ethnic health disparities occupy a place on the nation's healthcare

agenda. Several agencies reports document poorer health[1] outcomes for minorities

compared to Caucasian Americans (e.g., IOM, 2009; Smedley, Stith, & Nelson, 2003;

U.S. Department of Health and Human Services, 2011). Empirical research demonstrates

that African-Americans and Hispanics have lower life expectancy than Caucasians

(Williams, Yu, & Jackson, 1997; Wong et al., 2005). This is likely due to minorities

being diagnosed at a much higher rate with a multitude of chronic diseases and acute

illnesses. For example, racial minorities, especially African-Americans and Hispanic-

Americans, are disproportionately diagnosed with preventable diseases such as

cardiovascular disease, diabetes, asthma, and cancer (Betancourt, Green, Carrillo, &

Ananeh-Firempong, 2003; Deuster, Kim-Dorner, Remalay, & Poth 2011).

Racial minorities also experience disparities in health outcomes for chronic

disorders and conditions. Deaths from coronary heart disease accounted for 31.7%

percent of mortality for all racial groups in 2006 (Centers for Disease Control and

Prevention, 2011a). Mortality from coronary heart disease disproportionately affects

African-Americans, Hispanics, and Asian-Americans at a rate of 161.6, 106.1, and 77.1

per 100,000 people, respectively (Centers for Disease Control and Prevention, 2011a). A

similar trend was observed for mortality from stoke where African-Americans (61.6 per

100,000) suffered from much higher rates of death from stoke than Caucasians (41.7;

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[1] Health will be used to refer to physical health, while mental health will be used to
denote mental disorders or psychological distress.

Centers for Disease Control and Prevention, 2011a). Hypertension is another

cardiovascular ailment which disproportionately affects some races and is often comorbid

with heart disease. Age-adjusted prevalence per 100,000 for adult hypertension is

significantly higher for African-Americans (42.0) compared to Mexican-Americans

(25.5) and Caucasians (28.8; Centers for Disease Control and Prevention, 2011d).

Obesity, defined as a body mass index (BMI) greater than or equal to 30 kg/m^2,

also demonstrates significant racial divides in morbidity (Centers for Disease Control and

Prevention, 2011c). Among men over the age of 20, African-Americans and Mexican-

Americans suffer from obesity at a significantly higher rate than Caucasian-Americans

(Centers for Disease Control and Prevention, 2011c). Similarly, African-American (51%)

and Mexican American (43%) women are more obese than Caucasian women (33%;

Centers for Disease Control and Prevention, 2011c). African-Americans (11%) and

Hispanics (10.7%) suffer from significantly higher levels of diabetes, a common

complication of obesity, than Asian-Americans (8.3%) and Caucasian- Americans (7%;

Centers for Disease Control and Prevention, 2011b).

These health disparities affect both minorities born in the United States and

minority immigrants who have lived in the States for an extended period of time. Recent

immigrants (e.g., those emigrating from Mexico), however, are typically as healthy or

healthier than their Caucasian-American counterparts (Peek et al., 2010). Furthermore,

according to empirical inquiry, after several years in the U.S., the health status of

Mexican immigrants falls below the average Caucasian-American, and begins to

resemble the health of minorities born in the United States (Kaestner, Pearson, Keene, &

Geronimus, 2009). These data suggest that living as a racial minority in the United States

leads to poorer health. Given that the health disparity problem is complex, researchers should consider interactions among several determinants (Betancourt et al., 2003). These determinants include biological, sociocultural, behavioral, psychological, and environmental factors, which interact in a transactional nature across the lifespan (Holliday et al., 2009).

Racial Discrimination: Definition & Impact on Health Outcomes

Considerable research has explored some determinants of health disparities in minorities; however, recent empirical inquiry has explored and deepened the possible mechanisms for racial discrimination influencing and sustaining these disparities. One likely explanation for the aforementioned change in health status for Hispanic immigrants is the impact of exposure to racial discrimination faced by racial minorities after moving to the United States (Kaestner et al., 2009). To better understand how racial discrimination can influence health outcomes it is first necessary to define racial discrimination and discuss possible pathways that may lead to poorer health.

Racism is a worldview in which a certain race is prescribed higher status (Jones, 1997). Racial discrimination is a manifestation of racism that presents as a persistent sociocultural stressor unique to racial minorities (Thompson, 2002). Chester Pierce coined the term *racial microaggression* to denote the most common contemporary form of racial discrimination. Microaggressions are defined as "stunning, automatic acts of disregard that stem from unconscious attitudes of Caucasian superiority and [constitute] a verification of black inferiority" (Pierce, Carew, Pierce- Gonzalez, & Willis, 1978, p. 66). Microaggressions manifest as automatic, unintentional derogations of racial minorities during daily social interactions (Sue et al., 2007). Microaggressions can be both verbal

and nonverbal and help to capture and reflect the chronic and covert nature of the modern day experience of racial discrimination (Sue, Nadal, et al., 2008; Sue et al., 2007). These contemporary forms of racial discrimination are ubiquitous and usually implicit or covert (Sue et al., 2007). Due to their covert nature, when faced with a microaggression, targets may second guess the legitimacy of their *own* thoughts and feelings, a process that has been linked to a detrimental impact on mental health functioning and leads to negative affect such as anger (Sue, Bucceri, Lin, Nadal, & Torino, 2009).

Racial discrimination is also a reminder of a history of maltreatment, abuse, and trauma perpetrated on minority races by a majority race and remind targets of their lesser position in the social hierarchy (Helms, Nicolas, & Green, 2011; Williams et al., 1997). In addition, acts of racial discrimination and microaggressions remind targets that they are "aliens in their own land" and disrupt social connectedness (Sue, Capodilupo, & Holder, 2008). Such social disruptions manifest as chronic stress in humans (Smith & Ruiz, 2002). Overall, chronic exposure to racial discrimination causes chronic disruptions in social relationships leading to additional stress and chronic negative affect states, through its association with a history of violence, segregation, and slavery. Racial discrimination may be experienced in every facet of daily life, including occupational, interpersonal, and institutional realms (Elizabeth Brondolo, Brady Ver Halen, Pencille, Beatty, & Contrada, 2009; Thompson, 2002).

Emerging research explores the impact of acts of racial discrimination on the physical health of the target through problematic coping and the direct physiological effects. There is a long history of empirical focus on how exposure to acts of racial discrimination harm an individual's health (Cardarelli et al., 2010; Din-Dzietham,

Nembhard, Collins, & Davis, 2004; Finch & Vega, 2003; Krieger & Sidney, 1996; Williams, Yu, Jackson, & Anderson, 1997). Exposure to racial discrimination has been linked with negative affective states such as anger, depression, and anxiety, which leads to chronic stress and impacts the target's health (Brondolo et al., 2011). A meta-analytic study on the mental and physical impact of racial discrimination supported the conclusion that exposure to racial discrimination leads to a detriment in physical health and that this relationship was mediated by both an increased stress response and engaging in unhealthy coping behaviors such as smoking and alcohol use (Pascoe & Richman, 2009). It is also thought that racial minorities employ unhealthy behaviors such as substance use and overeating to alleviate the resulting stress from chronic exposure to racial discrimination, which ultimately leads to poorer health outcomes (Jackson, Knight, & Rafferty, 2010). Furthermore, exposure to discrimination leads to increases in systolic and diastolic blood pressure and can partially account for the observed racial differences in blood pressure (Krieger & Sidney, 1996). Based on this research, acts of racial discrimination may be one pipeline to health disparities.

The pathway to the current health disparities has yet to be fully elucidated and the underlying physiological mechanisms remain unclear. For example, the mechanism through which increased exposure to racial discrimination contributes to cardiovascular disease has yet to be fully described. A newer behavioral medicine concept, allostatic load, is a useful explanatory model regarding biological determinants of disparate levels of health in response to racial discrimination. Allostatic load provides an explanation as to how sociocultural stressors, such as racial discrimination "get under the skin" to

negatively impact health (Green & Darity, 2010). Through allostatic load, racial

discrimination contributes and maintains racial health disparities.

Allostasis: Translating Environmental Stressors into Changes in Health

Allostasis is the process through which organisms adapt to environmental

stressors. Allostasis involves a host of physiological and behavioral changes (e.g. changes

in mating and foraging behaviors when resources are scarce) which, in the short-term,

allow the organism to successfully adapt to environmental stressors (McEwen &

Wingfield, 2003). In juxtaposition to the beneficial effects of short-term allostasis, long-

term activation of allostatic processes leads to the development of disease risk factors and

eventually chronic illness (McEwen & Wingfield, 2003). When an organism encounters

an environmental stressor, allostatic load theory posits that a cascade of physiological

activity is triggered and remains active until the organism has successfully adapted to the

challenge or the environmental stressor has diminished (Juster, McEwen, & Lupien,

2010; Legato, 2010; McEwen & Wingfield, 2003). When the allostatic load reaches a

level harmful to the organism (i.e. chronic activation of the cardiovascular and hormonal

responses) it is deemed allostatic overload (McEwen & Wingfield, 2003; A. Peters &

McEwen, 2012). McEwen and Wingfield (2003) adapted allostatic load theory for use

within behavioral medicine research.

In humans, allostatic overload damages and hinders the cardiovascular and

immune systems and influences the development of obesity (Offidani & Ruini, 2012).

Negative outcomes of allostatic overload within the cardiovascular systems include

hypertension, persistently increased blood pressure, and the development of

atherosclerosis, the accumulation of debris and swelling of artery walls (Logan &

Barksdale, 2008). Hypertension and atherosclerotic plaques, buildup of debris on arterial walls, have been discovered to be risk factors for cardiovascular disease and stroke, one potential path to chronic illness (Nelson, Reiber, Kohler, & Boyko, 2007).

Chronic activation of hormones and glucocorticoid activity through the physiological adaption processes has been linked with other risk factors for chronic diseases, such as obesity and increased morbidity; these diseases occur at higher rates in some racial minority communities than in Caucasian populations (Deuster, Kim-Dorner, Remaley, & Poth, 2011; Mattei, Demissie, Falcon, Ordovas, & Tucker, 2010; A. Peters & McEwen, 2012). Research also suggests that chronic activation of the hypothalamic-pituitary-adrenal axis *(HPA-axis)*, the seat of the stress response, can lead to suppression of immune function (McEwen & Wingfield, 2003). This suppression suggests that allostatic overload may be occurring (McEwen & Wingfield, 2003). It is through chronic allostasis and allostatic overload that environmental stressors manifest as maladaptive physiological responses and lead to poorer health.

Along with physiological changes, humans also cope with environmental challenges through behavioral changes that can be beneficial or detrimental to the individual's health. Under chronic stress conditions individuals tend to rely on unhealthy coping strategies to assist in emotion regulation and self-soothing. Indeed, research suggests high levels of stress are associated with increased rates of substance use, smoking, and the likelihood of engaging in risky sexual behavior (e.g. Cohen, Schwartz, Bromet, & Parkinson, 1991; Holahan, Moos, Holahan, Cronkite, & Randall, 2003; Horwitz & White, 1991). In contrast to problematic coping, Deuster and colleagues (2011) also included examples from Mays, Cochran, and Barnes (2007) who advocated

for added consideration of behavioral responses such as cardiovascular exercise,

utilization of social support, and sleep, deeming them "resistance sources." It is believed

that these behaviors protect against additional allostatic load (Deuster et al., 2011).

Engaging in these resistance sources can alter the final impact of environmental stressors

on allostatic load and ultimately prevent further decline in the target's health. In essence,

resistance sources may buffer against allostatic overload. Therefore, the coping

mechanisms employed to deal with exposure to racial discrimination can have a

significant impact on physical health and by extension the identified disparities in health.

Racial Discrimination and Allostatic Consequences

Based on current research, the impact of exposure to acts of discrimination

resembles the effects one might expect from allostatic load mechanisms. Research

indicates that exposure to racial discrimination is associated with outcomes such as

hypertension, heart disease, obesity, and diabetes (Deuster et al., 2011; Mays et al.,

2007; Pascoe & Richman, 2009). These outcomes are in line previous research regarding

chronic environmental stressors and can be explained by the allostatic load model

(Cardarelli et al., 2010; Din-Dzietham et al., 2004; Krieger & Sidney, 1996). In an

attempt to investigate psychosocial factors which lead to cardiovascular disease, it was

found that increased exposure to racial discrimination led to elevated coronary artery

calcification, a sub-clinical marker of developing heart disease (Cardarelli et al., 2010).

Din-Dzietham and colleagues (2001) explored the impact of exposure to racial

discrimination in the workplace on African-Americans. Their study indicated that as

exposure to work-related racism increased the likelihood of developing higher levels of

blood pressure and hypertension was amplified (Din-Dzietham et al., 2004). In a similar

study, Krieger and Sidney (1996) examined the impact of racial discrimination on blood

pressure, comparing African-Americans and Caucasians. Exposure to racial

discrimination increased the blood pressure of African-Americans and when racial

discrimination was accounted for, the difference between African-Americans and

Caucasians was minimized (Krieger & Sidney, 1996). Overall, this research is consistent

with the hypothesis that allostatic processes are linked with exposure to discrimination

leading to poorer health.

Chronic exposure to racial discrimination leads to higher levels of allostatic load,

disease states, and problematic coping (e.g. substance use and overeating). The research

on discrimination and health provides an understanding of the specific health effects

which can be understood through an allostatic load model. The stress resulting from

racial discrimination can lead to several adverse health outcomes such as hypertension,

heart disease, and poorer immune function (Brondolo, Rieppi, Kelly, & Gerin, 2003;

Clark & Anderson, 1999; Din-Dzietham et al., 2004). These outcomes are consistent with

an understanding that experiences of racial discrimination incite biological and

behavioral responses that are activated during allostasis. If the effects of racial

discrimination through allostatic load are further explored, we can better understand the

potential mechanisms through which these negative experiences impact the health of

minorities and contribute to disparate health outcomes.

Racial discrimination and mental health: contribution to allostatic load

A potential link exists between racial discrimination and psychological distress

(Jackson et al., 1996). One year and lifetime rates of experiencing racial discrimination

are related to increased rates of psychiatric symptoms (Landrine & Klonoff, 1996).

Increased frequency of racial discrimination is also related to more negative

psychological outcomes for specific events and overall psychological well-being (Sellers

& Shelton, 2003). Racial discrimination has also been connected with increased trait

anxiety and state anxiety, a form of psychological distress (Brondolo et al., 2008). When

racial discrimination is less overt, or implicit, it leaves the perceiver to decide whether

their experience was discriminatory. African-Americans who in the past have

experienced discrimination are more likely to interpret ambiguous situations as racist,

thus increasing the potentially negative psychological effects of racism (Bennett, Merritt,

Edwards, & Sollers, 2004).

        Although the physical effects of implicit discrimination may not be as direct and

violent as overt racism, implicit racism has been associated with detrimental

physiological effects for its targets in addition to its psychological effects. Implicit racial

discrimination, such as microaggressions, can result in similar or worse psychological

distress than overt discrimination by causing more negative affect. As a result of

increased negative affect, the target experiences an increased state of arousal (Bennett et

al., 2004). Given the chronic nature of racial microaggressions, the target likely

experiences enduring arousal states. This constant state of arousal has been linked to

physiological effects, such as hypertension among African-Americans (Peters, 2004).

        As a form of implicit racial discrimination, microaggressions become especially

problematic due to their ambiguous nature and daily occurrence. Since implicit

discrimination is related to increased psychological distress and negative physiological

outcomes for minorities, the daily occurrence of racial microaggressions is especially

concerning. The daily occurrence of microaggressions may exacerbate the negative

psychological effects of discrimination due to their high frequency (Sue et al., 2007).

Responding to microaggressions is also difficult and problematic, which may result in the

target's inability to challenge a microaggression (Sue et al., 2007). If microaggressions

continue to occur, unchallenged by targets, the targets may experience frustration or

anger, or question their own experiences with reality, resulting in psychological distress

and detrimental effects on the welfare of targets (Sue, Capodilupo, et al., 2008).

Racial discrimination has been empirically linked with detriments in mental

health and the development of psychological distress such as anxiety, depression, and

anger. Jackson and colleagues (2010) suggest that racial minorities may use unhealthy

coping mechanisms such as substance use and overeating to manage the resultant

psychological distress from stressful experiences like racial discrimination, which is in

keeping with an allostatic load model (Mays et al., 2007). Evidence supports that in non-

psychiatric populations African-Americans have lower than expected diagnoses of mental

illnesses, especially for depression, despite experiencing more psychological distress

(Breslau et al., 2006; Hayward, Miles, Crimmins, & Yang, 2000; Kessler et al., 1994). It

is likely that African-Americans engage in behaviors to prevent psychological distress

from developing into psychiatric disorders and it is these coping mechanisms which have

implications for health. Racial minorities, especially African-Americans, experience

much higher rates of morbidity and mortality than their Caucasian counterparts; therefore

the coping mechanisms (i.e. substance use/abuse and overeating) used by racial

minorities may lead to a detriment in health contributing to documented health disparities

in race (Jackson, 1993). Further understanding of the impact of approaches to coping

with racial discrimination on physical and subsequent health disparities is needed to

continue to unravel and reverse the differences in health outcomes for racial minorities.

Racial Discrimination and Coping: Indirect Link to Allostatic Load

   Substance use. Although racial discrimination increases the allostatic burden

placed on minorities, additional detriments to health are related to the chosen coping

strategies for exposure to racial discrimination. Substance use has been identified as a

potential coping mechanism for racial minorities. Generally, empirical studies support a

correlation between increased discrimination and substance use, especially alcohol and

nicotine (e.g. Borrell et al., 2010; Krieger, Smith, Naishadham, Hartman, & Barbeau,

2005; Yoo, Gee, Lowthrop, & Robertson, 2010). Relatively few studies have attempted

to elucidate the direction of this relationship; however, it is often presumed that the

increased substance use is a by-product of exposure to discrimination for many

minorities. In fact, a longitudinal study, which examined this relationship, lent evidence

to the assertion that increased exposure to racial discrimination precedes increased

substance use. Racial discrimination experienced in adolescence predicted increased

illicit drug and alcohol use in adulthood (Brodish et al., 2011). More research is needed to

further explore the directionality of this relationship.

   Racial discrimination has been attributed to increased levels of tobacco, alcohol,

and chronic drug use in Latino, Asian-American, and African-American populations

(e.g., Borrell et al., 2010; Krieger et al., 2005; Landrine et al., 2006; Yoo, Gee, Lowthrop,

& Robertson, 2010). In addition, it has been noted that immigrants to the United States

initially have lower rates of alcohol use. However, these rates increase with more time in

the US, which has been linked with adapting to life in the US, including exposure to

racial discrimination. Additionally, research supports that racial discrimination becomes a

stronger threat to health as the individual's time in the US increases (Kaestner et al.,

2009; Yoo et al., 2010).

Increased negative affect states, such as hostility, also increase the probability of

using negative coping mechanisms such as smoking and alcohol use, which have a

cumulative negative impact on health (Borrell et al., 2010; Bunde & Suls, 2006). Over

4,000 college graduates were given a personality measure and the students with higher

levels of hostility used alcohol more often and exercised less (Siegler et al., 1992). It is

likely that the resultant anger, hostility, anxiety, and depression of exposure to racial

discrimination increases the risk of negative health consequences through problematic

coping, in keeping with allostatic load theory. Therefore, the affect states generated by

experiences of racial discrimination may lead to an increase in risk for alcohol or drug

usage as they are used to cope with these feelings. Continued alcohol, nicotine, and drug

use contribute to poorer health outcomes for minorities while protecting their mental

health by reducing the impact of negative affect states.

Emotional eating. According to empirical studies, chronic life stress was found

to be related to increases in emotional eating and, more specifically, an elevated desire

for energy and nutrient rich foods, high in fats and sugars (Torres & Nowson, 2007).

Studies on both animal models and humans have clearly shown that inducing mild

stressors increased subjects' intake of sweet foods (McCann, Warnick, & Knopp, 1990;

Rowland & Antelman, 1976). In a study of United States Marines, calorie intake

increased in response to mild stressors (Popper, Smits, Meiselman, & Hirsch, 1989).

Evidence also supports that humans and rats eat less as stressors become more severe.

Therefore, there appears to be some individual variability in the response to stress leading

to either increasing or restricting food intake. African-Americans appear to be at higher

risk for increasing intake of sweet foods than Caucasian-Americans (Schiffman, Graham,

Sattely-Miller, & Peterson-Dancy, 2000). The conclusions drawn by Torres and Nowson

(2007) in the meta-study of eating and stress suggest that stress can increase or decrease

eating. More research is needed to help explain why some individuals restrict eating,

whereas others increase calorie intake. Indeed, people who are overweight tend to report

eating more in response to stress (Greeno & Wing, 1994).

　　　　Changes in eating patterns are in keeping with allostatic load theory. According

to allostatic load (McEwen & Wingfield, 2003; Peters & McEwen, 2012), chronic stress

disrupts the normal adaptive functioning of systems within humans. Under chronic stress

conditions, the brain must limit its response to the stress system to reduce the damage to

the body as result of continuous allostasis. This reduces its responsiveness to the stress

system, throttling down cortisol, blood pressure, and inflammatory response. According

to Peters and McEwen (2012), the consequence of this throttling down of the brain's

response to the stress system results in obesity. However, obesity occurs only when the

body's stress response system does not provide sufficient energy resources to the brain

from the body. Therefore, more calories are consumed to "feed" the brain as the cortisol

system instructs the body to search out nutrient and energy rich foods. The surplus of

glucose then continues to accumulate and is stored as fat within the body (Peters &

McEwen, 2012). It is in this way that it is believed that chronic stressors contribute to the

development of obesity and diabetes through overeating (Torres & Nowson, 2007).

Minorities' experience with daily racial discrimination adds another chronic stressor to

the lives of minorities and likely contributes to the disparate rates of obesity and diabetes observed in Hispanics and African-Americans.

Compared to the amount of research exploring substance abuse as a coping mechanism for exposure to racial discrimination, there has been a dearth of empirical inquiry in the connections between overeating and racial discrimination. Despite this shortage of literature, a few studies suggest that racial discrimination increases the likelihood of engaging in unhealthy behaviors, such as over eating. For example, a study which explored the impact of racial discrimination longitudinally found that as perceived racial discrimination increased minorities were more likely to engage in overeating behavior (Brodish et al., 2011). This is especially of concern because diseases related to poor nutrition or emotional eating, such as diabetes and obesity, occur in higher rates in minorities, especially African-American women (Beydoun & Wang, 2009; Kahng, Dunkle, & Jackson, 2004).

Although the research remains mixed on the connection between racial discrimination and obesity, some evidence supports the assertion that exposure to racial discrimination is correlated with the increased incidence of obesity, as measured by increased waist circumference, body mass index, and diabetes (Cunningham et al., 2012; Gee, Ro, Gavin, & Takeuchi, 2008). This relationship has yet to be examined with as much empirical inquiry as substance use. Therefore, more research is needed to further the scientific understanding of the impact of exposure to discrimination in regards to eating behaviors. Additional research on emotional eating may provide other targets for intervention in the fight to reduce the prevalence of obesity and diabetes for minorities.

Seeking emotional support to cope. There is evidence in favor the role of social

support in coping with everyday stress (e.g. (Cohen & Hoberman, 1983; DeLongis,

Folkman, & Lazarus, 1988). Seeking social support has been identified as a positive

coping mechanism for stressors and has been empirically linked with an attenuation of

the stress response (Cohen & Hoberman, 1983; Cosley, McCoy, Saslow, & Epel, 2010).

Seeking social support likely does not have a direct relationship with physiological

health. However, it likely serves to protect physical health by reducing allostatic load

through a decrease in physiological reactivity. Receiving the comfort of social support

may reduce the distress associated with the experience of racial discrimination, thus

reducing the chronicity of the allostatic process of adapting to stressors, mitigating or

preventing overload.

The research on the role of social support as a moderator of the relationship

between racial discrimination and mental health remains equivocal. In a United Kingdom

study, the number of people identified as a close relationship as proxy for social support

was not supported as a protective factor against psychiatric illness (Chakraborty,

McKenzie, Hajat, & Stansfeld, 2010). Neither mobilization of social support nor

engagement of social support reduce the psychological impact of racial discrimination

(Prelow, Mosher, & Bowman, 2006). In other studies, social support moderated the

relationship between racial discrimination and mental health outcomes (Marshall & Rue,

2012). For example, increased racial discrimination was linked with lower amounts of

perceived social support. In a sample of older Black Caribbeans and African-Americans,

it was found that social connectedness and social support was associated with lower

amounts of depressive symptoms (Marshall & Rue, 2012). The role of social support as a

moderator in the relationship between racial discrimination and psychological distress

may be dependent on the individual's usage of their social support networks, the number

of racial minorities available for support, and the quality of support received.

There are also measurement issues regarding the manner by which social support

was measured in some of the studies. Social support may be measured by using the size

of one's social network, such as the number of people with whom someone is close. It

has also been measured by the amount of instrumental support. However, these

approaches to social support do not tap into whether the individual uses these supports for

general coping or racial discrimination, specifically. This may also account for the mixed

findings regarding the influence of social support in the relationship between racial

discrimination and health. More research needs to explore both the perceived support and

the way in which individuals use social support as a coping mechanism.

The amount of perceived social support has been operationalized as how much

participants believe that they receive support from others or that support is available to

them. Within perceived social support, the amount of emotional support received has also

been assessed. This specific type of support may fit best with a stressor like racial

discrimination. It has been put forth that emotional support can act as a buffer when it

creates a sense of belonging (Cohen & McKay, 1984). Emotional support may be best

suited as a moderator in the relationship between a stressor and discrimination, which

reduces one's sense of belonging such as racial discrimination (Cohen & McKay, 1984).

Racial discrimination signals to the target that they are different and challenges the

target's sense of social connectedness. Therefore, emotional support which reinforces a

sense of belonging may counteract the disruption of social connectedness linked with racial discrimination.

Despite the beneficial effects of seeking social support, there are no available studies which have examined how social support networks may also serve to increase engagement in behaviors, such as substance use and overeating as a reaction to racial discrimination. Anecdotally, this would be described as "getting a drink with the boys/girls," which may include indulging in alcohol consumption and eating unhealthy foods. In this way, social support may also fuel the engagement in unhealthy coping mechanisms. There is some evidence that marginalized individuals tend to seek out support from other marginalized people, which, in turn, puts them at higher likelihood of engaging in unhealthy behaviors, including substance use (Crawford et al., 2013). Seeking social support may lead targets of racial discrimination to seek social networks that engage in more risky behaviors, such as substance use (Myers et al., 2009). For example, it has been noted that in the Latino community social support may be linked with watching or celebrating following sporting events and, therefore, connected with increased alcohol consumption (Ornelas, Eng, & Perreira, 2011). More research into the associations between using social support and engaging in substance use is needed.

The Present Study

Research suggests that African-Americans have equal or lower amounts of psychiatric illnesses and higher physical health related mortality and morbidity rates than their Caucasian counterparts (Jackson, 2002; Jackson et al., 2010). Minorities may engage in unhealthy coping mechanisms to manage exposure to negative life events, such as poverty and crime, and protect their psychological well-being (Jackson, 2002; Jackson

et al., 2010). The goals of this study are four fold. First this study seeks to confirm that experiences with racial discrimination lead to increased compensatory behaviors in the form of substance use, overeating, and seeking social support. Additionally, this study seeks to deepen the understanding the role of social support on substance use and emotional eating. Third, this study also seeks to confirm that racial minorities may engage in unhealthy coping behaviors, which have a detrimental impact on physical functioning through health diagnoses such as cardiovascular disease, and diabetes, and perceptions of health. Finally, this study aims to include Hispanic-Americans in its investigations. Although the types of racial discrimination may differ between Hispanic-Americans and African-Americans, their experience of them likely translates to the same physiological reactions within the allostatic load model.

Research supports similar coping approaches to stressors, such as substance use in Hispanic-Americans; however, there is little research on Latinos coping with racial discrimination, especially regarding eating behaviors, and social support (Cochran, Mays, Alegria, Ortega, & Takeuchi, 2007; Tran, Lee, & Burgess, 2010). Men and women may engage in coping mechanisms at differing frequencies. For example, African-American women are more likely to cope with discrimination through eating, as evidenced by higher rates of obesity, when compared to Black men, who are more likely to use exercise or physical activity; however, gender as a moderator of substance use as a coping strategy remains inconclusive (Borrell, Dallo, & Nguyen, 2010; Brodish et al., 2011; Jackson et al., 2010). As a result, both men and women will be included in this study and treated as a covariate. The figure below is the proposed model that this study aims to validate.

Verification Hypotheses

Consistent with existing literature, we hypothesize that racial discrimination will
lead to reports of poorer physical health, as measured by a lower physical health score,
and more mental health concerns, as measured by anger and depressive symptoms.

Figure 1. Hypothesized Model

Stressor   Coping   Outcome
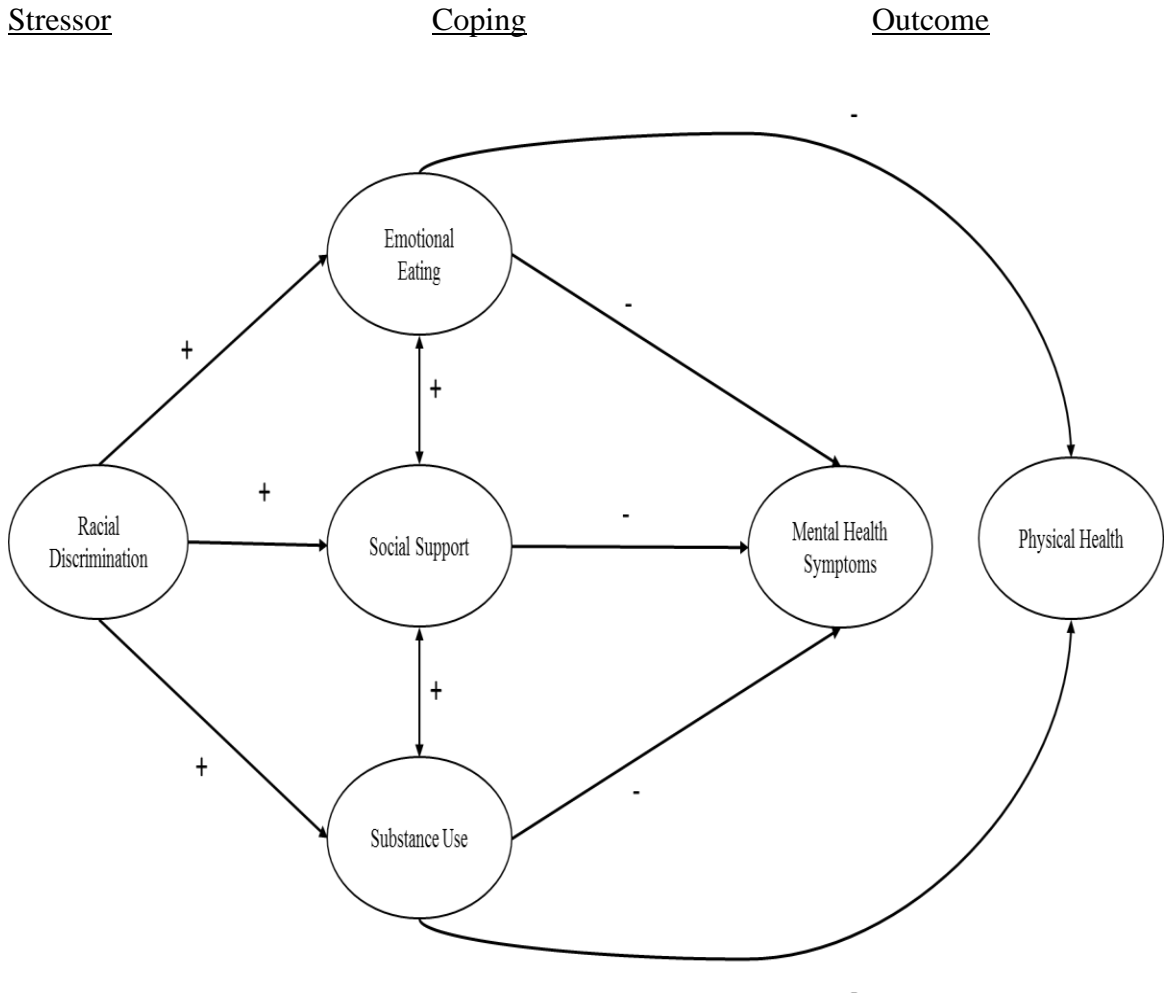
Present Study Hypotheses

The specified model will perform equal to or better than the appropriate fit indices
as elaborated by the following:

1. Increased social support will predict increased substance use and emotional eating.

2. Increased substance use and emotional eating will predict lower physical health.

3. Emotional eating will mediate the relationship between racial discrimination and physical health.

4. Substance use will mediate the relationship between racial discrimination and physical health.

5. Perceived social support will mediate the relationship between racial discrimination and physical health.

6. Emotional eating, substance use, and social support will moderate the relationship between racial discrimination and mental health.

Method

Participants. Participants were aged 18 and over. A total of 521 participants were recruited and 424 had less than 10% of their data missing and were included in the subsequent analyses. The remaining incomplete data was replaced with the median response from eligible participants. Demographic data for the 424 eligible participants are presented in Table 1. The average age for this sample was 30.43 years (SD = 8.87). Participants were majority female (57%) and identified as single or unmarried (45%). Most participants had at least some college (87.2%). Participants reported a mostly working to middle-class socioeconomic status (68%). Approximately 70.8% of the sample identified as African American/Black, 25.2% as Hispanic, and 4% as Bi-racial.

Table 1. Demographics (N = 424)		
Race\Ethnicity	**Frequency**	**Percent**
African-American	300	70.75
Hispanic	107	25.24
Biracial	17	4.01
Highest level of education		
Some high school	2	0.47
High school diploma	52	12.26
Some college/vocational training	147	34.67
College degree/completed vocational training	133	31.37
Some graduate school	36	8.49
Finished graduate school	54	12.74
Gender		
Male	178	41.98
Female	243	57.31
Transgender	3	0.71
M **age = 30.43 (SD = 8.87)**		
SONA Participants	115	22.95
Recruiting Site Participants	386	77.05

Procedure. This study was approved by the Institutional Review Board at the

University of Missouri – St. Louis. Participants were recruited through snowball

sampling using community organizations (e.g. churches and mental and physical health

organizations) and online social advertising websites (Craigslist, American Psychological

Association listserv, MTURK [Amazon Mechanical Turk], and Facebook). Participants

were also recruited through a participant recruiting pool at a large urban mid-western

university (SONA). Interested participants completed an online survey. The surveys were

confidential and took approximately 30-45 minutes to complete. When possible, student

participants were compensated with course credit, MTURK participants were provided

with $.75, and other participants were entered for a drawing for a gift certificate. In order

maintain the confidentiality of the participants, they were directed to a separate survey to

enter identifiable information for course credit, monetary compensation, and to be

entered in the drawing. Information such as names and e-mail addresses were stored

separate from their survey responses.

   Instruments. Descriptive information for instruments and correlation of instruments

of are presented in tables 2 and 3.

   Demographics questionnaire. A demographics questionnaire which assessed race (1

= African-American, 3 = Latino/Hispanic, 4 = Biracial), age, level of education (1 =

Some High School, 2 = High School Diploma, 3 = Some College/Vocational Training, 4

= College Degree/Completed Vocational Training, 5 = Some Graduate Training, 6 =

Finished Graduate School), income (1 = Lower Class, 2 = Working Class, 3 = Lower

Middle Class, 4 = Upper Middle Class, 5 = Upper Class), and gender (1 = Male, 2 =

Female, 3 = Transgender, 4 = Other) was utilized.

   Health related diagnosis. Within the demographics questionnaire, participants

were asked to indicate whether they were diagnosed with high blood pressure,

cardiovascular diagnoses, and diabetes. Participants were asked to identify whether they

had been diagnosed with cardiovascular aliments such as atherosclerosis or heart disease,

and if they had a stroke. Participants were also asked to state whether they had been diagnosed with type-2 diabetes.

   Racial discrimination. The Experiences of Discrimination Scale (EOD; Krieger et al., 2005) was used to assess exposure to racial discrimination in a variety of contexts. It assesses experiences of discrimination using frequency of and/or the number of contexts in which discrimination occurs. Sample items such as "Have you ever experienced discrimination, been prevented from doing something, or been hassled or made to feel inferior in any of the following situations because of your race, ethnicity, or color?" were used to assess racial discrimination in nine different contexts over the participants lifetime with a range of once (1) to four or more times (3). The scale has a 9-item and a 7-item version both with high test-retest reliability (all correlations above 0.69; Krieger et al., 2005). This measure was validated on African-American and Latino working-class populations with both English and Spanish versions. In the validation study the EOD had a correlation of 0.79 with a latent construct of racial discrimination. The 9-item scale had a Cronbach's alphas of 0.81 for African-Americans and Latinos independently with when evaluated by the number of contexts and Cronbach's alphas of 0.86 and 0.79 for African-Americans and Latinos, respectively, when assessed using the frequency score (Appendix A).

Mental health status. A combination of measures for psychological distress and anger were used to assess mental health as a latent variable.

   Depression. The Center for Epidemiological Studies – Depression (CES-D; Radloff, 1977) was used to measure depressive symptoms. The 20 items in the CES-D ($\alpha = 0.85$) was based on items from previously validated measures (Radloff, 1977). This

scale has also been validated for use in racial minority populations and is frequently used

to assess depression in large epidemiological studies. Items such as "I felt that I could not

shake off the blues even with help from my family or friends" and responses are scored

from rarely or none of the time (one day over the past week; 0) to most or all of the time

(5-7 days; 3). Items 4, 8, 12, and 16 are reverse scored. See Appendix B.

Anger. The Clinical Anger Scale (CAS; Snell, Gum, Shuck, Mosley, & Hite,

1995) was used to the presence of clinical levels of anger. Each item is a cluster of

choices, for example, participants may choose "I do not feel angry", "I feel angry", "I am

angry most of the time now", or "I am so angry and hostile all the time that I can't stand

it" for a single item. The scale has 21 items with an internal consistency (Cronbach's

alpha) of 0.94. See Appendix C.

Coping with discrimination.

Substance use. The Coping with Discrimination Scale (CDS; Wei, Alvarez, Ku,

Russell, & Bonett, 2010). This scale assesses how minorities cope with experiences of

discrimination with five different subscales. The Drug and Alcohol Use subscale was

used in this study to assess substance use. Coping by using drugs and alcohol is assessed

by the Drug and Alcohol Use subscale using items such as "I use drugs or alcohol to take

my mind off things". This subscale has a Cronbach's alpha of 0.72 and test-retest

reliability of 0.64. The response options range from "never like me" (1) to "always like

me" (6) on a Likert-type scale (Appendix D). Additional questions with regard to nicotine

use were created by mirroring the questions within the Drug and Alcohol Use subscale.

These questions were combined with the existing questions of the Drug and Alcohol Use

subscale to create an adapted form. For the purposes of this study the adapted Drug and

Alcohol Use subscale was used. Items 13 and 18 of the original scale and items 23 and 25

of the added questions are reverse scored.

> *Emotional eating to cope*. An adapted form of the the emotional eating subscale

(Strien, Frijters, Bergers, & Defares, 1986) from the Dutch Eating Behavior

Questionnaire (DEBQ) was used to assess eating as a result of stress and negative

emotions. This scale has 13 items, such as "Do you have the desire to eat when you are

irritated" ($\alpha = 0.94$). The items are assessed on a Likert-type scale ranging from "never"

(1) to "very often" (5). See Appendix E.

> *Social support.* Social support was measured using the Multidimensional Scale of

Perceived Social Support (MSPSS; Zimet, Dahlem, Zimet, & Farley, 1988). Items are

assessed using a Likert-type scale with seven responses ranging from very strongly

disagree (1) to very strongly agree (7). Example items are "My friends really try to help

me" and "I can talk about my problems with my family." The MSPSS assesses social

support from Family, Friends, and Significant Other (α's .87, .85, .91, respectively). The

test-retest reliability of Significant Other, Family and Friends subscales were .72, .85, .75

and .85 for the whole scale. See Appendix F.

> **Health status**. The health status of participants was measured in two ways,

including assessing for perception of physical health and the health related diagnosis

listed in the demographics questionnaire.

> *Physical health*. The Medical Outcomes Survey Short-form General Health

Survey 20-item version (SF-20; Stewart, Hays, & Ware, 1988) is a short form used to

physical health functioning. Each item is evaluated on a Likert-type scale of varying

ranges. Example items include "Does your health keep you from working at a job, doing

work around the house, or going school" and "During the last month has your health

limited your social activities." The SF-20 assesses limitations in several domains

including physical functioning (α = .86), role functioning (α = .81), social functioning[2],

mental health (α = .88), current health perceptions (α = .87), and pain[2] (Stewart et al.,

1988). See Appendix G. The physical health functioning score derived from this measure

will be used as the physical latent construct for the structural model and items were

standardized according the procedure documented in (Stewart, Hays, & Ware, 1988).

Table 2. Correlation Matrix of Variables in Main Analyses

	Substance Use	Physical Health	Emotional Eating	Discrimi- nation	Mental Health	Social Support
Substance Use	***					
Physical Health	-.492**	***				
Emotional Eating	.254**	-.378**	***			
Discrimination	.290**	-.353**	.361**	***		
Mental Health	.599**	-.666**	.528**	.504**	***	
Social Support	-.398**	.338**	-.187**	-.126**	-.423**	***

---

[2] Reliability was not reported due to the subscale consisting of one item.

Table 3. Descriptive Statistics for Scales Used in Study				
	Minimum	**Maximum**	**Mean**	**Std. Deviation**
Clinical Anger Scale	21	84	33.70	11.29
Center for Epidemological Studies – Depression Scale	20	74	39.72	12.01
Multidimensional Scale for Perceived Social Support	12	84	60.23	16.51
Medical Outcomes Survey Short-form General Health Survey 20-item version – Physical Health Score	70	1400	1026.89	310.36
Adapted Dutch Eating Behavior Questionnaire – Emotional Eating subscale	13	65	32.55	13.11
Experiences of Discrimination – Discrimination subscale	0	27	10.07	6.51
Coping with Discrimination Scale – Substance Abuse subscale	8	48	19.30	9.87

Results

Missing Data Analysis

Prior to performing analyses, the extent and pattern of missing data was assessed. Data was determined to be missing at random according to MCAR test (p = .056). Cases with more than 10% of the data missing were deleted (97 cases). The remaining missing data was imputed and the highest total of missing data for one response was 8 missing responses. In order to maintain as much data as possible and to meet the requirements for bootstrapping analyses, the remaining missing data was replaced using the median from

other responses (as shown by Gaskin, 2011 tutorial). Assumptions of normality were

tested and all variables were within accepted ranges for skew and kurtosis for structural

equation modeling (SEM) based on the suggested cut-offs (Gaskin, 2011). Parcels were

created using the CES-D and CAS to form a latent endogenous construct, mental health.

Although not displayed in the model, educational status, race, and gender were entered

into the model as covariates. However, age was not included due to missing a significant

portion of responses due to an error in the data collection software. The hypothesized

model, including covariates, was assessed using goodness-of-fit indices. The statistical

program IBM SPSS AMOS 21 was used to test the proposed model.

Due to statistical concerns regarding the inclusion of dichotomous observed

variables into a latent construct, physical health was entered as an observed variable

based on the score Physical Health Score from the SF-20 (Byrne, 2001). Secondary

analyses were completed using mediated logistical regression to test hypotheses related to

the mediating role of the coping variables. Atherosclerosis and stoke were combined with

those who reported heart disease to create a variable called cardiovascular disease due too

few positive cases (less than 10 per predictor) for atherosclerosis and stroke.

Model Testing

Hypothesized relationships between variables were assessed using structural

equation modeling (SEM). This method was used to assess the significance and size of

the effect the overall path model. SEM can also be used to test the modeled relationships

within the model providing validity and size estimates for total, direct, and indirect

effects for the hypothesized paths. Furthermore, mediation of variables with the model

can be tested using SEM by modeling direct effects, direct effects with mediator, and

testing the significance of the indirect effect using bootstrapping with bias corrected

confidence intervals (e.g., Hypotheses 2 and 3). Structural equation modeling also

assesses the relationship between observed and unobserved (latent) variables and controls

for the measurement error that is woven within responses to the survey items.

In the first step of completing structural equation modeling, observed variables

(indicators) and the composition of latent variables are identified or developed. The

survey item responses are combined into indicators and the relationships amongst these

indicators are thought to give rise an underlying latent variable, while controlling for

measurement error and providing a closer approximation of the "true" score for the latent

construct. In the next step, a measurement model is developed to test the various

relationships between the variables and indicators. Confirmatory factor analysis is used to

test whether the measurement model appropriately models the observed variance of the

indicators through goodness-of-fit tests, such as root mean square (RMSEA). Once the

measurement model is determined to be a close fit with the variance of the indicators, the

hypothesized structural model is tested in the third step, using the goodness-of-fit indices.

However, if the hypothesized structural model is not a good representation of the

underlying indicators, a respecified model can be developed until a proper fitting model

is developed to test the hypothesized paths, before assuming that all of the hypothesized

paths are inappropriately specified. Once an acceptably specified model has been

developed, mediation can be tested by removing competing mediating relationships and

the inclusion of the direct effect for the independent variables and then further testing

changes in the size of direct effect with the mediator relationship present.

Development of Item Parcels: Domain Representative Parceling. The initial

step of developing a structural model is to develop the indicators for the latent constructs

to be used in the model (Byrne, 2010; Kline, 2010). In the study, domain representative

parcels were used to develop the latent variables to be used in SEM process. Although

there is much debate regarding using item parcels versus each item to develop latent

variables, there are several advantages to using aggregated scores or parcels over

individual items. These advantages include needing smaller sample sizes to model the

relationships, due to fewer parameters, increased reliability, and less likelihood of

violating assumptions of SEM, such as univariate and multivariate normality (Little,

Cunningham, Shahar, & Widaman, 2002). Therefore, item parceling has been identified

as an important tool for the use of SEM. Just as there has been debate on whether to use

parceling, there has been debate on how parcels are to be created. Due to the high

likelihood that latent constructs assessed within social sciences are multivariate, domain

specific parceling was used, as it has been shown to be most likely to estimate the "true

score" of the latent variables and is more robust in the presence of multivariate latent

constructs (Graham & Tatterson, 2000). Exploratory factor analysis using maximum

likelihood estimation and promax rotation was used to determine the underlying factors

for each scale. An Eigenvalue of 1 was used to determine the number of appropriate

factors for each scale. Once the factor structure was determined, items were, as evenly as

possible, spread across each of the three parcels. Three parcels were used as the most

ideal number of parcels for use in structural equation modeling (Graham & Tatterson,

2000; Little et al., 2002). For example the CDS substance use scale was found to have

two factors underlying it and items of the first factor were spread across the three parcels.

Next the remaining items on that loaded on the second factor were spread across the three parcels.

Univariate and multivariate normality is an assumption of SEM and violations of these assumptions can be problematic (Byrne & van de Vijver, 2010; Kline, 2010). The absolute value of skewness and kurtosis were examined for the indicator parcels to determine univariate normality. Absolute values of skew above 3.0 and indices of kurtosis above 7 indicate problematic violations of the assumptions of univariate normality (Byrne, 2001; Kline, 2005). The highest absolute value of the skew index for the indicators in the study was .818, which is well below the suggested cutoff. Similarly, the highest absolute value statistic for kurtosis was .947. Based on these statistics, the indicators were all determined to be consistent with univariate normality. Multivariate normality is another concern for SEM, which can bias the regression weights estimates and the fit statistics. The data violated the assumption of multivariate normality, with a Mardia's normalized estimate of multivariate kurtosis statistic above of 40 (above the suggested cutoff of 3; (Mardia, 1970, 1974). This indicates significant multivariate kurtosis; however, the utilization of bootstrapping to obtain regression weights was used to control for multivariate non-normality. The latent variables were found to be within linearity assumptions. Similarly there were no issues of multicollinearity as the (VIF) statistic for all predictors in the model were below the cutoff of 3, with the highest statistic being 1.28. After assessing assumptions and instituting means to correct for any violations, the next step is to develop an acceptable measurement model.

Measurement Model. A measurement model was developed with the parceled indicators for each of the latent variables within the model. Figure 2 depicts the

measurement model for this study. Latent variables are shown as circles and observed

indicator variables are shown as rectangles. Predicted relationships between the observed

and latent variables are drawn with arrows and curved arrows are used for the

relationships between latent variables as they are allowed to covary with each other.

Figure 2. CFA Model
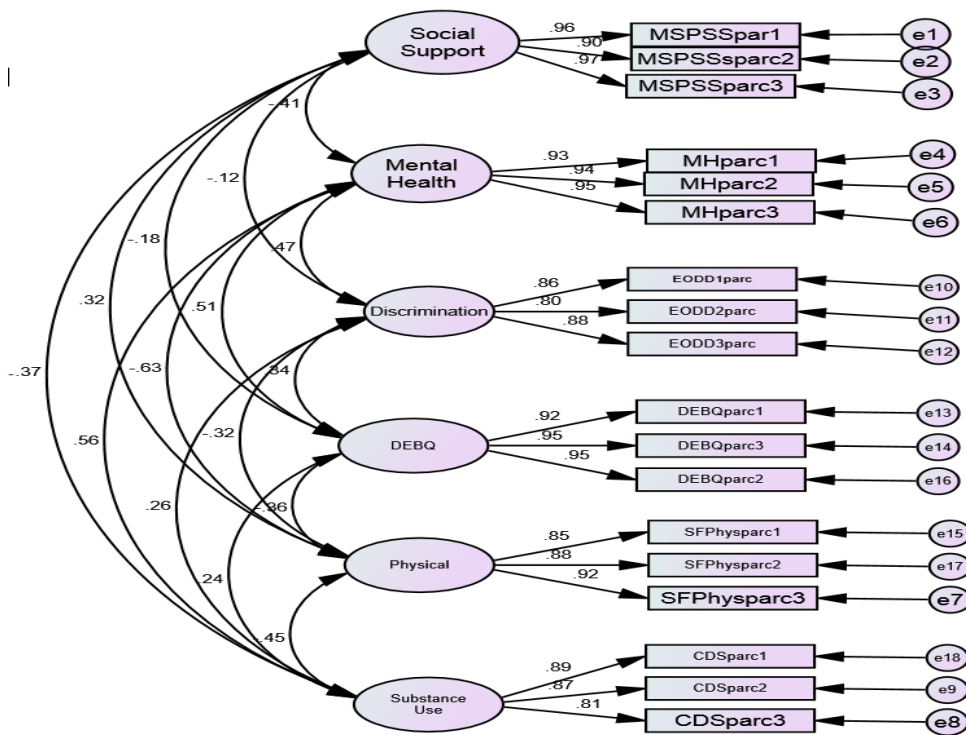
Testing the validity of a measurement model is the next step of SEM (Byrne,

2010; Kline, 2005). The measurement model is tested using confirmatory factor analysis

(CFA) and goodness-of-fit indices are viewed to assess whether the measurement model is representative of the covariances between indicators and the covariance of the sample data (Byrne, 2010; Kline, 2010). A valid model fits closely with the data and goodness-of-fit indices such as RMSEA provide the information necessary to test the appropriate fit of the model. Best practice is to evaluate several fit indices to distinguish the adequacy of the measurement model (Byrne, 2001).

The measurement model (Figure 2) was evaluated using IBM SPSS AMOS 21 to determine whether the proposed model is good fit for the data. The model fit was assessed using the various fit indices testing both normally distributed and non-normally distributed data. The chi-square test (CMIN) was significant (CMIN = 236.60, $p < .001$), which indicated that the model is not close fit to the data. However, it has been suggested that this is not as useful of a measure of model fit, due the need for a large sample size and the likelihood of a non-significant result is unrealistically difficult to achieve in most research (Joreskog & Sorbom, 1993; McCallum et al., 1996). Due to this limitation, other fit indices were also assessed. The root mean square error of approximation (RMSEA, .048) tests indicated a good model fit, as the indices should range from .01 to .08 to show acceptable fit between the proposed model and the data, with RMSEA below .06 showing good fit (Hu & Bentler, 1999). Likewise the PCLOSE statistic for RMSEA was about the suggested cut-off of .5 (PCLOSE = .636), confirming an appropriately specified model based on the RMSEA goodness-of-fit index. The normed fit index (NFI, .969) and comparative fit index (CFI, .984) are above the suggested cutoff (.95; Hu & Bentler, 1999), which is an indication that the hypothesized model is of good fit when compared to independence model, which proposes no relationship between variables. The Revised

Fit Index (RFI) for the measurement model was .960 and above the suggested cutoff of

.95. The Hoelter critical N test (CN) statistic is used to indicate appropriate sample size

and fit for the data (Hoelter, 1983). Statistics greater than 200 for .01 and .05 are indicate

good fit and the hypothesized model is above this cut-off (CN .05 = 214 and CN .01 =

230). Based on the aforementioned information it appears that the model is a good fit and

likely appropriately specified. The standardized betas, standard error, and significance

level for the proposed relationships are presented in Table 4.

Table 4. Measurement Model Fit Indices (N = 424)		
Fit Index	**Value**	**Recommended Value**
Chi-Squared Probability	.001	>.01
Root mean squared error of approximation (RMSEA)	.048	<.08
PCLOSE	.636	>.5
Normed Fit Index (NFI)	.969	>.950
Comparative Fit Index (CFI)	.984	>.950
Relative Fit Index (RFI)	.960	>.950
Comparative Fit Index	.980	>.950
Hoelter .05 (CN)	263	>200
Hoelter .01 (CN)	285	>200

Note: PCLOSE = probability of a RMSEA value less than .05.

      Hypothesized Structural Model. Due to a high index of modification for the

error terms for the latent variables of Mental and Physical Health, their error terms were

covaried. The influence of gender, race, and education were controlled for by adding

covariance arrows with observed variables, such as racial discrimination, and single

arrows for relationships with latent variables. Figure 3 depicts the hypothesized structural

model. The model fit was assessed using the various fit indices previously discussed. The

chi-square test (CMIN) was significant (CMIN = 357.87, p < .001), which indicated that

the model is not close fit to the data. The root mean square error of approximation

(RMSEA, .057) tests indicated a good model fit. The PCLOSE statistic for RMSEA was

below the suggested cut-off (PCLOSE = .066), indicating problems with model fit.

Similarly, the normed fit index (NFI, .953) and comparative fit index (CFI, .972) are

above the suggested cutoff (.95; Hu & Bentler, 1999). In regards to the Hoelter critical *N*

test (CN), the hypothesized model is above this cut-off indicating that model is

representative of the data (CN .05 = 214 and CN .01 = 230, Hoelter, 1983). Based on the

aforementioned information it appears that the model fit could be improved. Fit indices

are summarized in Table 5.

Figure 3. Hypothesized Structural Model
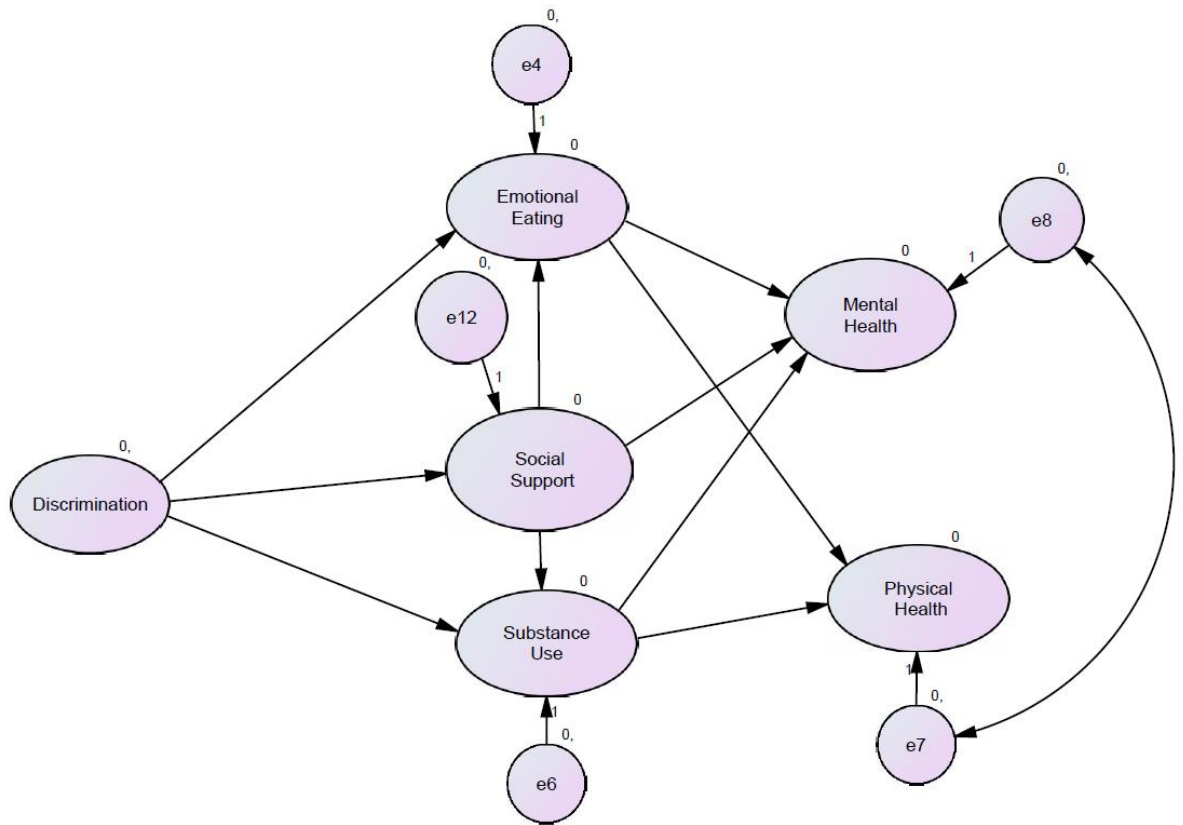
Note: Race, education, and gender were added as controls, but are not shown in the

figure.

Table 5. Hypothesized Structural Model Fit Indices (N = 424)		
Fit Index	**Value**	**Recommended Value**
Chi-Squared Probability	.001	>.01
Root mean squared error of approximation (RMSEA)	.057	<.08
PCLOSE	.066	>.5
Normed Fit Index (NFI)	.953	>.950
Comparative Fit Index (CFI)	.972	>.950
Relative Fit Index (RFI)	.944	>.950
Hoelter .05 (CN .05)	214	>200
Hoelter .01 (CN .01)	230	>200

Note: PCLOSE = probability of a RMSEA value less than .05.

Respecified Structural Model. A respecified model (Figure 4) was developed by adding the arrows to denote assessing for the direct effect of discrimination as a predictor for mental health and physical health and an arrow to signify a direct effect of social support on physical health. In addition, insignificant relationships with gender, race, or education were trimmed from the model as they may artificially lower the fit of the model, due to inappropriately specified parameters. The chi-square test (CMIN) was significant (CMIN = 335.17, p < .001), which indicated that the model is not close fit to the data. The root mean square error of approximation (RMSEA, .049) tests indicated a good model fit. The PCLOSE statistic for RMSEA was not significant (p = .618), confirming an appropriately specified model based on the RMSEA goodness-of-fit index. Similarly, the normed fit index (NFI, .956) and comparative fit index (CFI, .978) are above the suggested cutoff (.95; Hu & Bentler, 1999). The CN for the hypothesized model is also above the suggested cut-off (CN .05 = 252 and CN .01 = 270, Hoelter, 1983). Based on the aforementioned information it appears that the model is a good fit

and likely appropriately specified. The fit indices are summarized in Table 6 and the

standardized betas, standard error, and significance level for the proposed relationships

are presented in Table 7. Although the model was a good fit statistically, the path

between racial discrimination and social support was not significant. This indicates that

exposure to discrimination did not predict changes in social support. However, all other

paths in the model were significant.

Table 6. Respecified Structural Model Fit Indices (N = 424)		
Fit Index	**Value**	**Recommended Value**
Chi-Squared Probability	.001	>.01
Root mean squared error of approximation (RMSEA)	.049	<.08
PCLOSE	.618	>.5
Normed Fit Index (NFI)	.956	>.950
Comparative Fit Index (CFI)	.978	>.950
Relative Fit Index (RFI)	.946	>.950
Hoelter .05 (CN .05)	252	>200
Hoelter .01 (CN .01)	270	>200

Note: PCLOSE = probability of a RMSEA value less than .05.

Figure 4. Respecified Structural Model
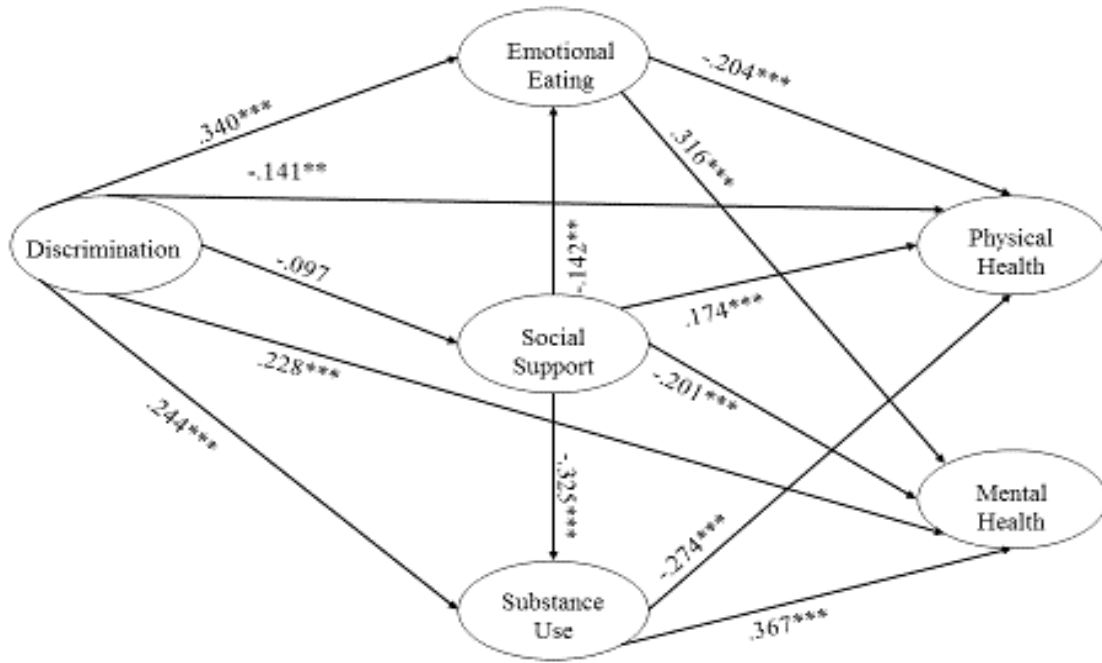
Note: Race, education, and gender were added as controls, but are not shown in the figure. * = p <.05, ** = p<.01, *** = p < .001

Table 7. Regression Weights for Respecified Structural Model (N = 424)					
Paths			**Estimate**	**S.E.**	***p***
Discrimination	→	Substance Use	.244	.078	<.001
	→	Emotional Eating	.340	.073	<.001
	→	Social Support	-.097	.103	.067
	→	Physical Health	-.141	1.471	<.01
	→	Mental Health	.228	.03	<.001
Social Support	→	Emotional Eating	-.142	.035	<.01
	→	Substance Use	-.325	.039	<.001
	→	Mental Health	-.201	.014	<.001
	→	Physical Health	.174	.707	<.001
Emotional Eating	→	Mental Health	.316	.019	<.001

	→	Physical Health	-.204	.956	<.001
Substance Use	→	Mental Health	.367	.02	<.001
	→	Physical Health	-.274	.97	<.001

Direct Effects. Bootstrapping using bias corrected 90% intervals was used to test the significance of direct and indirect effects within the model. Based on the hypothesized model, exposure to discrimination did not have significant direct effects on social support (C.I. = -.199, .020, p = .155), but had a significant direct effect on substance use and emotional eating (C.I. = .150, .329, p < .001; C.I. = .244, .431, p < .001, respectively). The direct effects of racial discrimination on mental and physical health were also significant (C.I. = .152, .304, p < .001; C.I. = -.227, -.048, p < .05). Social support was found to have significantly negative direct effects on substance use and emotional eating (C.I. = -.416, -.226, p < .001 and C.I. = -.244, -.046, p < .05). Social support was found to have a significant effect on mental health (C.I. = -.282, -.121, p < .001) and physical health (C.I. = .094, .260, p < .01). Substance use also directly impacted physical health (C.I. = -.374, -.174, p < .001) and mental health (C.I. = .287, .449, p < .001). Likewise, emotional eating also possessed a significant direct effect for physical health (C.I. = -.291, -.118, p < .001), and mental health (C.I. = .244, .384, p < .001).

Indirect Effects. Significance of the indirect effects was also tested. Discrimination was not found to have significant indirect effects for substance use or emotional eating (C.I. = -.003, .065, p = .119 and C.I. = 0, .041, p = .088). This finding signifies that social support was not a mediator of the relationship between substance use or emotional eating, as the paths through social support are the only indirect pathways for discrimination to have an impact on substance use or emotional eating. Exposure to

discrimination was found to have significant indirect effects on mental and physical

health (C.I. = .170, .293, p < .001 and C.I. = -.226, -.119, p < .001). This would indicate

the coping variables act as partial mediators in the relation between racial discrimination

and the health constructs. Social support was found to have indirect effects on mental and

physical health (C.I. = -.222, -.108, p < .001; C.I. = .071, .179, p < .001), indicating that

emotional eating and substance use may act as mediators in the relationship between

social support and the health variables. Indirect effects for substance use and emotional

eating could not be tested due to the nature of the model.

   Hypothesis 1. It was proposed that increased social support will lead to increased

substance use and emotional eating. However, the standardized regression weight of

social support on substance abuse was found to be negative (β = -.325, p < .001).

Likewise, the standardized regression weight of social support on emotional eating was

negative (β = -.142, p < .01). Based on this these effects Hypothesis 1 was not supported

and the relationship occurs in the opposite direction than hypothesized. As social support

increased by a standard deviation, substance use decreased by .33 standard deviations and

emotional eating decreased by .14 standard deviations.

   Hypothesis 2. This hypothesis proposed that emotional eating and substance use

would predict poorer physical health. The standardized regression weights for the paths

between physical health and the predictors, emotional eating and substance use, were

significant (β = -.204, p < .001, and β = -.274, p < .001, respectively). As emotional

eating increased by a standard deviation, physical health decreased by .200 standard

deviations. Similarly, as substance use increased by one standard deviation, physical

health decreased by .274 standard deviations.

Hypothesis 3. Hypothesis 3 posited that emotional eating would mediate the relationship between racial discrimination and physical health. There was a significant direct effect as evidenced by the standardized regression weight for discrimination on physical health (β = -.150, p < .01), indicating that, as discrimination increases more symptoms of depression and anger are reported. In the next step the direct effect was assessed with the mediator included in the variable and direct effect remained significant (β = -.149, p < .01). In addition, the indirect effect of exposure to discrimination and physical health was significant (C.I. = -.112, -.041, p < .001), as assessed by bootstrapping with a two-tailed 90% bias-corrected confidence interval. Therefore, the inclusion of emotional eating reduced the size of the direct effect of racial discrimination on physical health, but the direct effect remained significant. Therefore, emotional eating partially mediated the relationship between racial discrimination and physical health. Emotional eating partially accounted for the impact of racial discrimination on physical health. Table 8 provides a summary of the mediation effects.

Hypothesis 4. Hypothesis 4 stated that substance use would act as a mediator in the relationship between racial discrimination and physical health. There was a significant direct effect as evidenced by the standardized regression weight for discrimination on physical health (β = -.150, p < .01), indicating that, as discrimination increased more symptoms of depression and anger were reported. The direct effect was then assessed with the mediator included in the variable and direct effect remained significant (β = -.144, p < .01). In addition, the indirect effect of exposure to discrimination and physical health was significant (C.I. = -.105, -.038, p < .01), as assessed by bootstrapping with a two-tailed 90% bias-corrected confidence interval.

Therefore, the inclusion of substance partly accounted for the impact of racial

discrimination on physical health, but the direct effect remained significant. Therefore,

emotional eating partially mediated the relationship between racial discrimination and

physical health and partially accounted for the role of racial discrimination as a detriment

to physical health.

     Hypothesis 5. In contrast to emotional eating and substance use, social support

did not mediate the relationship between racial discrimination and physical health.

Although there was a significant direct effect for discrimination on physical health (β = -

.150, p < .01), the direct effect did not change significantly (β = -.149, p < .01), as shown

by the bootstrapped two-tailed 90% bias-corrected confidence interval (C.I. -.070, .001, p

= .106). Therefore, social support did not act a as mediator in the relationship between

racial discrimination and physical health.

Table 8. Results for Mediation of the Relationship between Racial Discrimination and Physical Health (N = 424)

Relationship	Direct without mediator	Direct with mediator	Indirect
Racial Discrimination to Emotional Eating to Physical Health	β = -.150 (p<.01)	β = -.149 (p<.01)	C.I. = -.112, -.041; p < .001; partial mediation
Racial Discrimination to Substance Use to Physical Health	β = -.150 (p<.01)	β = -.144 (p<.01)	C.I. = -.105, -.038; p < .001; partial mediation
Racial Discrimination to Social Support to Physical Health	β = -.150 (p<.01)	β = -.149 (p<.01)	C.I. = -.070 .001; NS; no mediation (p=.106)

     Hypothesis 6. Each of the coping variables were assessed for their effects as

moderators in the relationship between racial discrimination and mental health using a

process AMOS (Gaskin, 2011). Composite variables were created for all of the constructs

in the study using an AMOS 21 plug-in. Then the composite variables for racial

discrimination, emotional eating, social support, and substance use were centered and

each coping variable was multiplied with discrimination to construct interaction terms.

Next, the interaction terms were added to the model and covaried with the original latent

constructs for racial discrimination and their respective original latent variables. Results

are presented in Table 9.

Racial discrimination ($\beta = .251$, $p < .001$) and emotional eating ($\beta = .259$, $p <$

.001) were both associated with a report of more mental health complaints The

interaction between racial discrimination and emotional eating was also significant ($\beta =$

.059, $p < .05$), suggesting that the effect of racial discrimination on mental health changed

with respect to emotional eating. Emotional eating strengthened the link between racial

discrimination and mental health. For the high emotional eating group racial

discrimination had a stronger effect on mental health than in the low emotional eating

group. Figure 5 plots the simple slopes for the interaction.

Racial discrimination ($\beta = .251$, $p < .001$) and social support ($\beta = -.208$, $p < .001$)

were both associated with reporting more symptoms of depression and anger. The

interaction between racial discrimination and social support was not significant ($\beta = .009$,

$p = .750$), suggesting that the effect of racial discrimination on mental health did not

change with respect to levels of social support.

Racial discrimination ($\beta = .251$, $p < .001$) and substance use ($\beta = .373$, $p < .001$)

were both associated with more mental health problems. The interaction between racial

discrimination and substance use was also significant ($\beta = .089$, $p < .01$), suggesting that

the effect of racial discrimination on mental health changed with respect to substance use.

Substance use strengthened the link between racial discrimination and mental health. For the high substance use group racial discrimination had a stronger effect on mental health than in the low substance use group. Figure 6 plots the simple slopes for the interaction.

Figure 5. Moderation graph for emotional eating
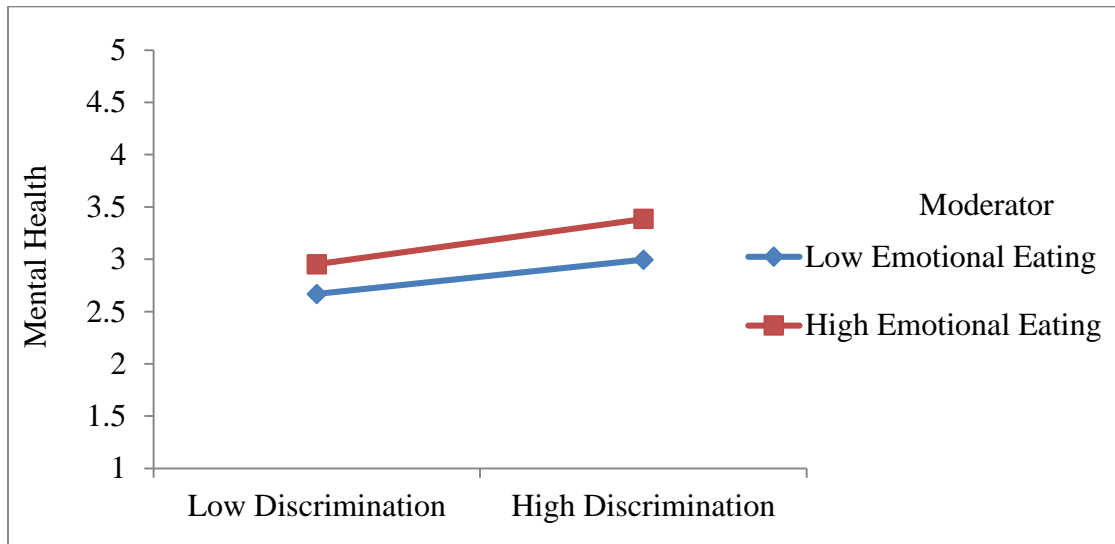
(Gaskin, 2011)

Figure 6. Moderation graph for substance use
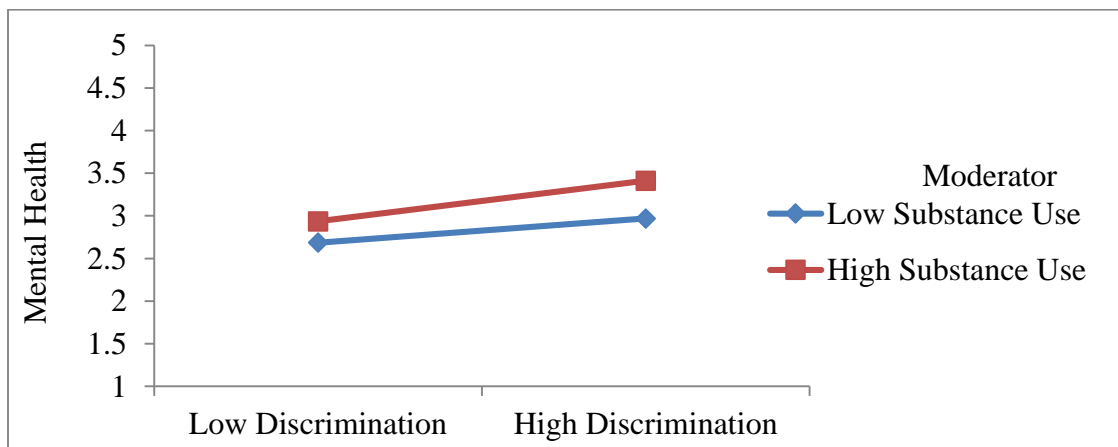
(Gaskin, 2011)

Table 9. *Results for Moderation of the Relationship between Racial Discrimination and Mental Health (N = 424)*

Relationship	Direct effect independent variable	Direct effect moderator	Interaction
Racial Discrimination to Emotional Eating to Mental Health	β = .251, SE = .026, p < .001	β = .259, SE = .019, p < .001	β = .059, SE = .013, p < .05
Racial Discrimination to Substance Use to Metal Health	β = .251, SE = .026, p < .001	β = .373, SE = .016, p < .001	β = .009, SE = .016, p = .750
Racial Discrimination to Social Support to Mental Health	β = .251, SE = .026, p < .001	β = -.208, SE = .013, p < .001	β = .089, SE = .013, p < .01

Secondary Analyses

In order to test the aforementioned hypotheses with respect to the health related variables, mediated logistic regressions were completed using IBM SPSS 21 and using the PROCESS script for testing mediation (Hayes, 2013). Each coping variable (substance use, social support, and emotional eating) and the discrimination variable were transformed into a composite variable using AMOS 21 and was entered into the logistic regression as a mediating variable. Age, education, economic status, race, and gender were entered as covariates at each step. Due to missing data with the age variable, 295 cases were used to assess the medicated relationships.

Blood Pressure

Substance Use. Mediated regression analysis was used to investigate whether substance use mediates the impact of discrimination on the predicted likelihood that the participant reported a diagnosis of high blood pressure. Racial discrimination was a

significant predictor of whether a participant reported having high blood pressure (β = .921, SE = .22, p < .001). The results also supported that exposure to discrimination is a significant predictor of substance abuse (β = .494, SE = .089, p < .001) and that substance abuse is a significant predictor of the likelihood of developing high blood pressure (β = -.319, SE = .162, p < .05). Discrimination remained a significant predictor of high blood pressure (β = .800, SE = .2131, p < .001) when substance use was included in the model. Approximately 22.3% of the variance in high blood pressure was accounted for by the predictors (Nagelkirk R^2 = .223). The indirect effect was tested using a bootstrap estimation approach with 1000 samples (Shrout & Bolger, 2002). These results indicated the indirect coefficient was not significant, β = .158, SE = .086, CI = -.003, .339, p = .068. This result indicated that substance use was not mediator with regard to the relationship between racial discrimination and hypertension.

  Social support. Mediated regression analysis was used to investigate whether social support mediates the impact of discrimination on the predicted the likelihood that the participant reported a diagnosis of high blood pressure. Exposure to racial discrimination is not a significant predictor of availability of social support (β = -.162, SE = .120, p = .177) and that availability of social support is not a significant predictor of the likelihood of blood pressure (β = -.163, SE = .113, p = .150). Therefore exposure to discrimination on the likelihood of being diagnosed with high blood pressure was not mediated by the availability of social support, discrimination remained a significant predictor of high blood pressure (β = .921, SE = .222, p < .001). Approximately 21.5% of the variance in high blood pressure was accounted for by the predictors (Nagelkirk R^2 = .215). The indirect effect was tested using a bootstrap estimation approach with 1000

samples (Shrout & Bolger, 2002). These results indicated the indirect coefficient was not

significant, β = .026, SE = .030, CI = -.009, .129, p = .379.

    Emotional Eating. Mediated logistic regression analysis was used to investigate

whether emotional eating mediates the impact of discrimination on the predicted

likelihood that the participant reported a diagnosis of high blood pressure. The results

supported that exposure to discrimination is a significant predictor of emotional eating (β

= .484, SE = .070, p < .001) and that emotional eating is a significant predictor of the

likelihood of developing blood pressure (β = .392, SE = .186, p < .05). The impact of

exposure to discrimination on the likelihood of being diagnosed with high blood pressure

was partial mediated by emotional eating; discrimination was remained a significant

predictor of high blood pressure when emotional eating was included (β = .756, SE =

.236, p < .01). Approximately 22.6% of the variance in high blood pressure was

accounted for by the predictors (Nagelkirk R^2 = .226). The indirect effect was tested

using a bootstrap estimation approach with 1000 samples (Shrout & Bolger, 2002). These

results indicated the indirect coefficient was significant, (β = .190, SE = .095, CI = .026,

.393, p < .05). Results of mediational analyses are presented in Table 10.

Table 10. *Results for Mediation of the Relationship between Racial Discrimination and Blood Pressure (n = 295)*				
Relationship	**Direct effect without mediator**	**Direct effect with mediator**	**Indirect Effect**	**Nagelkirk R²**
Racial Discrimination to Emotional Eating to Blood Pressure	.921 (p<.001)	.756 (p<.01)	C.I. = 026, .393; p < .05; partial mediation	.226
Racial Discrimination to Substance Use to Blood Pressure	.921 (p<.001)	.925 (p<.001)	C.I. = -009, .129; p = .379; No mediation	.223
Racial Discrimination to Social Support to Blood Pressure	.921 (p<.001)	.800 (p<.001)	C.I. = -.003, .339; NS; no mediation (p=.068)	.215

Note: 75 participants reported a diagnosis of high blood pressure. Race, age, education, and gender were included as covariates. Estimates are not standardized due to constraints of logistical regression in SPSS.

Type-2 Diabetes. Racial discrimination was not a significant predictor of the likelihood that a participant reported having a diagnosis of type-2 diabetes (β = .306, SE = .216, p = .157). Therefore, mediational analyses were not run.

Cardiovascular Disease. If respondents selected either heart disease or atherosclerosis they were combined into a dichotomous variable in order to test the effects of mediators on the relationship between racial discrimination and cardiovascular disease. Exposure to racial discrimination significantly predicted an increased likelihood of respondents reporting cardiovascular disease (β = 1.11, SE = .348, p < .01).

Substance Use. Mediated regression analysis was used to investigate whether substance use mediates the impact of discrimination on the predicted likelihood that the

participant reported a diagnosis of cardiovascular disease. The results supported that

exposure to discrimination is a significant predictor of substance abuse (β = .494, SE =

.089, p < .001), but that substance abuse was not found to be a significant predictor of the

likelihood of cardiovascular disease (β = .373, SE = .270, p = .167). The impact of

exposure to discrimination on the likelihood of being diagnosed with cardiovascular

disease was therefore not mediated by substance use. Approximately 14.2% of the

variance in high blood pressure was accounted for by the predictors (Nagelkirk R^2 =

.142). The indirect effect was tested using a bootstrap estimation approach with 1000

samples (Shrout & Bolger, 2002). These results indicated the indirect coefficient was not

significant, β = .184, SE = .140, CI = -.047, .455, p = .187.

Social support. Mediated regression analysis was used to investigate whether

availability of social support mediates the impact of discrimination on the predicted

likelihood that the participant reported a diagnosis of cardiovascular disease. The results

found that exposure to discrimination is not a significant predictor of social support (β = -

.162, SE = .120, p = .177) and that availability of social support is not a significant

predictor of the likelihood of developing cardiovascular disease (β = -.000, SE = .172, p =

.999). Therefore, exposure to discrimination on the likelihood of being diagnosed with

cardiovascular disease was not mediated by social support. Approximately 12.6% of the

variance in cardiovascular disease was accounted for by the predictors (Nagelkirk R^2 =

.126). The indirect effect was tested using a bootstrap estimation approach with 1000

samples (Shrout & Bolger, 2002). These results indicated the indirect coefficient was not

significant, β = .000, SE = .035, CI = -.097, .074, p = .974.

Emotional Eating. Mediated regression analysis was used to investigate whether

emotional eating mediates the impact of discrimination on the predicted likelihood that

the participant reported a diagnosis of cardiovascular disease. The results supported that

exposure to discrimination is a significant predictor of emotional eating (β = .484, SE =

.070, p < .001) and that emotional eating is a significant predictor of the likelihood of

developing cardiovascular disease (β = .651, SE = .302, p < .05). The impact of exposure

to discrimination on the likelihood of being diagnosed with cardiovascular disease was

not mediated by emotional eating; discrimination remained a significant predictor of

cardiovascular disease when emotional eating was added to the model (β = .835, SE =

.377, p < .05). Approximately 16.4% of the variance in cardiovascular disease was

accounted for by the predictors (Nagelkirk R^2 = .164). The indirect effect was tested

using a bootstrap estimation approach with 1000 samples (Shrout & Bolger, 2002). These

results indicated the indirect coefficient was not significant, (β = .315, SE = .155, 95% CI

= .057, .634, p < .05). Results of mediational analyses are presented in Table 11.

Relationship	Direct effect without mediator	Direct effect with mediator	Indirect Effect	Nagelkirk R^2
Table 11. *Results for Mediation of the Relationship between Racial Discrimination and Cardiovascular Disease (n = 295)*				
Racial Discrimination to Emotional Eating to Cardiovascular Disease	1.11 (p<.01)	.835 (p<.01)	C.I. = .057, .634; p < .05; partial mediation	.164
Racial Discrimination to Substance Use to Cardiovascular Disease	1.11 (p<.01)	1.02 (p<.01)	C.I. = -.047, .455; p = .187; No mediation	.142
Racial Discrimination to Social Support to Cardiovascular Disease	1.11 (p<.01)	1.11 (p<.01)	C.I. = -.097, .074; p = .999; No mediation	.126

Note: 22 participants reported a diagnosis of cardiovascular disease. Race, age, education, and gender were included as covariates. Estimates are not standardized due to limitations of logistical regression in SPSS.

Race/Ethnic Structural Model Analysis. Secondary analyses were also conducted regarding the respecified model for African-American and Hispanic American participants. The fit indices for the model were assessed for each race. African-American participants were entered as the grouping value in AMOS. Then Hispanic Americans were assessed. Next African-Americans and Hispanics were entered into the same model and based on separate groupings. The influence of gender and education were controlled for by adding covariance arrows with exogenous variables, such as racial discrimination, and single arrows for relationships with latent variables. The fit indices for African-Americans and Hispanics, separately are presented below. Relationships that were significantly different are explicated following the fit indices.

Model Fit for African-Americans. The chi-square test (CMIN) was significant

(CMIN = 289.57, p < .001), which indicated that the model is not close fit to the data.

The root mean square error of approximation (RMSEA, .055) tests indicated a good

model fit; however the PCLOSE statistic for RMSEA was not significant (.190),

indicating potential problems with model fit. The normed fit index (NFI, .947) was below

the suggested cut-off, but the comparative fit index (CFI, .974) was above it (.95; Hu &

Bentler, 1999). The Hoelter critical *N* test (CN), the respecified model gives mixed

results as the value for CN .05 was below the cut-off and the value for CN .01 was above

the cut-off (CN .05 = 214 and CN .01 = 230, Hoelter, 1983). The aforementioned model

fit indices for African-Americans provide mixed results. This is likely due to paths that

were included, but were not significant. Table 12 provides a summary of the various fit

indices. The standardized betas, standard error, and significance level for the proposed

relationships are presented in Table 14. Although the model was generally a good fit

statistically, the paths from racial discrimination to social support and physical health

were not significant (β = -.074, SE = .120, p = .233, β = .098, SE = 1.74, p = .119). This

indicates that exposure to discrimination did not predict changes in social support, nor

changes in physical health. Similarly, the path from social support to emotional eating

was not significant (β = -.031, SE = .041, p = .173). All other paths in the model were

significant.

Table 12. African-American Respecified Model Fit (N = 300)		
Fit Index	**Value**	**Recommended Value**
Chi-Squared Probability	<.001	>.01
Root mean squared error of approximation (RMSEA)	.055	<.08
PCLOSE	.190	>.5
Normed Fit Index (NFI)	.947	>.950
Comparative Fit Index (CFI)	.974	>.950
Relative Fit Index (RFI)	.934	>.950
Hoelter .05 (CN)	188	>200
Hoelter .01 (CN)	202	>200

Model Fit for Hispanics. As with the previous testing of the specified model the influence of gender and education were controlled. Model fit was assessed using the various fit indices testing both normally distributed and non-normally distributed data. The chi-square test (CMIN) was significant (CMIN = 221.65, $p < .001$), which indicated that the model is not close fit to the data. The root mean square error of approximation (RMSEA, .066) tests indicated a moderate model fit. The PCLOSE statistic for RMSEA ($p = .066$) is below the suggested cutoff confirming some potential concerns regarding model based on the RMSEA goodness-of-fit index. Similarly, the normed fit index (NFI, .899) and comparative fit index (CFI, .965) provided mixed results, as the CFI is above the cut-off, but not the NFI (.95; Hu & Bentler, 1999). The Hoelter critical *N* test (CN), for the respecified model is below the cut-off indicating that model may not be representative of the data or there are concerns regarding the sample size (CN .05 = 87 and CN .01 = 94, Hoelter, 1983). A summary of the goodness-of-fit indices is obtainable in Table 13. The model appeared to be poorly specified for Hispanics. The standardized betas, standard error, and significance level for the proposed relationships are presented in Table 14. The model fit is likely negatively influenced by the insignificant paths that are specified for this portion of respondents. The path between racial discrimination and

social support remained nonsignificant, indicating that exposure to discrimination did not

predict changes in social support (β = -.137, SE = .208, p = .173). Social support did not

predict changes in mental or physical health for Hispanic participants (β = -.071, SE =

.028, p = .173, β = .019, SE = 1.57, p = .85). All other paths in the model were

significant.

Table 13. Hispanic-American Respecified Model Fit (N = 107)		
Fit Index	**Value**	**Recommended Value**
Chi-Squared Probability	<.001	>.01
Root mean squared error of approximation (RMSEA)	.066	<.08
PCLOSE	.091	>.5
Normed Fit Index (NFI)	.899	>.950
Comparative Fit Index (CFI)	.965	>.950
Relative Fit Index (RFI)	.966	>.950
Hoelter .05 (CN)	87	>200
Hoelter .01 (CN)	94	>200

Race/Ethnic Path Differences. In order to assess differences in the path related

weights between African-Americans and Hispanic Americans, a process discussed by

Gaskin (2011) was used. The unstandardized estimates for each group and the critical

ratios (C.R.) were utilized to develop standardized z-scores and the level of significance,

using a Microsoft Excel script created by Gaskin (2011). Significant Z-scores were used

to indicate a significant difference in the paths. Based upon these analyses, several

differences between the two populations of respondents emerged. In regards to

discrimination as a predictor, race was found to moderate the paths to emotional eating,

mental health, and physical health (p <.05, p < .01, and p < .05, respectively). In

comparison to African-American respondents, discrimination had a smaller effect on

Hispanic participants. The path from discrimination to physical health was not significant

for African-Americans, but remained significant for Hispanic Americans. The path from

discrimination to mental health had a stronger effect for Hispanic Americans. For social

support as a predictor, significant differences were observed for emotional eating and

mental health ($p < .001$ for both). In contrast to African-Americans social support had no

effect on mental health. The path between social support and emotional eating for

Hispanic Americans was significant, but was not for African-Americans. African-

American and Hispanic participants differed in regards to the path between substance use

and mental health ($p < .05$); the path estimate was lower for Hispanic Americans. No

significant differences were observed for the other paths. See Table 14 for information on

standard estimates, standard error, and significance of all paths assessed in the study.

Paths			African-American (N = 300) Standard Estimate	S.E.	P	Hispanic American (N = 107) Standard Estimate	S.E.	P
Discrimination	→	Substance Use	.268	.088	<.001	.222	.162	<.05
	→	Emotional Eating*	.404	.085	<.001	.181	.143	<.05
	→	Social Support	-.074	.120	.233	-.137	.208	.173
	→	Physical Health**	-.098	1.740	.119	-.331	2.97	<.001
	→	Mental Health*	.199	.036	<.001	.368	.054	<.001
Social Support	→	Emotional Eating***	-.031	.041	.586	-.439	.068	<.001
	→	Substance Use	-.306	.047	<.001	-.411	.085	<.001
	→	Mental Health***	-.230	.017	<.001	-.071	.028	.404
	→	Physical Health	.209	.817	<.001	.019	1.57	.85
Emotional Eating	→	Mental Health	.308	.024	<.001	.402	.035	<.001
	→	Physical Health	-.195	1.160	<.001	-.337	2.051	<.001
Substance Use	→	Mental Health*	.391	.026	<.001	.286	.035	<.001
	→	Physical Health	-2.920	1.200	<.001	-.211	1.91	<.05

Table 14. Regression Weights for Paths for African-American and Hispanic Americans

Note: Significant differences in standardized estimates are denoted with * = p <.05, ** = p<.01, *** = p < .001.

Gender-based Structural Model Analysis. Secondary analyses were also

conducted regarding the respecified model for male and female participants. The fit

indices for the model were assessed for each gender. Male participants were entered as

the grouping value in AMOS, followed by female participants. Next men and women

were entered into the same model and based on separate groupings. The influence of race

and education were controlled. The fit indices for men and women, separately are

presented below. Relationships that were significantly different are explicated following

the fit indices.

　　　　Model fit for men. The chi-square test (CMIN) was significant (CMIN = 199.44,

$p < .001$), which indicated that the model is not close fit to the data. The root mean square

error of approximation (RMSEA, .052) tests indicated a good model fit; however the

PCLOSE statistic for RMSEA was below the suggested cut-off but not significant (.404),

indicating potential problems with model fit. The normed fit index (NFI, .936) was below

the suggested cut-off, but the comparative fit index (CFI, .978) was above it (.95; Hu &

Bentler, 1999). The Hoelter critical *N* test (CN), the respecified model indicated problems

with fit as the value for CN .05 and CN .01 were below the cut-off (CN .05 = 145 and CN

.01 = 157, Hoelter, 1983). The aforementioned model fit indices for men provide mixed

results. This is likely due to paths that were included, but were not significant. Table 15

provides a summary of the various fit indices. The standardized betas, standard error, and

significance level for the proposed relationships are presented in Table 17. Although the

model was generally a good fit statistically, the paths from racial discrimination to social

support was not significant ($\beta = -.005$, SE = .146, p = .954). This indicates that exposure

to discrimination did not predict changes in social support. Similarly, the path from social

support to emotional eating was not significant ($\beta = -.107$, SE = .054, p = .154). All other

paths in the model were significant.

Table 15. Men Respecified Model Fit (N = 178)		
Fit Index	**Value**	**Recommended Value**
Chi-Squared Probability	<.001	>.01
Root mean squared error of approximation (RMSEA)	.052	<.08
PCLOSE	.404	>.5
Normed Fit Index (NFI)	.936	>.950
Comparative Fit Index (CFI)	.978	>.950
Relative Fit Index (RFI)	.919	>.950
Hoelter .05 (CN)	145	>200
Hoelter .01 (CN)	157	>200

Model fit for women. As with the previous testing of the specified model the

influence of gender and education were controlled. Model fit was assessed using the

various fit indices testing both normally distributed and non-normally distributed data.

The chi-square test (CMIN) was significant (CMIN = 218.55, p < .001), which indicated

that the model is not close fit to the data. The root mean square error of approximation

(RMSEA, .051) tests indicated a moderate model fit. The PCLOSE statistic for RMSEA

(p = .456) is below the suggested cutoff confirming some potential concerns regarding

model based on the RMSEA goodness-of-fit index. Similarly, the normed fit index (NFI,

.952) and comparative fit index (CFI, .981) provided strong support for good model fit, as

the CFI and NFI are above the cut-off (.95; Hu & Bentler, 1999). The Hoelter critical *N*

test (CN), for the respecified model is below the cut-off indicating that model may not be

representative of the data or there are concerns regarding the sample size (CN .05 = 181

and CN .01 = 196, Hoelter, 1983). A summary of the goodness-of-fit indices is

obtainable in Table 16. The model appeared to be poorly specified for women. The

standardized betas, standard error, and significance level for the proposed relationships

are presented in Table 17. The model fit is likely negatively influenced by the

insignificant paths that are specified for this portion of respondents. The path between

racial discrimination and physical health for women was not significant (β = -.101, SE =

2.384, p = .157). All other paths in the model were significant.

Table 16. Women Respecified Model Fit (N = 243)		
Fit Index	**Value**	**Recommended Value**
Chi-Squared Probability	<.001	>.01
Root mean squared error of approximation (RMSEA)	.051	<.08
PCLOSE	.456	>.5
Normed Fit Index (NFI)	.952	>.950
Comparative Fit Index (CFI)	.981	>.950
Relative Fit Index (RFI)	.939	>.950
Hoelter .05 (CN)	181	>200
Hoelter .01 (CN)	196	>200

Gender-based Path Differences. Based upon these analyses, several differences

between the two populations of respondents emerged. In regards to discrimination as a

predictor, gender was found to moderate the paths to social support and mental health (p

<.05 and p < .01 respectively). For men discrimination was unrelated to social support,

while discrimination was predicted poorer social support for women. In comparison to

male respondents, discrimination had a smaller effect on female participants in regards to

mental health. The path from social support to substance use had a stronger effect for

women (p < .01). There was also a stronger effect for substance use on mental health for

men than women. No significant differences were observed for the other paths. See Table

17 for information on standard estimates, standard error, and significance of all paths

assessed in the study.

Table 17. Regression Weights for Paths for Men and Women

Paths			Men (N = 300) Standard Estimate	S.E.	P	Women (N = 107) Standard Estimate	S.E.	P
Discrimination	→	Substance Use	.340	.099	<.001	.152	.117	<.05
	→	Emotional Eating	.311	.104	<.001	.376	.108	<.001
	→	Social Support*	-.005	.146	.954	-.182	.143	<.01
	→	Physical Health	-.194	2.00	<.05	-.101	2.38	.157
	→	Mental Health**	.306	.043	<.001	.143	.042	<.05
Social Support	→	Emotional Eating	-.107	.054	.154	-.135	.048	<.05
	→	Substance Use**	-.208	.054	<.01	-.355	.059	<.001
	→	Mental Health	-.205	.021	<.001	-.223	.019	<.001
	→	Physical Health	.182	.986	<.05	.171	1.07	<.05
Emotional Eating	→	Mental Health	.346	.031	<.001	.317	.025	<.001
	→	Physical Health	-.228	1.44	<.01	-.188	1.40	<.01
Substance Use	→	Mental Health*	.382	.038	<.001	.359	.024	<.001
	→	Physical Health	-.276	1.68	<.001	-.220	1.32	<.001

Note: Significant differences in standardized estimates are denoted with * = p <.05, ** = p<.01, *** = p < .001.

Discussion

Impact of Discrimination on Health

The current study sought to better understand the impact of racial discrimination

on mental and physical health. This aim was to be completed by assessing the role of

emotional eating, substance use, and social support as mediators in the relationship

between discrimination and physical health and moderators in the relationship between discrimination and mental health. First, this study attempted to confirm the detrimental impact of discrimination on mental and physical health. Then, the coping variables were assessed as moderators and mediators. The coping variables were also assessed as mediators in the relationship between racial discrimination and three health maladies: hypertension, diabetes, and cardiovascular disease. Finally, the differences in the model fit based on African-Americans and Hispanics was assessed to provide more information race-based nuances of the role of discrimination on mental and physical health.

Racial discrimination in the current study was associated with increased reports of poorer mental health, measured as increased anger and depressive symptoms. The data collected from respondents support both direct and indirect relationships with mental health, based upon the significant and positive pathways between the racial discrimination and the mental health latent variables. These results add to a preponderance of literature supporting the detrimental impact of racial discrimination on mental health (e.g. Araújo & Borrell, 2006; Chakraborty, McKenzie, Hajat, & Stansfeld, 2010; Krieger, Kosheleva, Waterman, Chen, & Koenen, 2011; Noh & Kaspar, 2003). A meta-analytic study completed by Pascoe and Richman (2009) examined 110 studies of discrimination and the associated impact on mental health outcomes. Their results found no difference in race regarding the negative impact of perceived discrimination on mental health variables, such as depression, psychological distress, and general well-being. Overall, the results of the analyses in the current study focused on the impact of racial discrimination on mental health add to a large and consistent body of research indicating that exposure to discrimination leads to a worsening of mental health.

The role of emotional eating as a protective factor against the detrimental mental health effects of racial discrimination was assessed. Results indicate emotional eating strengthened the problematic influence of racial discrimination on reported depression and anger symptoms. As such, emotional eating may be engaged in as a protective measure for the target of racial discrimination's mental health, but ultimately is ineffective and serves to worsen symptoms. This finding is in conflict with previous research exploring emotion focused eating as a mental health protective coping measure for stressors, such as racial discrimination (Brodish et al., 2011; Gibson, 2012; Jackson et al., 2010; Raspopow, Matheson, Abizaid, & Anisman, 2013). However, at least one study supports links between poorer mental health and emotional eating. Emotional eating was found to occur in response to life dissatisfaction in an African-American population (Wickrama et al., 2012). The inference can be drawn that life dissatisfaction is accompanied by anger and depression symptoms. It may be that racial minorities experience racial discrimination and cope with negative affect through eating, but it only works in the short-term. In summation, the current study does not support emotional eating as a protective strategy for mental health, in response to racial discrimination, and conflicts with previous literature theorizing or supporting the protective role of emotional eating in the relationship between racial discrimination and mental health.

Theorized connections between racial discrimination, substance use, and mental health were assessed, with substance use proposed as a moderating factor. Results confirm that substance use moderated the relationship between racial discrimination and negative mental health outcomes, but not as a protective factor. High amounts of substance use increased the strength of the association between racial discrimination and

increased mental health symptoms. This would indicate that coping through substance

use led to a worsening of mental health associated with exposure to racial discrimination.

It may be that the negative consequences of substance use increase the likelihood of

additional mental health problems. Another theory may be that those who are

experiencing depression and anger seek out substances, in order to feel better, but this

only successful in the short-term or with continued use. This is corroborated by the extant

literature linking increased substance use, alcohol consumption, with more depressive

symptoms, especially for those who drink to cope (Holahan et al., 2003). In response to

minority stress, substance use was positively associated with mental health problems in

other stigmatized groups, such as sexual minority women and Latino sexual minorities

(Cochran et al., 2007; Lehavot & Simoni, 2011). Overall, substance use did not protect

against the mental health consequences of exposure to discrimination and instead

worsened the impact.

The impact of social support as a potential moderator was assessed to better

understand the complexity of mental health outcomes in relation to exposure to

discrimination. Despite the positive role of social support on mental health, no

moderation occurred regarding the impact of racial discrimination on mental health. This

is likely due to the insignificant relationship between racial discrimination and social

support identified in this study. There are likely a plethora of rationales for the failure of

social support as a moderator. One likely explanation is that social support can be

assessed in a multitude of ways and depending on which dimension is assessed there may

be differential effects. The current study assessed perceived availability of support from

various sources. However, without utilization of that support system and the access to

needed resources, it may not serve as a protective measure. Empirical inquiry has examined in more detail distinct domains of social support, such as social integration and emotional support, and potential interaction effects of different domains regarding race, socio-economic status, and gender (see Gorman & Porter, 2011; Uchino, Cacioppo, & Kiecolt-Glaser, 1996). The source of support as previously discussed can have differential effects. This study is consistent with a few studies, which do not support social support as a protective factor (e.g. Chakraborty et al., 2010; Prelow et al., 2006). A more comprehensive approach to assessing the role social support may be needed.

The assertions made by Jackson et al. (2010), which posited that African-Americans may engage in unhealthy coping behaviors, such as overeating and substance use, in order to combat the troublesome effects of exposure to racial discrimination on mental health, were partially supported in regards to discrimination. However this study found that substance use and emotional eating enhanced the effect of racial discrimination on mental health instead of dampening it. Therefore substance use and emotional eating did not service as a protective measure for mental health. This study provides a more complex understanding with the inclusion of social support and anger. However it appears that the availability of social support did not provide protection against the mental health repercussions of racial discrimination.

Another aim was to confirm and further explore the detrimental impact of racial discrimination on physical health outcomes. Racial discrimination related to poorer outcomes for reported physical health functioning. In the respecified model, discrimination was associated with more limitations in functioning and poorer perceptions of one's physical health, as measured by the SF-20 physical functioning

score. These findings are consistent with previous research identifying the negative

impact of discrimination on general physical health (Finch & Vega, 2003; Mays et al.,

2007; Pascoe & Richman, 2009; David. R. Williams et al., 1997). In a meta-analytic

study of 43 studies assessing the impact of racial discrimination on health, results

supported the robust role of discrimination in poorer health outcomes (Pascoe &

Richman, 2009).

The link between racial discrimination and poorer physical health has been well

researched; however, the mechanisms by which discrimination negatively influences

physical health have yet to be fully explored. Therefore, a major aim of the current study

is to add to the growing body of literature exploring whether coping through emotional

eating, social support, or substance use may alter the physical health consequences of

discrimination. Analysis of the data support the hypothesis that substance use and

emotional eating partially explain the relationship between mental and physical health.

Substance use and emotional eating attenuated the strength of the direct relationship

between racial discrimination and physical health and the indirect paths through both

mediators reached significance. The results of the analyses lend support to the previous

literature positing that substance use and emotional eating may be used as attempts to

cope with the detrimental impact of stressors, such as discrimination, on mental health,

but lead poorer physical health (Jackson, Knight, & Rafferty, 2010, Jackson, 2002). In

contrast to emotional eating and substance use, the relationship between racial

discrimination and social support was insignificant and as a result social support could

not act as a mediator.

This study sought to further explore the role of social support as a coping mechanism in emotional eating and substance use. Increased social support was posited to predict an increase in the engagement of emotional eating and substance use, as it was thought that these behaviors may be occurring in the context of receiving support from others. Although the paths from social support to emotional eating and substance use were significant, social support was related to a *decrease* in emotional eating and substance use. Current results provide further evidence of the impact of social support on eating behaviors, which is in keeping with previous literature that implicate negative social interactions as influencing eating behaviors. Tiller et al. (1997) found that individuals with disordered eating behaviors such as anorexia nervosa and bulimia have fewer social support reserves, than those without disordered eating. African-American focused research supports the role of social support as a mediator of the relationship between life dissatisfaction and unhealthy eating (i.e. poor nutrition and high-fat diets, Wickrama et al., 2012). Furthermore, those who tend to use an emotion focused coping style to stressors appear to be at a higher risk for engaging in problematic emotional eating (Raspopow et al., 2013). The current results are, therefore, consistent with the extant literature supporting problematic emotionally based eating was found to be related to low social support.

A stronger effect was found for relationship between social support and substance use, than emotional eating. Support was related negatively related to substance, meaning that as social support increased participants were less likely to engage in substance use. These results conflict with other literature, which identifies social support as a potential liability for engaging in substance use, especially for marginalized groups (Crawford et

al., 2013; Myers et al., 2009; Ornelas et al., 2011). There is high level of interest in

understanding the influence of various sources of support in adolescence in relation to

substance use. Therefore examining this literature base provides a more detailed

understanding of how differing sources of social support impact substance use. Increased

social support reduces the likelihood of substance use in adolescents and social support

also reduced the likelihood of substance use for some problematic attachment styles

(Caspers, Cadoret, Langbehn, Yucuis, & Troutman, 2001). Heavy marijuana and tobacco

users indicated less social support from friends and family than mild users (Averna &

Hesselbrock, 2001). More complex research evidences potentially differing effects for

alcohol use and sources of social support, where heavy alcohol users report higher friend

related social support than those who use less (Averna & Hesselbrock, 2001). For

adolescents peer social support was associated with an increased tobacco and alcohol use,

when other peers engaged in those behavior, but support from adults was inversely

related with use (Wills & Vaughan, 1989). This may help explain the moderate effect of

social support on substance use, given that peer support and parental support appear to

have differing effects for alcohol versus other substances. Similar results regarding the

differential impact of social support on substance use was found for adult substance

misusers. Social support from peers, children, significant others, and family was assessed,

in a study of men in substance abuse recovery, and support from children was the lone

source with a consistent positive influence on recovery and ending misuse (Kim, Davis,

Jason, & Ferrari, 2006)

        The analysis of the respecified model provided more in depth information on the

influence racial discrimination has on perceived physical health status of the respondents,

whereas, secondary analyses sought to assess the coping variables influence as mediators

in relation to racial discrimination and specific health maladies. Empirical research has

elucidated links between racial discrimination and the health diagnoses hypertension and

cardiovascular disease (e.g. Brondolo et al., 2011, 2009; Krieger, 1990; Krieger et al.,

2008; Lepore et al., 2006; Szanton et al., 2012). Extant literature has also proposed

associations between exposure to demeaning race-based experiences and increased risk

for developing diabetes (Mattei et al., 2010; Qiao et al., 2013; Szanton et al., 2012).

Although there have been numerous hypothesized connections between them, the current

study serves to provide more information on the impact of potential mediating variables,

such as substance use, social support, and emotional eating.

The Role of Mediators in Specific Health Maladies

   Hypertension (High blood pressure). The first set of secondary analyses

examined the role coping variables as mediators in the relationship between racial

discrimination and the reporting of a diagnosis of hypertension or high blood pressure.

The current study did not support the role of substance use as a mediator between

exposure to racial discrimination and high blood pressure. Results in the extant literature

mirror this work and suggest that the direct linkages between substance use (with a

primary focus on alcohol and nicotine usage) and hypertension appear to be tenuous.

Nicotine has been associated with short-term increases in blood pressure, but when

controlling for body mass index, it appears that it does not lead to developing persistent

hypertension (Halimi et al., 2002). However some research does support links with

hypertension, smoking in women has been associated with a dose response, with heavy

smokers having much higher risk for developing hypertension (Bowman, Gaziano,

Buring, & Sesso, 2007). It remains unclear as to whether smoking leads directly or indirectly, in conjunction with other risk factors such as obesity, to worsen hypertensive outcomes. The association of alcohol use with developing high blood pressure is also diffuse. In fact, some alcohol use was associated with a reduction in blood pressure for some groups and no link was found for the other groups (Halanych et al., 2010). In contrast, the current study found a significant association between blood pressure and substance, but substance use did not fully nor partially explain the relationship between racial discrimination and reported diagnosis of high blood pressure.

Social support did not have an impact on the relationship between racial discrimination and reports of hypertension. The existing literature regarding social support as a mediator between stress and health, including hypertension, is mixed. There is paucity of literature assessing the impact of social support as a mediator in the relationship between racial discrimination and hypertension. Examining more general research focused on the role of social support protective measure against the negative effects of race-based disparate treatment on hypertension. Strogatz et al. (1997) measured differences in systolic and diastolic blood pressure in African-Americans based on stress and social support and found that for the high stress and low social support group experienced increased blood pressure. As a result, it was posited that chronic exposure to high stress with weak emotional support could lead to hypertension (Strogatz et al., 1997). There is also evidence which suggests that emotional support does not have a significant impact on hypertension in rural African-Americans (Strogatz & James, 1986). In this same study low access to instrumental support was related to higher risk for hypertension in African-Americans (Strogatz & James, 1986). It may be that without

access to resources that may promote better access to healthcare social support will not

serve as a protective measure against the impact of discrimination on the likelihood of

developing hypertension. In the current study, the focus was on the role of availability of

social support and instrumental support was not assessed.

Emotional eating partially mediated the relationship between the discrimination

and high blood pressure. Coping with exposure to discrimination by emotional eating

partially explained the likelihood of reporting a diagnosis of hypertension. Although not

assessed in the current study, diet may play a significant role in the emotional eating and

mediate the relationship between emotional eating and physical health. When participants

engage in emotional eating resulting from a perceived stressor, such as discrimination, it

is probable they are consuming foods high in fat, have an abundance of salt, and lacking

in nutrition (Brodish et al., 2011; Hickson et al., 2012; Jackson et al., 2010; Schiffman et

al., 2000; Torres & Nowson, 2007). A diet of this nature is significantly correlated with

the development of high blood pressure and exposure to racial discrimination (e.g.

Manuel, 2004; McCann et al., 1990; Qiao et al., 2013). Therefore exposure to

discrimination, as a stressor, may also likely lead to the cravings for salty, sugar-laden

foods, which is supported by allostatic load and the related research on linking dietary

changes with environmental hassles, which has also been seen in animal models (Deuster

et al., 2011; Hagan et al., 2002).

Overall, emotional eating was the only coping variable, which partially explained

the link between racial discrimination and hypertension. Social support and substance use

did not help explain the impact of racial discrimination and hypertension. The results of

these analyses provide valuable information on the ways in which racial discrimination may or may not lead to hypertension.

       Diabetes. Secondary analyses also sought to better understand the role of emotional eating, social support, and substance use as mediators in the relationship between discrimination and participants' report of a diagnosis of diabetes. The relationship between exposure to racial discrimination and report diabetes was not significant, after controlling for age, race, and gender; therefore the coping variables could not act as mediators. Although there have been theorized links between racial discrimination and diabetes, there is little evidence linking discrimination directly to the development of diabetes. However, one potential pathway for discrimination to lead to diabetes in older age, where a common factor in response to stressors, red blood cell oxidative stress, was found to be elevated in African-Americans, but not Caucasians (Szanton et al., 2012). It may be that the sample (mean age of 30) was not old enough to detect the development of diabetes in response to discrimination. An additional explanation may be that when covariates such as age and gender better account for the observed reports of being diagnosed with diabetes in this dataset.

       Cardiovascular disease. The relationship of exposure to discrimination and increased risk for cardiovascular disease has strong support within the literature base (e.g. Brondolo et al., 2003; Cardarelli et al., 2010; Krieger, 1990; Krieger et al., 2008; Mattei et al., 2010; Szanton et al., 2012). To this end, the current analyses examined the role of the coping variables, substance use, social support, and emotional eating, as mediators in the path between exposure to racial discrimination and cardiovascular disease. The relationship between substance use and reporting of cardiovascular disease was not

significant. Nicotine, substance use, and other drug use were combined in a composite

variable to assess the effects of substance use as a mediator. The combination of these

multiple substances may obscure the influence of individual substances. Smoking and

nicotine use have strong associations with poorer outcomes for cardiovascular health,

especially the younger the age of onset (Halimi et al., 2002; U.S. Department of Health

and Human Services, 2004). Smoking has been associated with a dose response, with

heavy smokers having much higher risk for developing cardiovascular disease (Bazzano,

He, Muntner, Vupputuri, & Whelton, 2003; U.S. Department of Health and Human

Services, 2004; Wilson et al., 1998).

The association of alcohol seems to be complex. In moderation alcohol has been

associated with better cardiovascular and heart functioning, whereas heavy use is

associated with more incidences of disease (Friesema et al., 2007; Hvidtfeldt et al., 2008;

Vogel, 2002). Differing effects have been observed for wine, as compared to other forms

of alcohol (Vogel, 2002). The combination of smoking and alcohol into one predictor

may have obscured differences in the choice of substance and choice of alcohol. The

relative amounts of consumption were not assessed, it may have been that this sample

used alcohol in moderation, which can be cardio-protective. The number of participants

with cardiovascular disease was small, which is likely related to the relatively young

average age of participants.

Social support did not have help explain on the relationship between racial

discrimination and report of cardiovascular disease. Although some research supports the

role of social support as a mediator in the relationship between stressors and

cardiovascular disease (Uchino et al., 1996), there is also evidence which suggest that

emotional support does not have a significant impact on cardiovascular health outcomes
(Gorman & Sivaganesan, 2007). Availability of support may not be enough to fully or
partially explain the relationship between discrimination and health outcomes such as
cardiovascular disease.

Emotional eating partially mediated the relationship between the discrimination
and cardiovascular disease. In addition the effects are still significant even when
controlling for gender, despite the empirical literature supporting the assertion that
women may engage in emotional eating behavior more than men. The explanation for
these results likely mirrors that of the explanation of the mediating role emotional eating
in the relationship between racial discrimination and hypertension. Chronic poor diet
likely explains the partial role of emotional eating leading to poor cardiovascular health,
due to increased cravings for foods poor in nutrition (Brodish et al., 2011; Hagan et al.,
2002; Manuel, 2004). Previous research has confirmed that a poor diet as modifiable risk
factor for cardiovascular disease (Canto & Iskandrian, 2012; Gyárfás, Keltai, & Salim,
2006).

Emotional eating was the sole coping variable, which partially explained the link
between racial discrimination and cardiovascular disease. Social support and substance
use did not help explain the impact of racial discrimination and cardiovascular disease.
This study provides essential information on at least one pathway in which racial
discrimination may lead to cardiovascular disease.

Race/Ethnic Differences in the Model.

The impact of racial discrimination on health has been chiefly studied in African-
Americans, with much less focus on Hispanic Americans. A meta-analytic study by

Pascoe and Richman (2009) examined over 150 studies examining the impact of

perceived discrimination on mental and physical health and they identified a paucity of

research regarding other minority races besides African-Americans. In order to provide

additional information on any race based differences regarding the impact of racial

discrimination, the respecified model was examined for significant differences in race.

Overall model fit differences by race/ethnicity suggest differential relationships

among the variables of study. Given this is the case African-American and Hispanic

Americans appear to experience differing responses to discrimination, which are likely

culturally bound styles of coping, leading to a differential impact on mental and physical

health. Social support was not significantly associated with emotional eating in African-

Americans, but was meaningfully related linked in Hispanic participants. Conversely,

social support was significantly related to reporting of anger and depressive symptoms

for African-Americans, while this was not true for Hispanics. Culturally bound

differences in the ways in which social support is expressed and therefore serves a

protective measure, likely accounts for the observed differences (Gorman & Porter,

2011). Discrimination predicted change in physical health for Hispanic Americans, but

not African-Americans. This finding is contrary to previous literature supporting poorer

health outcomes in response to increased discrimination for African-Americans. A meta-

analytic study found that only 42% of the analyses from 43 studies supported a

significant link between increased exposure to discrimination and poorer physical health,

a large proportion of these studies examined African-American physical health. (Pascoe

& Richman, 2009). However the authors found the linkages between discrimination and

poorer physical health to be robust. One explanation for the finding in this study may be

that African-Americans did not experience significant changes in role functioning or self-reported illness, but the detrimental health impact of discrimination manifested in other, less physically limiting ways, such as hypertension and cardiovascular disease. More discrimination was associated with a greater likelihood of reporting these health maladies, even when controlling for race.

Hispanic Americans experienced poorer physical health and mental in response to discrimination. Although discrimination significantly impacted mental health for both groups, discrimination was more strongly associated with more mental health symptoms in Hispanic participants. Therefore Hispanics may experience a larger detriment to their mental and physical health than African-Americans. This may be due to differences in cultural patterns of somatization of stress and psychological symptoms (Consedine et al., 2006; Rao, Poland, & Lin, 2012). Sadness was associated was linked with a greater report of physical symptoms in Caribbean men, but not Black-English speaking Caribbeans (Consedine et al., 2006). Similarly, research suggests race-based differing patterns of symptom expression, where Mexican-Americans exhibited more anxiety and somatic symptoms (Rao et al., 2012). These differences in expression of psychological symptoms may help explain disparate findings regarding race in the population from which this data was derived.

Differences in the strength of other relationships within the model were also present. Discrimination was more strongly connected with emotional eating in African-Americans, compared to Hispanics. Hispanic participants' mental health appeared to suffer more strongly from exposure to racial discrimination by comparison. In contrast, African-American respondents reported a stronger relationship of increased anger and

depression as substance use increased. Based on these results, race is a moderator

regarding the fit of the model and several of the relationships within the model.

Gender Differences in the Model

The model is a better fit for women than men, based upon the examination of the fit

indices. There were several significant path differences observed in this study.

Discrimination held a stronger relationship with poorer mental and physical health for

men than women. Discrimination was unrelated to physical health for women. Exposure

to discrimination was unrelated with the amount of perceived social support for men, but

was related to decreased support in women, which may highlight the importance of social

support for women. Less perceived support was more strongly linked with a higher

likelihood of substance use for women than men. It appears that social support may play

more important role for women than men. Previous research indicates there may be

differences in the ways in which social support impacts health differently for men and

women (Gorman & Porter, 2011). Substance use held a stronger relationship with poorer

mental health for men compared to women. Extant literature suggests mixed results

regarding gender differences in health behavior related coping for men and women in

response to discrimination. For example, some studies have found that gender did not

moderate the relationship between discrimination and substance use (e.g. Borrell, Diez

Roux, et al., 2010), while others have (e.g. Wiche, et al., 2010; Brodish et al., 2011).

Results of this study are consistent with previous literature identifying differences in

coping based on gender, in reference to health related behaviors, especially that men are

more likely to engage in substance use (e.g. Brodish et al., 2011). Overall the current

study lends evidence to the gender differences in the experience of and coping with

discrimination.

Limitations

There are several limitations to this study. This study was collected online and

participants were provided with possible incentives to completion. Evidence suggests that

there are no significant differences between online samples and samples collected in

person, including the relatively new data collection method MTURK (e.g. Buhrmester,

Kwang, & Gosling, 2011; Gosling, Vazire, Srivastava, & John, 2000; Krantz & Dalal,

2000; Miller et al., 2003). However, this study may have excluded participants who are

less likely to use online data collection tools. Structural equation modeling was used to

assess the paths within this study; however, SEM does not fully prove causation

regarding the variables assessed (Byrne & van de Vijver, 2010). SEM provides

correlational information and the bidirectional versus unidirectional nature of the

relationships cannot be full determined. SEM may be able to rule out some plausible

possibilities if they are inconsistent with the data, but other possibilities not included in

the model may exist. Data was also collected in a cross-sectional nature, which does not

provide potentially important information on the course of developing illnesses or

worsening of mental and physical health and would assist in better determining causality.

Another possible limitation of the current study is the overall young age of the

sample, mean was approximately 30 years of age, this may obscure health related

findings, such as cardiovascular disease and diabetes, which may not develop until later

in life. In addition, the negative health effects of substance use and emotional eating may

be more pronounced as age increases. The young age of the sample may also limit the

generalizability of this study, in regards to older cohorts of African-Americans or

Hispanic Americans. In addition age was not controlled for in the main analyses is likely

a limitation. The discrimination measure assessed the frequency of exposure to

discrimination over the lifespan and ended with four or more times as the highest option.

It is unlikely that an 18-year-old participant and a 65-year-old participant will experience

the same quantity of discrimination and older participants may experience a much higher

amount than four times. Therefore, without controlling for age there may be age related

differences in exposure to discrimination that were not accounted for in the main

analysis. Only participants who identified as African-American or, Hispanic were

included in the study, so the current results may not be reflected of other minority

experiences and coping with discrimination (e.g. Asian-Americans). This inquiry

examined only a few coping strategies that likely impact the development of health

disparities in relationship to racial discrimination. This study assessed social support

through the availability of support from several sources. Previous research indicated that

interrelationships with social support and the method in which it is measured can have

substantial impact on whether it serves as mediating or moderating factor (e.g. Gorman &

Porter, 2011; Strogatz & James, 1986; Uchino et al., 1996). It may be that assessing

social support through availability was insufficient to understand full role of social

support.

Future Research

Future research should seek to confirm the aforementioned results with respect to

older age, race, and other mediational variables. Furthermore the current set of analyses

did not examine the complex relationship in regards to the mediational variables, such as

differing trends regarding the health impacts of differing substances and the differing

types of social support. More research is needed to further develop the understanding of

these constructs in relation to coping with discrimination. Although the analyses of

respecified model and the health variables adds a degree of complexity to the

understanding of the interrelationships between exposure to discrimination and health

(mental and physical), mental and physical health status remains a complex concept,

which has a multitude of social, biological, and environmental determinants. Additional

research assessing the impact and interaction amongst these determinants is warranted.

Given the observed differences between African-American and Hispanic respondents,

more research is necessary to better understand the effects of discrimination and

subsequent coping among Hispanic populations. The current model was not a good fit

and future research should seek to better refine this model for them. Additional research

should also seek to further explicate gender differences regarding experiences with

discrimination and subsequent coping strategies. This study being completed in a cross-

sectional nature, future longitudinal studies would likely be beneficial in further

elucidating the development of mental and physical health maladies, in response to

discrimination and the impact of emotional eating, substance use, and social support.

Conclusions

Overall the coping variables assessed had varied impact on the mental and

physical health effects of exposure to racial discrimination. Discrimination was directly

associated with poorer mental and physical health, with exception to diabetes. Social

support, as assessed in this study, did not mediate nor moderate the relationship between

racial discrimination and mental health nor the health variables (hypertension, diabetes,

and cardiovascular disease). Emotional eating mediated the relationships discrimination held with mental health, self-reported physical health, hypertension, and cardiovascular disease. Substance use moderated the effects of discrimination on mental health and partially mediated the relationship between discrimination and physical health, but not the specific health diagnoses. There were significant differences in the coping variables in response to exposure to discrimination, which led to a detrimental impact of the model fit for Hispanic Americans. Overall, this study provides evidence as to the impact of discrimination on mental and physical health for African-Americans and Hispanic Americans and a more complex understanding of the interplay of attempts to cope. Future research should seek to refine these results for other racial groups, including Hispanics.

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