Kaiser, Kathryn Ann, Is All Fat Created Equal?: Assessing Self-Reported Obesity Risk between Racial Groups with Structural Equation Modeling of the Eating Inventory. Doctor of Philosophy (Biomedical Sciences), May, 2009, 89 pp., 15 tables, 7 figures, references, 126 titles.

Background: Obesity prevalence rates differ widely between gender and racial groups in the United States. African American women have the highest prevalence rates, while Caucasian women have the lowest rates. Better assessment methods are needed to discern the varied and complex biopsychosocial risk factors for this disease for each individual. Psychological measures must be tested for cultural validity in the populations in which they will be used. One commonly used measure of eating behavior, the Eating Inventory (Stunkard & Messick, 1988), has not been assessed for cultural bias between the major ethnic groups in the Unites States in terms of its validity for assessment of obesity risk.

Methods: Structural equation modeling techniques are used to compare invariance of factor structure of the Eating Inventory between adult Caucasian (n=110) and African American women (n=100). Alternate proposed factor structures are also tested for factor invariance between groups. Body Mass Index (BMI) is used as an optional covariate in the models. Additionally, socio-economic status indicators are examined for differential influence in the models, due to previously demonstrated, highly positive correlation with rates of obesity. Experimenter/participant race dyads are examined for systematic influence on response patterns.

Results: The factor invariance of the original, three factor structure (Cognitive Restraint, Disinhibition and Hunger) of the Eating Inventory is supported with this sample. The invariance of alternate factor structures could not be supported with this sample, with one exception (Cognitive Restraint). Relative covariance of factors with BMI appears to be consistent between groups for all models tested. No participant/experimenter race interaction with factor scores was observed.

Conclusions: The original three factor structure of the Eating Inventory appears to have invariance of the factor structure between the two groups measured, implying cultural validity. The relative covariance of factor scores with BMI is equivalent between groups. However, alternate factor structures could not be fully assessed due to sample size limitations. Further research is needed to replicate and expand these findings.

IS ALL FAT CREATED EQUAL?: ASSESSING SELF-REPORTED OBESITY RISK BETWEEN RACIAL GROUPS WITH STRUCTURAL EQUATION MODELING OF THE EATING INVENTORY

DISSERTATION

Presented to the Graduate Council of the

University of North Texas

Health Science Center at Fort Worth

in Partial Fulfillment of the Requirements

For the Degree of

DOCTOR OF PHILOSOPHY

Kathryn Ann Kaiser, B.S.

Fort Worth, Texas

May 2009

ACKNOWLEDGEMENTS

In memory of Dr. Jerry McGill, Ph.D.

My parents: Carol and Owen Kaiser, for their sustaining love and support

My committee:

Dr. Susan Franks, Ph.D. - Chair
Dr. Matthew Avila, Ph.D.
Dr. Vicki Nejtek, Ph.D.
Dr. Craig Neumann, Ph.D.
Dr. Adam Smith, D.O., F.A.C.O.S
Dr. Michael Smith, Ph.D. – University Member

Assistance with data collection: Joella Morris, School of Public Health

Assistance with recruitment: Sybil Allen; Folasade Asekun, Nurse Practitioner, JPS Women's Health Clinic, Arlington; Kathy Campbell, MSW – Vitas Hospice; Andrea Harmon – Salons at the Centrum; April Kyle – Jade Salon; City of Fort Worth, Martin Luther King, Jr. Community Center – Paula Jackson (Director) and staff; South Dallas Cultural Center – Victoria Meek, Director; Pastor Jack and Mrs. Regenia Crane, Carol Aidoo and LlaBetta Sowells – Truevine Missionary Baptist Church; Judy and Grant Johns – Curves on Camp Bowie; Grant Whittenburg, Janet Whitten, Sherry Pope, Becky Diefenderfer and Principal David Priddy – Burleson Independent School District; University of North Texas Cultural Diversity Center – Cheylon Brown, Director; NORTEX Primary Care Research Network - Dr. Roberto Cardarelli, D.O., Director, Dr. Kim Fulda, Dr.PH and Anna Espinoza, M.D.

Proofreading: Karin Vaught, M.A.

TABLE OF CONTENTS

LIST OF TABLES

LIST OF FIGURES

CHAPTER I

INTRODUCTION: UNDERSTANDING OBESITY

It is important to understand the complexity of biopsychosocial processes that have been studied in obese persons and why obesity is so difficult to treat. This investigation examines two groups of women (African American and Caucasian) that have significantly different prevalence rates of obesity, not to find out what is “wrong” with one group or “more right” with another. Rather, the study seeks to evaluate a tool commonly used to measure eating behavior for invariance of the factor structure between two groups that have significant differences in obesity prevalence rates.

Psychological measures must be tested for cultural bias before they can assume to be measuring the same constructs in the individuals from different groups being measured. A secondary purpose of this study is to compare the relationship of these factors to body composition and aspects of socio-economic status. With appropriate tools of measurement, the evaluation and treatment of obesity will be improved for all who seek treatment as individuals within their own unique environments.

Scope of the Problem

Obesity is a growing epidemic, not only minority groups in the Unites States, but throughout the developing world (Kumanyika, 1994; Ogden et al., 2006; Ogden, Yanovski, Carroll, & Flegal, 2007). Along with the increasing prevalence rates are the increasing associated medical costs with the various comorbidities such as diabetes, cardiovascular disease, hypertension, and sleep apnea (Ogden et al., 2007). Minorities in the United States are disproportionately affected by most of these comorbidities (Kumanyika, 1993; Kumanyika, 1994). See Figure 1 for gender and racial prevalence rates of overweight (BMI > 25) and obesity in the United States (Centers for Disease Control, 2006; Ogden et al., 2006).

Definitions and Causes of Obesity

While there are newly proposed systems for classifying obesity to include weight status, functional limitations and comorbidity risk (Sharma & Kushner, 2009), the current World Health Organization classification system defines the various classes of obesity solely according to body mass index (BMI – a ratio of kilograms divided by height in meters squared): Class I Obesity = BMI ≥ 30 & <35; Class II Obesity = BMI ≥ 35 & <40; and Class III Obesity = BMI ≥ 40 (World Health Organization, 2009). Obesity is a heterogeneous disorder of multifactorial origin (Brownell & Wadden, 1992). Its forms are sometimes referred to as "obesities" to reflect the variety of manifestations (Bouchard, 1996). While there are some distinct monogenetic forms, most obesities reflect complex biopsychosocial factors that change significantly over the lifespan (Grilo, 2006).

The fundamental energy balance equation is: net balance is equal to the amount of energy intake minus the energy expended (Williams & Considine, 2001). The relative proportions of energy expenditure are approximately: 60% resting activities (life support functions); 30% physical activities (movement); and 10% thermic effect of food, that is, energy required to process food (Weinsier et al., 1995; Williams et al., 2001). The resting energy expenditure is sometimes called "Resting Metabolic Rate" (RMR). This proportion of energy expenditure is primarily a function of fat-free mass, but RMR is greater in obese people (Williams et al., 2001). It is generally agreed that excess energy intake *in combination with* reduced activity is the most important determinate of weight gain (Weinsier, Hunter, Zuckerman, & Darnell, 2003).

Energy expenditure and intake are coordinated within the central nervous system by several nuclei in the hypothalamus (Schwartz, Woods, Porte, Jr., Seeley, & Baskin, 2000; Williams et al., 2001). Central and peripheral neuroendocrine signaling mechanisms form a complex network of communication to respond to food intake and energy demands, at the same time managing energy stores in the form of fat. Studies of people who exhibit a high degree of gene-related obesity have revealed the points of dysregulation within this neuroendocrine network, with significant influence from the environment. This environment includes thoughts and emotions.

Genetics and the Environment

The Pima Indians of Arizona have been studied closely for their extremely high rates of obesity and subsequent comorbid conditions. The original tribe lived in the Sonora desert in Mexico until the mid 19th century, when European immigration and land occupation forced a segment of the tribe to migrate north to Arizona. This "American" branch of the tribe has been exposed to distinct environmental and cultural changes in the past few generations that the Mexican branch of the tribe has not (Ravussin, Valencia, Esparza, Bennett, & Schulz, 1994).

While the Mexican tribe members have maintained a similar environment and lifestyle with little incidence of obesity, the American Pimas have suffered a significant increase in obesity prevalence after the change to a different diet and activity level (Esparza et al., 2000). Studies of men and women in the two segments of the Pima tribe have shown two key observations pertinent to the present study: 1) lower physical activity increases risk for obesity in men but not women, and 2) there are gender and genetically-linked differences in energy expenditure from physical activity (Esparza et al., 2000). Women burn less energy than men at rest and at play. In Pima Indians, there is support for the concept of a "thrifty gene" that is at high risk in an obesogenic environment. Both men and women Pimas in the United States have similar rates of highly heritable obesity, and evidence of male-linked neuropeptide receptor variations exists (Ma et al., 2005). In contrast, African American men and women do not share similar prevalence rates (Figure 1). However, the inference of a gene-gender-environment interaction that further increases risk in African American women has not been studied comprehensively.

Another highly genetic form of obesity, Prader-Willi syndrome (PWS) results from a deletion of paternally inherited genes on chromosome 15. This leads to insatiable hunger and obesity from early childhood with accompanying developmental brain defects, particularly of the hypothalamus (Goldstone, 2006). While the obesity seen in PWS is clearly a result of a central dysregulation, the wide variety of symptomatology exhibited in the afflicted have illustrated the importance of environmental and developmental influences on the etiology of this form of genetic obesity, even with the presence of a distinct genetic disadvantage. Indeed, identical twin studies have shown polygenic causes for obesity in most cases. Based on these studies, it is estimated that approximately 33% of obesity is directly linked to genetics alone, with the remaining being a result of the environment interacting with many different genes (Stunkard, 1996). This environmental influence has been shown to begin *in utero* (Stunkard, 1996; Williams et al., 2001). However, the complex genetic and environmental influences (known as "epigenetics") contributing to obesity are not yet understood (Grilo, 2006).

Other Biological Influences

As previously highlighted in Figure 1, there are some striking differences in prevalence rates of overweight/obesity between the gender and racial groups in the United States. Closer examination of the rates of African American women versus African American men rules out a simple, racially-associated cause due to genetics or shared environment (Kumanyika, 2008). There must be a gender and race interaction if one can assume relatively similar living and dietary conditions (Kumanyika et al., 2005). As of 2004, for African Americans the gender gap of body mass index is 12.5%; for Caucasians it is 12.6%. While the gap is similar, Caucasian men have the higher prevalence than Caucasian women; African American women have the higher prevalence over African American men. The gap between genders within the Mexican-American population is very small (0.6%).

Social Factors

Social factors must be considered among the most important influences on the current prevalence of obesity. Many researchers view obesity to be under the control of the social environment (Stunkard, 1980). Since the relationship of socio-economic status (SES) to obesity has been frequently observed to be highly inverse (Stunkard, 1996; Stunkard & Sorensen, 1993), this phenomenon has been investigated for specific causal links which have not been established. For developed societies such as the United States and Western Europe, this strong inverse relationship is true for women, but is inconsistent for men and for children of either gender. In societies that are in the developmental transition, such as some countries in Eastern Europe and Northern Africa, there is a direct, positive relationship between obesity and SES for women, men and children (Sobal & Stunkard, 1989). Factors theorized to be associated with higher SES women and their attitudes towards thinness in developed societies include dietary restraint, more leisure time for recreational physical activity, social mobility and financial resources (Sobal et al., 1989). However, more recent data indicate that women in the upper SES range have increased in BMI at a faster rate than lower SES women to the point where the strong linear relationship between BMI and SES is no longer statistically significant (Zhang & Wang, 2004a; Zhang & Wang, 2004b).

Dietary Quality

Very closely tied to SES and obesity are theories that dietary quality has a strongly inverse relationship with obesity. However, the studies examining systematic differences in quality and quantity of food have been equivocal in terms of explaining large effects between SES groups by this factor alone (Kumanyika, 2002; Kumanyika, 1993; Kumanyika, 1994; Yancey et al., 2004) . Various studies have evaluated the geographical locations of supermarkets as compared to neighborhood grocery stores in low and middle-income, urban neighborhoods (Moore & Diez Roux, 2006; Powell, Slater, Mirtcheva, Bao, & Chaloupka, 2007; Zenk et al., 2005; Zenk et al., 2006). Generally, there are fewer chain supermarkets, less variety and decreased availability of fresh fruits and vegetables in lower SES neighborhoods, along with a significantly higher number of liquor stores. While a greater proportion of participants in the WIC and Food Stamp Programs are in lower SES neighborhoods, less variety of healthier food choices further increases the likelihood of choosing more highly processed and energy dense foods (Zenk et al., 2006).

The relationship between obesity and the Food Stamp Program (FSP) had been studied, and there was a period when this positive relationship was significant for women only (Devaney & Moffitt, 1991; Gibson, 2003). However, there are many methodological issues with the cross-sectional nature of the available data and lately, this relationship is considerably weaker. In fact, while the increase in BMI of FSP non-Hispanic white women was faster in the period between 1976 and 1988 as compared to eligible non-participants or higher income women, this trend has changed. The National Health and Nutrition Examination Survey (NHANES) data from the 1988-1994 interval to the 1999-2002 NHANES data set indicates the BMI increase of FSP non-Hispanic white women leveled while the eligible non-participants and higher income groups continued to increase (Ver Ploeg, Mancino, & Lin, 2006). The latest data indicates women FSP participants are not getting relatively heavier over time. While the 1999-2002 NHANES data showed that male FSP participants were least likely to be obese, more recent data show that differences between all groups are disappearing (Ver Ploeg et al., 2006).

While some would blame the soft drink industry for having the largest single influence on obesity rates in the United States (Bray, Nielsen, & Popkin, 2004; Malik, Schulze, & Hu, 2006), it is difficult to apply that as a significant cause in every person's case. However, the transition to increased use of high fructose corn syrup (HFCS) as a sweetener in soft drinks and processed foods mirrors the overall trends in obesity in the last quarter of the 20th century (Bray et al., 2004). Estimates of the consumption of HFCS indicates a daily average of 132 kilocalories for all Americans aged two years or older, and the top 20% of consumers of caloric sweeteners average an intake of 316 kilocalories from HFCS per day (Bray et al., 2004).

Race

Other factors related to the strong race-SES-obesity relationship include fetal nutrition and early development, but research findings are mixed as to any strong epigenetic effects in one group over another. Overall, there are more than 600 genes, markers, and chromosomal regions associated or linked with human obesity phenotypes (Perusse et al., 2005). While all of these genes have not been investigated for prevalence differences between races, it is generally accepted within the entire human genome that there is more intra-racial genetic variation than interracial variation (National Research Council Panel, 2004).

Since obesity is practically non-existent in Africa, it is difficult to attribute a strong genetic cause of obesity in African Americans outside of environmental influence. The epigenetic phenomenon that has been observed in Pima Indians may be similarly at work in the African American population. African American women are at a distinct disadvantage when current cultural conditions are compounded with other factors that further increase risk, such as lower SES. Studies that have controlled for SES when comparing African American women to other groups of women show that a significant increased risk for obesity still exists (Caldwell, Brownell, & Wilfley, 1997).

Additionally, while many popular perceptions are that African American men prefer larger body shapes in women, studies do not consistently support that African American women have a similarly larger body shape preference for themselves (Cachelin, Rebeck, Chung, & Pelayo, 2002; Caldwell et al., 1997; Rosenberger, Henderson, & Grilo, 2006). Meta-analyses of body shape preference studies in male-dominated societies have not shown that female body shape preferences in males have a significant effect on female body shape self-ideals (Anderson, Crawford, Nadeau, & Lindberg, 1992). It may be that underlying normative beliefs and attitudes are not being accurately measured, so this question deserves further investigation.

Mood States and Stress Influences

In addition to social norms, eating behavior can be strongly influenced by mood states. Certain moods can have a “disinhibition” effect, meaning that if one is usually able to avoid eating certain foods or eating larger amounts than normal, normal inhibition can be lost during mood states such as depression, anxiety or mania. While depression can either increase or decrease appetite (Weissenburger, Rush, Giles, & Stunkard, 1986), it is rare for males to experience an increase in appetite – depression-associated increased appetite is most often found in females (Paykel, 1977). Stress and anxiety can also have disinhibiting effects on eating behavior (Stunkard, 1980).

A proposed biopsychosocial model of racism as a stressor for African Americans (Clark, Anderson, Clark, & Williams, 1999) states that an exaggerated psychological and physiological stress response that results from an environmental stimulus perceived as racist is influenced by socio-demographic, psychological, behavioral and coping factors. These factors all moderate the impact on health, according to the model. Postulated health outcomes of perceived racism include depression, increased substance abuse, increased cardiovascular reactivity, and overeating (Browman, 1996; Clark & Harrell, 1982; Fernando, 1984; Johnson & Browman, 1987).

Psychological Constructs that Affect Eating Behavior

There are two long-standing theoretical constructs that describe the efforts we make to either control our intake or our response to losing control of eating. These constructs have been termed "Cognitive Restraint" and "Disinhibition", respectively (Stunkard & Messick, 1985). Whether a person normally has a high or low amount of each of these characteristics determines the relative risk of excessive eating (Stunkard et al., 1985; Stunkard & Messick, 1988). These constructs are part of a questionnaire known as the Eating Inventory (originally published as the Three Factor Eating Questionnaire, or TFEQ), (Stunkard et al., 1985)

Recent research indicates that there are individual differences in the experience of hunger and food cravings (White, Whisenhunt, Williamson, Greenway, & Netemeyer, 2002). Cognitive Restraint normally serves to help avoid cues and opportunities to overeat or eat high calorie foods. However, evidence shows that those high in Cognitive Restraint are at greater risk for "relapse", depending upon the corresponding degree of Disinhibition (Bond, McDowell, & Wilkinson, 2001). Disinhibition refers to the loss of control one experiences when a self-imposed boundary is violated. Both of these constructs have not been fully explored in any prior studies focusing on obesity risk with regard to potential differences in interpretation of these concepts between racial or ethnic groups.

Summary

This brief introduction serves to highlight the complexity of the obesity problem in the United States. There are no clear, simple answers for the questions that have been raised in previous examinations of this issue. However, it is becoming clearer that the techniques and tools used to investigate this problem require continuous improvement to elucidate meaningful data. Epigenetics, nutrition, and social factors have proven difficult to control and compare across studies. While the methods to examine these causes are improving, it is also important to improve the measurement of eating behavior. This key component of the energy balance equation cannot be ignored as a therapeutic target.

This study is an important first step in answering these questions. By performing an assessment of cultural validity of one of the most commonly used instruments used to assess eating behavior (the Eating Inventory) between the two groups at the extremes of the prevalence rates of obesity in the United States, the results of this study illuminate the next steps to investigating the psychosocial contributions to the obesity epidemic for all groups affected.

To further this effort, the study addresses the following research questions:

1. *Is the Eating Inventory a culturally valid measure for obesity risk in African American women?*
2. *Is there a factor structure or alternate item set of the Eating Inventory that would reveal any behavioral contribution as to why African American women are at a much higher risk for obesity than Caucasian women in the United States?*

Several studies have attempted to replicate or expand upon the original factor structure of the EI on varying population samples (Bond et al., 2001; Ganley, 1988; Mazzeo, Aggen, Anderson, Tozzi, & Bulik, 2003; Ricciardelli & Williams, 1997; Westenhöfer, Stunkard, & Pudel, 1999). One that investigated only the Cognitive Restraint factor did so by adding more questions to the original list to improve discrimination between two theoretically proposed types of restraint, termed “flexible control” and “rigid control” (Westenhöfer et al., 1999). Another more recent examination proposed that each of the original factors had two or three sub-factors

(Bond et al., 2001). While the validity and generalizability of these theoretical expansions of the original EI have not been established, they each merit further study. The above questions are addressed in this investigation by testing the following null hypotheses:

Hypothesis 1: The factor structure for the original, three-factor Eating Inventory will be statistically equivalent between racial groups.

Hypothesis 2: The factor structure for an alternative, two-factor Cognitive Restraint model will be statistically equivalent between racial groups. (Fourteen additional questions will be added to the Eating Inventory Cognitive Restraint questions to form the Flexible Control factor and Rigid Control factor.)

Hypothesis 3: A proposed sub-factor structure of the Cognitive Restraint factor Eating Inventory (subset of original questions only) will be statistically equivalent between racial groups.

Hypothesis 4: A proposed sub-factor structure of the Disinhibition factor Eating Inventory (subset of original questions only) will be statistically equivalent between racial groups.

Hypothesis 5: A proposed sub-factor structure of the Hunger factor Eating Inventory (subset of original questions only) will be statistically equivalent between racial groups.

CHAPTER II

PSYCHOLOGICAL THEORIES OF OBESITY AND EATING BEHAVIOR

It is always important to evaluate problems within a theoretical framework; however, there are currently no comprehensive models that adequately describe the complex biopsychosocial factors that affect eating behavior patterns. While physiological models are quite advanced in terms of the neuroendocrine systems that control the acute starting and/or stopping of eating (Schwartz et al., 2000), these models do not address the cognitive processes that can easily override any physiological signals resulting in abnormal eating patterns. This chapter discusses the theoretical components on which the EI was built, describes some of the other theories of obesity and suggests a more recent behavioral theory that proves useful in interpreting the results of the present study.

Restrained Eating

Restrained eaters are defined as those who restricted their food intake to control their weight. The 10-item Restraint Scale has been used to predict food intake under varied conditions: 1) preloads of food, 2) alcohol intake, and 3) dysphoric emotions (Herman & Polivy, 1980). Restrained eaters have been experimentally shown to have paradoxical behavior in relation to a meal pre-load. They eat *more* food following a small "preload" meal, while unrestrained eaters eat *less* following a preload meal (Hibscher & Herman, 1977). This pattern of increased consumption despite recent eating indicates either dysfunctional feedback mechanisms or inattention to satiety cues. It has been said that obesity happens "one meal at a time" (Powley, Chi, Schier, & Phillips, 2005), meaning that regular deposits without equal withdrawals leads to a chronic energy imbalance.

Latent Obesity

The other theoretically-based measure that drove the development of the EI was the Latent Obesity Questionnaire (Pudel, Metzdorff, & Oetting, 1975). The investigators who developed the LOQ were seeking to understand a possibly moderating role of personality in obesity, centering on inadequate conflict management as the trait that leads to excess weight. By applying a social learning view of how high risk eating habits as a child can interact with a "special environment", Pudel et al. (1975) determined that people who had an acquired disposition for obesity could preserve a normal weight by using cognitive control. The term "latently obese" was used to describe these normal weight people who exhibited an external sensitivity to appetite and satiety cues and did not slow their rate of eating during a meal, yet were not overweight.

The LOQ was based on a model of eating-related learning experiences and personality as they combined in the psychogenesis of obesity. A major flaw in this theory, according to Stunkard and Messick (1985), was that the concept of latent obesity represented either an intermediate on the obesity-non-obesity dimension or a confounding of the obesity dimension with the restraint dimension. Stunkard and Messick (1985) asserted that the LOQ was inadequate due to lack of empirical evidence and that it could not address the significant group of restrained obese.

Other Theories of Eating, Hunger and Obesity

Other theories of obesity include the Internal-External Theory (Schacter & Rodin, 1974), Set Point Theory (Keesey & Powley, 1986) and the Positive Incentive Theory (Berridge, 2004). Schacter's Internal-External Theory focuses on a person's sensitivity to hunger cues. This asserts that overweight individuals are relatively insensitive to internal hunger cues and oversensitive to external cues as compared to normal weight individuals, who experience the opposite. Although weight-related differences predicted by the Internal-External Theory have been supported in many studies, it is appears that these differences were not solely a function of body composition (Rodin, 1981).

The Set Point Theory concerns the regulation of adipose tissue around a narrow range that is controlled by a variety of physiological mechanisms (Keesey et al., 1986). If there are significant changes in the amount of adipose tissue detected by neuroendocrine mechanisms such as leptin, then physiological adaptations occur that drive a return to the set point, inducing the storage of fat in periods of starvation (metabolizing lean tissue instead) and metabolizing fat when too many calories are taken in. This theory explains why it may be more difficult to lose fat under low intake conditions and to gain weight in higher intake situations. However, it does not explain why some populations are more obese than others. Therefore, the Set Point Theory may be considered a physiological description that does not account for effects of behavioral drive.

In contrast, the Positive Incentive Theory explains some of the psychological factors involved in overeating, such as socio-cultural and hedonic drive (Berridge, 2004). When a cultural group primarily consumes highly processed foods that are energy dense, the hedonic rewards become reinforcing. These incentives, along with family traditions and other cultural factors such as media influence, drive a greater tendency to consume a diet that is obesogenic for many.

Theory of Planned Behavior

Since eating is an intentional behavior and involves the planning and coordination of motor actions to ingest available food, there are varying components of behavioral control. Out of analysis of the relationship between attitudes, intentions and behavior came the Theory of Reasoned Action (Ajzen & Fishbein, 1980; Fishbein & Ajzen, 1975), which then evolved into the Theory of Planned Behavior (Ajzen, 1988; Ajzen, 1991). The Theory of Planned Behavior (TPB – Figure 2) has been applied to many areas of psychological research, especially in health psychology (Ajzen, 1991). The TPB has proven to be widely applicable and useful in hypothesis testing for health behaviors and attitudes such as smoking, alcohol consumption, low-fat diets, safe sexual practices, illegal drug use, medication adherence, sun exposure, using dental floss, wearing safety equipment, and exercise. In general, these studies have found support for the general theory and its applicability to a variety of behavioral domains (Ajzen, 2001).

The TPB leaves many specific questions in the "black box" that is Actual Behavioral Control (Figure 2), especially when attempting to apply this general model to maintenance of body weight. While we generally have control over our amount and intensity of physical energy expenditure, as well as our food choices and amounts we eat, there are strong environmental influences on the degree to which we maintain tight control of these aspects of the energy equation. As the environment in the United States is particularly well-suited to low activity levels and high intake of energy-dense foods, it is important to dissect the aspects of behavioral control involved in eating behavior required to improve strategies to resist this obesogenic environment.

CHAPTER III

DEVELOPMENT AND VALIDATION OF THE EATING INVENTORY

The EI was originally published under the name “Three Factor Eating Questionnaire” or TFEQ, but is now copyrighted as the “Eating Inventory”. The main motives for development of this instrument (Stunkard et al., 1985) were to improve upon the construct validity and usefulness in predicting behavior of two previously published tools: the Restraint Scale (Herman & Polivy, 1975) and the Latent Obesity Questionnaire (Herman et al., 1975; Pudel et al., 1975). The main issues cited regarding the Restraint Scale (RS) included: 1) it did not adequately discriminate between two types of eating behavior observed after a meal preload, 2) the weight fluctuation components alone were biased towards obese persons when there existed a type of non-obese who were high in restraint, and 3) that a social desirability component observed in obese persons resulted in underestimation of the construct (Stunkard et al., 1985). The primary limitation raised about the Latent Obesity Questionnaire was it did not address the significant subset of obese persons who measure high in restraint (Stunkard et al., 1985).

The Eating Inventory (EI) was not developed within a strictly theoretical structure, but rather was empirically compiled based on two prior theories of obesity: 1) restraint theory (Herman & Mack, 1975; Herman & Polivy, 1975) and 2) latent obesity (Pudel et al., 1975) in conjunction with additional items based on clinical observations (Stunkard et al., 1985). Stunkard and Messick (1985) asserted the two prior theories were inadequate alone or in combination. By developing the Three Factor Eating Questionnaire, the goal was to improve construct validity in measuring restrained eating and related issues that indicate disordered eating patterns (Stunkard et al., 1985).

In the process of developing and validating the Eating Inventory with only slight modifications from the TFEQ, Stunkard and Messick evaluated several different versions of the questionnaire in order to determine which items mostly closely covaried with each other in a theoretically valid way. Items that did not provide good discrimination between the factors (also known as the latent constructs of Cognitive Restraint, Disinhibition and Hunger) were eliminated. The final, three-factor structure was put forth as a set of characteristic in eating behaviors which, depending upon the response patterns, might indicate an individual is at greater risk for a clinical eating disorder (such as anorexia nervosa or bulimia nervosa) or obesity.

Original validation studies – Stunkard & Messick – 1985

In the process of developing the EI, a combination of the RS, the LOQ, and other items based on clinical observations were compiled and tested via factor analysis. The stated goal of this combined questionnaire was to study individuals, as well as to detect group differences. The base assumption when testing this measure with various groups was that the factors have the same meaning in the different groups. This is an important assumption that is tested in the present study with regard to obesity risk in women of different racial groups.

Ganley – 1988

This study was the first reported factor analysis of the EI on a large sample of adult women (Ganley, 1988). The original EI reliability data was of the 55-item version. Four weight-lability items loading on the Disinhibition factor were deleted to form the final instrument. This was done due to data from Drewnowski, et al. (Drewnowksi, Riskey, & Desor, 1982) indicating these items inordinately affected scores of obese persons. The criticism of this item deletion stated this action to revise the EI was the only thing done as a result of data *not* included in the EI validation process by Stunkard (Ganley, 1988). The 55-item version was chosen for Ganley's study due to interest in the emotional component of eating and because several studies had shown that the Disinhibition factor was a better predictor of behavior related to weight change during depression (Weissenburger et al., 1986), following cessation of smoking (Björvell, Rush, & Stunkard, 1986), and binge severity (Marcus & Wing, 1983).

While the subjects were all women recruited from a non-clinical setting (no racial data reported), ten percent were recruited from a Weight Watcher's program. Due to the inclusion of the four weight lability items, the original Disinhibition scale reflected two factors in this sample, called "Weight Lability" (ten items) and "Emotional Eating" (six items).

Hyland, Irvine, Thacker, Dann & Dennis, 1989

This study examined the factor structure of the Eating Inventory (referred to only in this study as the "SMEQ", for Stunkard-Messick Eating Questionnaire) compared to the Dutch Eating Behavior Questionnaire, or DBEQ (van Strien, Frijters, Bergers, & Defares, 1986). While the DBEQ has also been found to have a three-factor structure, there are differences in the theoretical basis upon which each was based. Both have the first factor associated with restrained eating. The second factor of the DBEQ is called "emotional eating" and was based on psychosomatic theory (Kaplan & Kaplan, 1957). The third factor of the DBEQ is called "externality" and is based on the theory of Schacter and Rodin (1974) which stated that obese people were more sensitive to external cues related to food (e.g., characteristics like smell, appearance), and less sensitive to internal physiological signals related to hunger. Additionally, according to Schacter and Rodin, as well as other theorists (Kaplan et al., 1957), *non-obese* persons are *more* sensitive to internally-based hunger cues.

The first factor extracted by Hyland, et al. was almost identical to the EI Cognitive Restraint factor. The second extracted factor consisted to items which were a combination of the EI Disinhibition and Hunger factors. This second factor was against hypothesis based on externality theory, i.e., obese persons are more sensitive to external food cues (Schacter &

Rodin, 1974). The second factor was made up of items about internal *and* external cues related to food, so it was called “food interest” (Hyland, Irvine, Thacker, Dann, & Dennis, 1989). The third factor consisted of three items from the EI, all stating that eating occurs when anxious, blue, or lonely. This third factor was therefore called, “emotional eating”. There were six items that loaded significantly on two factors, and Hyland, et al. attributed this to these items having compound meanings, e.g., “Dieting is so hard for me because I just get too hungry.”

The factor structure obtained from this sample was deemed to be very similar to the DEBQ, but the restricted item variety of the DEBQ, along with its complete lack of hunger and social items makes it of less utility (Hyland et al., 1989). The researchers questioned why the factor structure of the EI is apparently population-dependent. One answer is that the EI was not intended to be a universal measure of all eating behavior in all people, but rather to be used as a measure of individuals or as a way to compare groups (Stunkard et al., 1985). The Eating Inventory Manual (Stunkard et al., 1988) summarizes several studies by stating that gender difference appear to exist and therefore, studies and assessments should take that into consideration. However, there are no published reference ranges by gender.

Ricciardelli & Williams – 1997

This study evaluated only the EI Restraint factor in order to explore two theoretical sub-factors: rigid and flexible control (Ricciardelli et al., 1997). A Principal Components Analysis (PCA) determined three sub-factors among the CR items of the EI. These sub-factors were labeled “Emotional/Cognitive Concerns for Dieting” (33.5% of variance accounted for), “Calorie Knowledge” (7.8% of variance), and “Behavioral Dieting Control” (6.6% of variance). PCA is a form of factor analysis where the groupings of items are not pre-specified in the analysis, but rather the covariance of items determines which items seem to be more closely associated (Tabachnick & Fidell, 2001). While these results did not provide additional evidence for a flexible and flexible control hypothesis, it was posited that these latter constructs may be more strongly represented in a clinical population seeking weight loss, but less in a non-clinical sample like the college students who were the subjects this study (Ricciardelli et al., 1997).

Westenhöfer, Stunkard, Pudel – 1999

This report included three studies that were primarily focused on further elucidating the flexible and rigid types of restraint from the Cognitive Restraint factor of the EI (Westenhöfer et al., 1999). The results from the first study indicated that flexible control was significantly associated with lower BMIs at the beginning of a weight loss study and also was positively associated with weight loss when measured at the end of a year long weight loss study. While this study included a large number of participants (N = 7,407 West German men and women), the measures were all self-reported data, including weight.

The second study involved the addition of items to the flexible (nine new items) and rigid (five new items) constraint questions (Westenhöfer et al., 1999). The original, 51-item EI plus these new items were administered to 46 women and 39 men recruited by newspaper advertisements. While the mean BMI for the men and women put these samples in the normal weight to overweight category, this group would not likely have been seeking weight loss treatment. Therefore, the previous assertion by Ricciardeli and Williams (1997) that these constructs might be more applicable to a clinical population was not tested with this small pilot - the second study reported by Westenhöfer, et al., (1999). The apparent purpose of this pilot was to evaluate the reliability of the newly added items before testing them with a larger sample. The reliability values for both flexible control (FC) and rigid control (RC) were improved by the addition of the new items. However, whenever more items that are very similar are added, reliability values typically increase (Thompson, 2003). The key to a well developed set of items is to maximize reliability while keeping the number of items to a minimum (Thompson, 2003).

While this pilot also indicated that the Cognitive Restraint scores were correlated with Disinhibition, the exact values were not reported.

The third study in this report was performed on a population sample of 1,838 West German men, women and children ranging from age 14 – 85. Again, BMI was determined by self-reported height and weight. Multiple regression analyses indicated that the overall Cognitive Restraint score was adequately explained by a linear combination of (FC and RC). Most interestingly, *higher FC* was associated with *lower* Disinhibition scores, while *higher RC* was associated with *higher* Disinhibition scores. This pattern was similar for men and women, and BMI was correlated with Disinhibition, while overall Cognitive Restraint was not. The conclusion drawn was that the two types of restraint (FC and RC) were interacting with each other in the composite Cognitive Restraint score, reducing the effect of each other in relation to BMI.

Since the data collection did not focus on clinically diagnosable eating disorders per se, the subjects were asked a series of questions that described seven increasingly “binge-like” scenarios and frequency of experiencing each were summed to give an overall binge score. The highest values indicated the most frequent and severe episodes. These binge scores were significantly correlated with Disinhibition for men and women, but not with overall Cognitive Restraint. The binge scores between men and women did not differ significantly from each other, but the Cognitive Restraint, FC, RC and Disinhibition scores were higher for women than men. Hunger scores were not significantly different between genders. As expected from the previous analysis with BMI and Disinhibition, RC was associated with higher binge scores, while FC was associated with lower binge scores. The summary findings of this series of studies revealed methodological concerns regarding the validity of some of the measures or techniques.

Bond, McDowell & Wilkinson – 2001

The first stated objective of this study was to replicate the Restraint factors reported by Ricciardelli and Williams (1997). However, despite the use of statistical techniques that were admittedly different from Ricciardelli et al., the results showed a three factor solution similar to that of the Ricciardelli et al. (1997) study (Bond et al., 2001). The Ricciardelli et al. (1997) Cognitive Restraint subscale called "behavioral dieting control" was renamed "strategic dieting behavior" by Bond et al. (2001). The two other Cognitive Restraint subscales proposed were "attitude to self-regulation" and "avoidance of fattening foods" but the content of the individual items was not different. Bond, et al. acknowledge a similarity in their "strategic dieting behavior" to Westenhöfer's flexible control, while "attitude to self-regulation" and "avoidance of fattening foods" shared common elements with Westenhöfer's rigid control concept.

Disinhibition was also divided into three subscales related to categories of susceptibility: habitual, emotional, and situational. Finally, Hunger was divided into two loci for hunger cues: "internal" and "external" after Rotter's Locus of Control theory, which pointed out the differential properties for reinforcement between internal and external cues (Rotter, 1975). These proposed subscales to the EI were generated from very demographically homogeneous population of female undergraduates, mostly of normal weight, with a mean age of 25 years. No racial or SES data were reported.

These proposed sub-factor scores exhibited significant intercorrelation with each other across other factors (particularly between Disinhibition and Hunger). A small sub-sample of participants completed the EI 12 months later with reliability of the scores being mixed. Three subscales reflected significant mean differences: attitude to self regulation, habitual susceptibility to disinhibition and external locus for hunger. Additionally, the avoidance of fattening foods item correlation (r = .53) was significant but below the desired cutoff ($\geq$ .70).

The conclusion was that some of these aspects of eating behavior are not observed to be stable traits, at least in this young sample. It is unlikely that college students have developed the stable set of eating habits they will maintain throughout their adult lives (Levitsky, Obarzanek, Mrdjenovic, & Strupp, 2005; Westenhöfer, 2005). In fact, this period may reflect the most dysfunctional eating patterns as compared to the rest of the adult lifespan (Levitsky, 2005; Levitsky & Youn, 2004; Lopez-Moreno, Gonzalez-Cuevas, & Navarro, 2006; Mrdjenovic & Levitsky, 2005; Westenhöfer, 2005).

Atlas, Smith, Hohlstein, McCarthy & Kroll – 2002

While this study examined EI factor structure between women in the same racial groups of interest in the present study, the 300 Caucasian and 200 African American undergraduates were not evaluated for factors directly related to obesity risk. Instead, the focus was risk for the development of eating disorders in a racially divided sample of young women. While the statistical method used by Atlas, et al. (2002) was similar to this study, no details were provided on methods used for data standardization or factor rotation. No specific software package was named, which makes later evaluation of these results more difficult. Different computer programs for SEM allow for differing levels of control for constraints and methods by which fit indices are calculated, so it is important to specify what methods are used (Tabachnick et al., 2001).

Regardless, with the data set used, the constraints that the two racial groups have fixed and equal factor loadings, variances and covariances resulted in a good fit of the data with the original EI factor structure. However, no weight data was reported or factored into the model. While SES was compared between groups, it was based on parents' occupations and family income. Since all participants were college undergraduates, the assumption may have been that education was roughly equal between the groups and therefore not compared. Since BMI is often found to be highly correlated with SES, adding these variables into the model would have been beneficial in this study. One EI factor (Cognitive Restraint) was significantly and negatively correlated with SES *only* for Caucasians. The authors dismissed this as being a likely result of chance and probably not an important difference between the racial groups.

Mazzeo, Aggen, Anderson, Tozzi & Bulik – 2003

While this investigation attempted to replicate two previously suggested factor structures (Ganley, 1988; Hyland et al., 1989) as well as the original by Stunkard & Messick (1985) with a large sample (N = 1,020), there were several major methodological issues. The most notable is the questionnaire was fundamentally changed to include only first 36 items, all true/false response sets. The reasons given for this approach reflected a misunderstanding of how the other 15 items are scored, which is also in a dichotomous fashion. The second modification made would likely affect the psychometric properties to a significant degree: the response sets for the 36 true/false items became four category Likert-type options labeled "always", "often", "sometimes", and "rarely". As a result of this change, the questions required rewording to support the new response sets. This series of modifications represents a different instrument that would not likely reflect the original factor structure even if the original validation sample were to be used with this modified version (van Prooijen & van der Kloot, 2001).

Another methodological issue with this study is the use of a sample of women from a twin registry. The reason for this is not stated, but implied to be a convenience sample. While twin pairs were allowed to be in this analysis, there was no mention of comparing responses for twins to determine any similarities. While the presence of an unknown percentage of twin pairs does not necessarily reduce the sample by half, it raises concerns about the randomness of sample selection, not to mention the shared genetic and environmental contribution to results (Mazzeo et al., 2003). These non-clinical participants were significantly older (M = 42.5, SD =

8.2) than the samples used in the Ganley or Hyland studies, and the lifetime stability of the EI has not been established.

Summary and Conclusions

The overwhelming majority of the subjects in all of the reviewed factor analytic studies of the EI were of Western European descent. Four of the eight studies used a convenience sample of college students. Food consumption patterns varies significantly by age (Baranowski, Cullen, & Baranowski, 1999), and college student eating patterns are not likely to be maintained throughout adulthood (Kopelman, Caterson, & Dietz, 2005). Since it has become clear that the means of factor scores on the EI are not universal between samples, it is of limited utility to continue to compare data between age- and gender-mixed groups. Unpublished data from a pilot study (Kaiser, 2009) provides further support for the correlation between the EI factors and body composition to be different for men and women.

By knowing when to predict theoretical similarities and differences between groups, the EI can be a useful instrument to increase understanding of how eating behavior relates to other factors. The present study addresses this goal by applying improved experimental and statistical techniques to evaluation of differences between non-clinical samples of African American and Caucasian women with the specific aim of exploring differential relationships of eating behavior constructs to BMI.

CHAPTER IV

STRUCTURAL EQUATION MODELING

Overview of Structural Equation Modeling

Structural equation modeling (SEM) is a general term for a specialized group of statistical methods that allow for examination of a set of relationships between one or more independent variables and one or more dependent variables (Tabachnick et al., 2001). SEM is also sometimes called causal modeling, causal analysis, analysis of covariance structures, path analysis or confirmatory factor analysis (Tabachnick et al., 2001). A diagram of hypothesized relationships between variables is referred to as a "model". While many psychological constructs can be modeled, the confirmatory factor analysis method is often used to examine a questionnaire to determine the relationships of each question to the others and to the underlying constructs they are designed to measure. Additionally, SEM can be used to compare within- and between-group variance and covariance structures to examine relationships not apparent in univariate or some other multivariate techniques.

Latent Constructs

SEM can be an iterative process when used to uncover characteristics that cannot be directly observed (Hoyle, 1995). The measures used to describe a phenomenon are frequently aimed at a "latent" construct. This means that the phenomenon being measured cannot be experienced by the observer directly, so must be evaluated using an indirect method. A good example of this is the construct of "hunger". A researcher cannot directly experience or measure another person's hunger as if it were his or her own. But based on several items that *describe* the phenomenon of hunger, one can begin to see that the phenomenon being experienced by the subject is what is commonly known as "hunger" to most people. This construct can then be quantified for statistical comparison. Developers of questionnaires may execute several factor analyses before arriving at the final set of questions that are sensitive and discriminating enough to be used with confidence (Kline, 2005).

In diagramming a factor structure using SEM, the directly observed variables (also "indicators", or "manifest variables") are represented with squares or rectangles, while latent variables (also "constructs" or "unobserved variables") are represented by circles - see Figure 3. Lines between variables with either one or two arrows indicate a hypothetical relationship. A two-arrow line specifies that the relationship is not necessarily causal, but that the two items covary with no implied direction of effect (Tabachnick et al., 2001). Figure 3 is a simplified SEM diagram of the Eating Inventory. The one-ended arrows indicate that the latent construct, such as Hunger, predicts the measured variables – the questions that make up that factor on the questionnaire.

Creating Item Parcels

Due to limitations on the application of SEM methods to dichotomous data and large numbers of indicators for a factor, researchers frequently create what are called "parcels" (Little, Cunningham, & Shahar, 2002). These are sums of item scores, which may be theoretically grouped, statistically grouped or randomly grouped, depending upon the goal of the researcher (Little et al., 2002). While some may object to creating aggregates of items, the use of items is disadvantaged by their lower reliability, communality, ratio of common to unique factor variance and larger problems of distribution (Bagozzi & Heatherton, 1994; Little et al., 2002). Models using parcels rather than items are more parsimonious due to the reduced number of parameters being estimated that, in turn, reduces the odds of residuals that might be correlated (MacCallum, Widaman, Zhang, & Hong, 1999).

Analyzing Structural Equation Models for Fit to the Data

Unlike univariate or simpler multivariate statistics, structural equation models have many different criteria by which a researcher may determine the results of the analysis (Tabachnick et al., 2001). SEM software programs offer a variety of "fit indices", which are summary numbers that assess how accurately the hypothesized model describes the relationships between the indicators and the factors within the tested data set (Dunn, Everitt, & Pickles, 1993). Some of these fit indices are referred to as "absolute", and others are called "incremental", and can be used to compare between models, or between different versions of the same model (Hoyle, 1995). In reality, with each test of a model, one can assess fit by either saying the model does or does not fit the data, or that the data does or does not fit the model (Kline, 2005). The various fit indices simply provide different means to help the researcher make those decisions based on issues like non-normal distribution, sample size and how likely the model is to be replicated in another data set (Tabachnick et al., 2001).

Benefits of Factor Analytic Methods

Since self-report measures have a certain amount of accompanying measurement error that is difficult to estimate, some statistical techniques are preferred over others because they are better at accounting for and correcting this problem in measurement (Kline, 2005). Structural equation modeling (SEM) is a form of factor analysis that allows for correction of certain types of measurement error. There are ways to estimate and control for measurement error statistically, such as the transformation of non-normally distributed variables and the use of robust error estimations of standard error (Ajzen, 2001; Bentler, 2006; Dunn et al., 1993; Schifter & Ajzen, 1985; West, Finch, & Curran, 1995).

Another of the benefits of SEM is that it allows the researcher to specify, or "constrain" certain variances within a phenomenological model to be fixed or to covary with other components of the model. This aids in minimizing error variances that can only be estimated in univariate methods (Kline, 2005). While several previous studies have evaluated the factor structure of the Eating Inventory, there are notable limitations to the approaches and findings. Each had a slightly different focus, and only one posed similar research questions to those raised in the present study (Atlas, Smith, Hohlstein, McCarthy, & Kroll, 2002).

CHAPTER V

RESEARCH DESIGN AND METHODOLOGY

Participants

Participants were recruited using advertising in area newspapers, posted flyers and distributed flyers at community events in the Dallas/Fort Worth metropolitan area. Flyers and advertisements stated preliminary screening criteria as follows: African American or Caucasian women, 25 – 45 years of age who are pre-menopausal, non-smokers who are not currently pregnant. Further preliminary screening items for exclusion criteria were: current diagnosis of anorexia nervosa, bulimia nervosa, binge eating disorder; diagnosis of diabetes; surgical history of the gastrointestinal system such as bariatric, ulcer, gall bladder, cancer; current smoking or having quit smoking in the past year; history of pituitary or thyroid function conditions requiring medication; complete hysterectomy; definitive menopausal status (no menses for 12 consecutive months). Participants who met the preliminary screening criteria were scheduled for an appointment. If the volunteer was within 6 months of being 25 years old, or if they were older than 45 but premenopausal, they were accepted to participate. See Table 1 for descriptive statistics. Informed consent was obtained from all participants by trained study personnel according to the requirements of the Institutional Review Boards at the University of North Texas Health Science Center in Fort Worth and the University of North Texas, Denton campus.

Materials

1. Demographic Questionnaire - Participants filled out a demographic questionnaire that included date of birth, race, current medications, education, occupation, household income, number of children and total number of persons in household.

Those who noted taking blood pressure medication, antidepressants, steroids and hormonal birth control methods were recorded for between-groups comparison, as some of these may influence body composition.

2. Human Chorionic Gondatropin Dip-Strip, San Diego, CA - A urine sample was collected to screen for possible unknown pregnancy.
3. Keto-Diastix, Bayer Corporation, Elkhart, IN - A urine sample was collected to screen for possible unknown diabetes.
4. DSM-IV Eating Disorder Criteria Checklist - A history of anorexia nervosa, bulimia nervosa and binge eating disorder were obtained by interview using the DMS-IV TR criteria (American Psychiatric Association, 2000). Past treatment for any of these did not determine exclusion if it was terminated 12 months prior to the study appointment.
5. SCID-I Substance and Alcohol Dependence sections for interview - Current drug/alcohol dependence (within the past 12 months) was assessed using the SCID-I, Clinician Version, Section E (First, Spitzer, Gibbon, & Williams, 1997).
6. Eating Inventory - Participants filled out the Eating Inventory (Copyright 1983, The Psychological Corporation).
7. Dieting Questionnaire - A separate “Dieting Questionnaire” consisting of the 14 supplemental cognitive restraint questions proposed by Westenhöfer, Stunkard & Pudel (1999) was completed.
8. Personality Questionnaire – This short, true/false questionnaire was made up of the Infrequency (infrequently false items used to assess social desirability) and Impulsivity subscales of the Zuckerman-Kuhlman Personality Questionnaire (ZKPQ) in order to assess possible influences of social desirability effects in

response patterns (Zuckerman, Kuhlman, Joireman, Teta, & Kraft, 1993). While impulsivity is not a primary outcome variable in this study, these 19 questions served to mask the purpose of the 10 social desirability questions.

9. Tanita scale (Models BC-418, BC-310 or BC-557, Tanita Corporation) – Used to measure weight.
10. Stadiometer - Used for height measurement to calculate body mass index (BMI).

Procedure

After informed consent was obtained, participants provided a urine sample for the pregnancy and diabetes screening tests to be performed. The researcher immediately provided the results to the participant and recorded them. Interviews for eating disorder history and drug/alcohol dependence status were performed by a trained researcher. Participants filled out the demographic questionnaire, Eating Inventory, Personality Questionnaire and Dieting Questionnaire. Height and weight measurements were taken and recorded.

Data Preparation

Raw data from the Eating Inventory were converted and summarized for the three factors per the Eating Inventory Manual (Stunkard et al., 1988). Raw responses were reverse scored (or converted in the case of the multiple choice items) so that “0” indicates the item does not contribute to its designated factor score, while a “1” indicates that item contributes to its designated factor score. Factor scores were summed for the pre-defined Cognitive Restraint, Disinhibition and Hunger factors. Reverse scoring was required for one items of the supplemental cognitive restraint items on the Dieting Questionnaire (Westenhöfer et al., 1999). Items from the Personality Questionnaire were reverse scored as appropriate and summed for the social desirability and impulsivity scores (Manual of Norms for the ZKPQ, unpublished, available from the authors).

For indicators of socio-economic status, an adjusted household income (Percent Poverty) variable was determined by dividing raw monthly household income by the appropriate reference poverty level (determined by total persons in the household). Reference values for poverty level were determined according to current U.S. guidelines for the 48 contiguous states and District of Columbia provided by the 2008 United States Health and Human Services - see Table 2 (U.S.Department of Health and Human Services, 2009). Income as a percent of poverty level was computed based on raw income total for number of persons in the household. This was based on the assumption that the total number of children who are dependents are indicated by those who are less than or equal to 21 years of age. Education level was ranked according to the Hollingshead scale: 1=less than or equal to 7th grade; 2 = 8th or 9th grade; 3 = 10th or 11th grade; 4 = high school graduate or GED; 5 = partial college (at least one year, or specialized training); 6 = Bachelor’s level degree; and 7 = any kind of graduate degree (Hollingshead, 1975). Occupational status based on self-reported job title was ranked according to the Hollingshead scale from 1 (lowest) – 9 (highest) (Hollingshead, 1975).

Statistical Analysis

The data were analyzed for descriptive statistics, correlations, between groups ANOVA and outlier analysis for group characteristics and outcome variables using SPSS version 15.0 for Windows (SPSS, Inc., Chicago, IL). The following primary outcome variables were analyzed for univariate outliers using a z-score cutoff of 3.29 (Tabachnick et al., 2001): BMI, cognitive restraint, disinhibition, hunger, income, educational level, and occupational status. For the determination of multivariate outliers, Mahalanobis’ distance (χ^2 cutoff value for three variables: 7.82, p=.05) was used to compare cognitive restraint, disinhibition and hunger. As a result of univariate and multivariate outlier status, one African American (AA) participant and 12

Caucasian (C) participants were excluded from the model testing. This left 100 AA and 110 C participants for the final model testing and analysis (see Figure 7 for CONSORT diagram).

Experimenter Effect

To determine possible influences on responses based on racial similarity or difference between participant and experimenter, cases were coded according to whether the experimenter was African American or Caucasian. The social desirability score was compared in a 2×2 analysis of variance (ANOVA) between groups for race, experimenter and interaction effects. See Table 1 for general between-group comparisons of the demographic variables.

Confirmatory Factor Analysis

Confirmatory factor analysis was performed using EQS version 6.1 for Windows (Multivariate Software, Inc., Encino, CA). The between-groups factor loading and factor covariances for each hypothesis were constrained to be equal (see Figures 4 – 6). Then the model was run again unconstrianed, allowing all parameters to be freely estimated. A χ^2 difference test was performed between the constrained and unconstrained models to assess whether the two groups appear to be sampled from the same population (Kline, 2005). Data set distribution and model fit were evaluated using several indicators described next.

A ratio of Mardia's Coefficient (Mardia, 1970) divided by the normalized estimate was examined for the presence of multivariate kurtosis both in the combined sample as well as in each racial group, with a value of greater than +/- 3.0 indicating significant multivariate kurtosis according to Bentler, 2006. The Satorra-Bentler Scaled χ^2 (termed "robust") was used to assess overall model fit (with an alpha level of .05) because it is scaled for use with non-normally distributed data, e.g. when significant multivariate kurtosis is present (Satorra & Bentler, 1990). For assessment of incremental fit between models, the McDonald's Fit Index (MFI), (McDonald, 1989) and the Comparative Fit Index (CFI), (Bentler, 2006) were examined. McDonald's fit index is considered a more robust statistic than most when testing for between group invariance as it is not correlated with the fit indices of the overall model, which reduces difference values that might be generated by a less than accurate unconstrained model (Cheung & Rensvold, 2002). The CFI is also robust in this regard (Cheung et al., 2002) and additionally considered the best index to use with small sample sizes (Kline, 2005). Values of MFI and CFI over .90 are desirable for indication of adequate data fit for the specified model.

Steiger's Root Mean Squared Error of Approximation (RMSEA) (Browne & Cudek, 1993; Steiger, 1989) was also evaluated for a value less than or equal to .05. RMSEA is based on the non-centrality parameter of the χ^2 distribution and defined as the square root of population misfit per degree of freedom. The generally accepted level for good fitting models is a RMSEA of .05 or less, between .5 and .8 is of reasonable fit, whereas models with a RMSEA of .10 or greater are considered to be poor fitting (Browne et al., 1993). However, the EQS program also provides a 90% confidence interval of the RMSEA value and fit should be evaluated based on the upper bound of the interval given, not simply the RMSEA value itself, as this interval is adjusted based on sample size (Kline, 2005).

Models with a RMSEA value $\leq$ .5 but with an upper limit of the 90% confidence interval > .10 cannot be rejected according to the hypothesis of poor approximate fit according to the RMSEA value. However, the upper limit of the confidence interval supports poor approximate fit, indicating a mixed outcome (Kline, 2005). Models indicating this combination of circumstances are more likely with smaller sample sizes, indicating need for a larger sample size to generate more precise results (Kline, 2005).

Study Design

This study was a cross-sectional, quasi-experimental, between groups design. A priori factor structures were compared for statistical equivalence between a community sample of

African American and Caucasian women for each of the three hypotheses. If a tested factor structure was not statistically equivalent between groups, a post hoc exploratory factor analysis was performed on each racial group separately. Race is a manifest, independent variable. Body Mass Index is a manifest dependent variable, while the Eating Inventory items are latent, dependent variables used to generate parameter estimates of the factor scores (independent variables).

Due to software limitations that allow only one weighting variable, a combined weighting variable was used as an optional covariate in each model. It was generated by a sum of z-scores of BMI, education rank and social desirability scores for each participant so as to proportionally weight the individual cases for the Eating Inventory scores in the models. Few SES indices are available that have been validated in the United States and the relationship of various aspects of SES to BMI vary according to a country's level of industrialization (Braveman et al., 2005). Education rank was chosen in the present study as the SES measure in the weighting variable as it is generally most highly correlated with BMI [negatively correlated in industrialized nations (Braveman et al., 2005)], which was also true in this sample (see Table 3). Many researchers agree that education is the most stable and robust indicator for SES over time (Zhang et al., 2004b).

CHAPTER VI

RESULTS

General and Group Characteristics

Descriptive statistics were generated for the overall sample and separately for the racial groups. Between groups analysis of variance (ANOVA) were performed to assess group differences in the demographic variables. See Table 1 for group descriptive statistics and between groups analysis of variance (ANOVA).

The two racial groups were similar in age (overall M = 35.1, SD = 7.1), BMI (overall M = 28.9, SD = 7.3) and occupational status (overall M = 6.0, SD = 1.9). Of the Caucasians, 39.1% had a BMI > 30, classifying them as obese; 46% African Americans had a BMI > 30 but the groups were not significantly different in BMI overall, p = .09. However, African Americans were significantly lower than Caucasians in educational status (p < .001) and income (p < .001), each with small effect sizes. African Americans had significantly higher Social Desirability scores than Caucasians (p = .001). Yet, a significant proportion of both groups had Social Desirability scores > 3 (African Americans = 84%, Caucasians = 71.8%, overall = 77.6%), therefore this variable was used as a covariate in each hypothesis in order to assess possible influence on model variances.

A minority of women in both groups reported taking medications that may be associated with effects on body composition. For African Americans, 15% reported taking blood pressure medication; none reported taking systemic steroidal medication; 3% reported taking at least one antidepressant; and 11% reported using a hormonal method of birth control. For Caucasians, 6.4% reported taking blood pressure medication; 1.8% reported use of a systemic steroidal medication;

17.3% reported at least one anti-depressant; and 26.4% reported using a hormonal method of birth control.

Experimenter Effects

Overall, 117 participants had an experimenter that was of the same race, while 93 participants has an experimenter of a difference race. The Caucasian experimenter processed 81% of the total sample, while the African American experimenter processed 19% of the total sample. The influence of race difference/similarity between experimenter and participant was assessed using a 2×2 analysis of variance (ANOVA) with the EI scores. There were no significant simple experimenter effects, or race by experimenter interactions with EI scores, all $p \geq .355$.

Hypothesis Testing

Hypothesis 1: *The factor structure for the original, three-factor Eating Inventory will be statistically equivalent between racial groups*.

Initial testing of an overall model using all items from the Eating Inventory resulted in a poorly fitting model. To prepare the data sets for testing this hypothesis, parcels of items in each of the three factors (Cognitive Restraint, CR; Disinhibition, DI; and Hunger, HN) were randomly created from an exploratory factor analysis using SPSS. Three factors were specified with all 51 items of the Eating Inventory. The extraction method used was Maximum Likelihood and the rotation method used was Varimax with Kaiser normalization. The resultant rotated factor matrix was unidimensional with minimal cross-loading of items to factors.

Items with a factor loading value greater than or equal to .30 were selected to make up the parcels for the three factors, making up a total of 25.6% of the variance. Each item was randomly assigned to a parcel within the original factor designation according to the following strategy: Each parcel for a factor got one of the highest loading items, then one of the lowest loading items, followed by middle loading items so that the sum of each parcel's items factor loading values was as close to equivalent as possible. In all, 27 of the original EI questions were selected to create the parcels used to test Hypothesis 1. See Table 4 for a listing of items to parcels to factors.

Hypothesis 2: *The two-factor Cognitive Restraint model will be statistically equivalent between racial groups.* Fourteen additional questions were added to a subset of the Eating Inventory Cognitive Restraint questions (Westenhöfer et al., 1999) to form the "Flexible Control" factor and "Rigid Control" factor. Initially, all of the original and supplemental items were organized in a model based on the two factors (four sub-factors) described by Westenhöfer, et al. However, this model did not adequately fit the overall data set (N=210). Two subsequent models were attempted based on a four factor EFA using all of the original and supplemental items, using only those items which loaded on at least one factor at $\geq .30$. However, these two models did not fit the data adequately either. SEM is sensitive to highly correlated items, and since the supplemental rigid factor was correlated at > .9 with the original items flexible factor, two separate analyses were performed using a two factor structure for the original items and a two factor structure for the supplemental items. This approach using only the *original* EI items into the prescribed flexible and rigid control factors resulted in a two factor model that fit well overall. Similarly, a two factor model using only the supplemental items resulted in a two factor model that fit well overall. Therefore, this hypothesis was tested between groups in two parts: a) original EI flexible and rigid control items, and b) supplemental flexible and rigid control items.

Hypothesis 3: *A proposed sub-factor structure (Bond et al., 2001) of the Eating Inventory Cognitive Restraint questions (original questions only) will be statistically equivalent between racial groups.*

The 11 items that make up the three Cognitive Restraint sub-factors were parceled by randomly creating two parcels for each of the three sub-factors. The overall and between groups models with this structure fit the data adequately for hypothesis testing.

Hypothesis 4: *A proposed sub-factor structure (Bond et al., 2001) of the Eating Inventory Disinhibition questions (original questions only) will be statistically equivalent between racial groups.*

The 13 items that make up the three Disinhibition sub-factors were parceled by randomly creating two parcels for each of the three sub-factors. The overall and between groups models with this structure fit the data adequately for hypothesis testing.

Hypothesis 5: *A proposed sub-factor structure (Bond et al., 2001) of the Eating Inventory Hunger questions (original questions only) will be statistically equivalent between racial groups.*

The 12 items that make up the two Hunger sub-factors were parceled by making three parcels, two items each for sub-factor 1 and three parcels, two items each for sub-factor 2. The overall and between groups models with this structure fit the data adequately for hypothesis testing.

Structural Equation Model Confirmatory Factor Analysis Results

See Table 5 for a comprehensive list of between factor correlations for all hypotheses. See Tables 6 – 8 for overall and group factor loadings for each model tested for each hypothesis.

Hypothesis 1

Hypothesis 1 was confirmed: there were no significant differences in the original EI factor covariances between groups according to the χ^2 difference test, indicating the samples were taken from the same population. Mardia's ratio of multivariate kurtosis (Mardia's coefficient/normalized estimate) for all models were within acceptable limits (between +/- 3), (Bentler, 2006). See Table 9 for a summary of the tested models and associated fit indices. All models indicated acceptable χ^2, MFI, CFI and RMSEA values, even at the upper limit of the RMSEA 90% confidence intervals (all < .10). This supports the adequacy of the sample size for testing this model in terms of "not bad fit".

All between-groups models indicated a non-significant χ^2 difference value. Models using BMI as a covariate and the combined covariate using summed z-scores of BMI, social desirability and education rank indicated no significant group differences. The model that fit the data best according to the lowest RMSEA value and highest MFI and CFI values was the overall model (groups combined) with the combined covariate variable.

Hypothesis 2a

Hypothesis 2a could not be confirmed or disconfirmed with the tested models. There were no significant differences in the original EI flexible and rigid control factor covariances between groups according to the χ^2 difference test. However, the upper limits of the 90% confidence intervals for the RMSEA values were all > .10, indicating a need for a larger sample size to determine a more precise result for this hypothesis test. Mardia's ratios for all models were within acceptable limits. See Table 10 for a summary of the tested models and associated fit indices. All models indicated acceptable Scaled χ^2, MFI, and CFI values.

All between-groups models indicated a non-significant Scaled χ^2 difference value. The model that fit the data best according to the lowest RMSEA value and highest MFI and CFI values was the overall model with no covariate, although the upper limit of the confidence interval for the RMSEA value was .118. Additionally, the model with the combined covariate could not be tested with all factors constrained to be equal, as the model failed to converge with a complete set of parameter estimates. Therefore, a version of the constrained model with only factor loadings constrained to be equal was used to test this model and indicated no significant group difference.

Hypothesis 2b

Hypothesis 2b could not be confirmed or disconfirmed with the tested models. There were no significant Scaled χ^2 differences in the supplemental EI flexible and rigid control factor covariances between groups. However, the upper limits of the RMSEA confidence intervals were all > .10 except for two (see Table 11), indicating a need for a larger sample size to determine a more precise result for this hypothesis test. Mardia's ratios for all models were within acceptable limits. All models indicated acceptable Scaled χ^2, MFI, and CFI values. All between-groups models indicated a non-significant Scaled χ^2 difference value. The model that fit the data best according to the lowest upper confidence interval of the RMSEA value and highest MFI and CFI values was the between groups, constrained model with BMI as the covariate.

Hypothesis 3

Hypothesis 3 was confirmed: there were no significant Scaled χ^2 differences in the cognitive restraint sub-factor covariances between groups. Mardia's ratios for all models were within acceptable limits. See Table 12 for a summary of the tested models and associated fit indices. All models indicated acceptable Scaled χ^2, MFI, CFI and RMSEA values. The upper limit of the 90% confidence intervals for the RMSEA values were all < .10, and the Scaled χ^2 values, MFI and CFI values were all within acceptable limits for all models. All between-groups models indicated a non-significant Scaled χ^2 difference value. The model that fit the data best according to the lowest RMSEA upper confidence interval value and highest MFI and CFI values was the overall model with no covariate.

Hypothesis 4

Hypothesis 4 could not be confirmed or disconfirmed with the tested models. There were no significant Scaled χ^2 differences in the disinhibition sub-factor covariances between groups. However, the upper limits of the RMSEA confidence intervals were all > .10. Mardia's ratios for all models were within acceptable limits. See Table 13 for a summary of the tested models and associated fit indices. All models indicated acceptable χ^2, MFI, CFI and RMSEA values. All between-groups models indicated a non-significant Scaled χ^2 difference value. The model that fit the data best according to the lowest RMSEA value and highest MFI and CFI values was the overall model with the combined variable as the covariate, although the upper limit of the RMSEA confidence interval = .103.

Hypothesis 5

Hypothesis 5 could not be confirmed or disconfirmed with the tested models. See Table 14 for summaries of the tested models and associated fit indices for each covariate condition. Mardia's ratios for all models were within acceptable limits. There were no significant Scaled χ^2 differences in the hunger sub-factor covariances between groups. However, there were significant Scaled χ^2 probability values for the between groups unconstrained model with no covariate, as well as for the between groups *unconstrained* model with BMI as the covariate, although the *constrained* models for each had acceptable Scaled χ^2 values.

For the between groups with no covariate model, only the factor loadings were constrained to be equal as the addition of the constraint for equal factor variances resulted in a significant Scaled $\chi^2(23)$ $p = .0357$. Rather than relying on suspect χ^2 difference values for the unconstrained models, examination of the MFI differences between constrained and unconstrained models indicate no significant difference (< -.02 change is non-significant per Cheung & Rensvold, 2002). Additionally, the upper limits of the 90% RMSEA confidence intervals for all models ranged from .066 to .137, indicating possible sample size issues.

CHAPTER VII

DISCUSSION

Summary and Integration of Results

This study assessed several variations of eating behavior constructs in a large sample of African American and Caucasian women. One purpose was to determine whether models of these constructs were significantly different between the racial groups. A second purpose was to examine whether these eating behavior constructs were related to BMI differently between groups. The results provided indicate no cultural bias in the Eating Inventory or in some proposed alternate models. Additionally, the data show that these models are related to body composition in a similar way in both groups.

The strongest hypothesis support is for the validity of the original Eating Inventory three factor structure. Additionally, none of the tested models support significant group differences. Of the alternate factor structures tested, the three sub-factor structure for cognitive restraint (Bond et al., 2001) was the best model fit with the data. The two factor structure of flexible and rigid control (Westenhöfer et al., 1999) was not supported by the tested models. Difficulties in determining an acceptable model for flexible and rigid control factors that met all statistical assumptions and requirements are likely a result of the high degree of correlation between these two factors using all items as originally proposed.

In addition to general model support and group invariance for the original three factor structure of the Eating Inventory, the relationship of each of the latent constructs to BMI appears to be invariant across groups. This data also indicates the relative insignificance of the influence of social desirability or education level on the variance/covariance structure of the EI latent constructs for most models tested, despite significant mean differences in these variables between groups. Thus, results demonstrate that the Eating Inventory has cultural validity when used to assess obesity risk in African American women.

Explanations for Findings

It must be reiterated that the items used for testing the original EI factor structure in the present study were 27 of the original 51 (Table 4). However, these items used for testing were those that loaded most highly on each factor in the exploratory factor analysis, which ensures the adequacy of the overall model fit. Regardless, since there are few exact redundancies built into the EI, it is interesting to note that the factors are supported with significantly fewer items. A large CFA study of the TFEQ did not find support for the all original factors (cognitive restraint was retained, disinhibition and hunger were not) in an obese sample, but found strong psychometric properties for an alternate, three factor structure using only 18 of the original items (Karlsson, Persson, Sjostrom, & Sullivan, 2000).

The fully combined two factor structure tested in hypothesis 2 (using both original EI items and the supplemental items) resulted in a model with a .832 correlation between factors with this sample. SEM techniques interpret two factors that are correlated at or near .9 as redundant and therefore, a poor model for describing the data if a one factor model adequately describes the data. Thus, the models and tests of the *original* items alone in this two factor structure did not meet minimum requirements for model fit, and the results for model tests of the *supplemental* items were also equivocal. See Table 5 for a listing of all factor correlations for all models tested.

The factor correlation in the overall model tested for hypothesis 2a was .977 and the factor correlation for the overall model tested in hypothesis 2b was .169. This indicates that the supplemental items provide better discrimination between these two constructs than those selected from the original EI CR items with this sample. However, the low number of degrees of freedom in this simple model indicates a need for a very large sample to test this model with adequate power.

In an effort to separate the influence of the combined covariate on improvement of fit with the between groups models for hypothesis 5, separate post-hoc analyses were run using education or social desirability alone as covariates (see Table 15), since the BMI covariate condition had issues of fit as indicated by the significant Scaled χ^2 probability value for the unconstrained model. Using education alone as a covariate resulted in the best overall fit of any model tested for this hypothesis. Upper limits of the RMSEA confidence intervals for the overall, constrained and unconstrained models were all $\leq .093$. There was no significant χ^2 difference with education as a covariate, but the CFI difference between the constrained model and unconstrained model was -.016. Difference values for CFI > -.01 indicate cause for rejection of the null hypothesis of invariance between groups (Cheung et al., 2002). χ^2 difference is more sensitive to sample size than MFI (Cheung et al., 2002). Therefore, education levels may be differentially correlated between groups in aspects of hunger as measured by this two sub-factor model. In the test using Social Desirability as the only covariate, only the overall model had acceptable Scaled χ^2, upper limits of the 90% RMSEA confidence intervals, MFI and CFI values. Consequently, this single covariate did not improve model fit to facilitate between groups testing, providing further evidence that social desirability is not a significant influence on scores for the Hunger factor.

Integration of Findings with Past Literature

There are no published studies that have examined these same questions using similar approaches, but some general comparisons with other investigations can be made. First, this study extends the work of Atlas, et al. (2002) in determination of factor invariance across groups of African American and Caucasian women when evaluating the possible relationship of SES to weight concerns. Since the focus for their study was risk for eating disorders (ED), it was appropriate to focus on young women. With a broader demographic range, present study adds support to this prior study for the lack of significant influence of SES measures on risk as measured by the Eating Inventory.

The present study adds support of other investigations (Mazzeo et al., 2003; Ricciardelli et al., 1997) that did not find significant construct validity for the flexible and rigid control factors (Westenhöfer et al., 1999). While the study reporting the validation of the flexible and rigid control factors used a very large sample (N=1,838), the sample was very heterogeneous, with a broad range of ages for both males and females. In contrast, others (Mazzeo et al., 2003; Williamson et al., 2007) have asserted that these flexible and rigid control constructs are not clearly distinct from the original EI restraint scale and may only be significant in clinical populations, which have not been the population sample used in any of the validation studies (including the present study) subsequent to the Westenhöfer, et al., 1999 study. In contrast, a large CFA study of the TFEQ using a sample of only obese men and women, the original Cognitive Restraint factor was consistently reproduced and scaling analysis demonstrated strong item-scale discriminant validity, but poor item-scale convergent validity (Karlsson et al., 2000). In aggregate with the present study, the validity of these constructs is indeed likely to be population-dependent.

The present study also adds modest support for the EI sub-factor structure proposed by Bond, et al. (2001), at least for the three constructs included in the original EI cognitive restraint factor. These were described at 1) strategic dieting behavior, 2) attitude to self-regulation, and 3) avoidance of fattening foods (Bond et al., 2001). Clearly, there is little consensus on the most consistent number of distinct constructs under the umbrella of cognitive restraint, but there is increasing evidence to support the claim that it is not a unitary construct (Ricciardelli et al., 1997).

Contribution of Findings to Literature

The present study adds important information to the existing literature in terms of extending the application of the Eating Inventory to investigations of relative risk for obesity across racial groups. Additionally, the data reflect what may be a fading phenomenon in terms of the correlation between SES measures and increased weight in the United States (Zhang et al., 2004b). No systematic attempt has been published to establish group norms for the Eating Inventory, although several studies have examined gender differences in the factor scores indicating that men sampled from a non-clinical population have lower factor scores than women (Boerner, Spillane, Anderson, & Smith, 2004; Neumark-Sztainer, 1999; Westenhöfer et al., 1999). A large-scale CFA study of the TFEQ in Sweden (Karlsson et al., 2000), including obese men and women cross-validated an alternate model between genders, but did not report whether there were significant mean differences. Before establishing group norms, instruments must be determined to be relatively free of cultural bias among groups to be compared. The present study is an important first step in that direction.

Implications of Findings

These findings together with other similar examinations have broad implications for theory, research and application in the clinical setting. The Eating Inventory has made a large contribution to eating behavior research for the past three decades. Much has been learned through its use which can inform the next generation of instruments to be developed. As disclaimed by the developers of the Eating Inventory, it was not intended to assess attitudes or normative behavior (Stunkard et al., 1988). As the importance of these aspects of human behavior has been demonstrated in many psychological instruments (Kremers, Visscher, Seidell, van Mechlen, & Brug, 2009), these must also be included in a more comprehensive instrument for measuring obesity risk. Additionally, the construct of personal agency is important in measuring health behaviors, especially in the context of changing high-risk behaviors (Siegrist, 2000).

A more comprehensive theoretical framework that encompasses the aforementioned issues is the Theory of Planned Behavior (TPB, see Figure 2), (Ajzen, 1991). Since the constructs are not domain-specific, it is more likely that an eating behavior instrument developed within this theoretical framework would have greater validity across cultures and population sub-groups. While the relative influence of each of the primary constructs may vary in their relative influence, many different applications of this underlying framework have indicated useful application of the constructs in measuring and predicting behavior (Ajzen, 2001). To attempt to map the constructs of the Eating Inventory to the TPB, it could be argued that the EI contains aspects of perceived behavioral control, actual behavioral control and intention.

The TPB is prospective in nature and studies using measures based on this prospective approach prior to weight loss programs have shown some predictive ability regarding weight loss success (Gardner & Hausenblas, 2005; Schifter et al., 1985). However, no studies have applied TPB-based measures to attitudes and intentions to reduce weight independent of subsequent tests of weight loss programs. When a person seeks weight loss treatment, there has been some degree of attitude shift that caused that person to take action. The attitude shift involves moving away from a degree of acceptance with the behaviors and/or conditions that created the body composition in the first place. A person who has a high degree of external control perception about their weight, e.g., genetics, will be less likely to have a very strong degree of Perceived Behavioral Control as applied to losing weight and may not seek help to lose weight.

Since the EI was not developed with the TPB as its theoretical basis, it is not possible to "reverse engineer" the EI into a TPB mold and expect a good fit. However, when attempting to understand possible *attitude* differences between groups, as has been hypothesized about African

Americans and ideal body shape, an interpretation of data through the lens of the TPB can be helpful. If one were to attempt to categorize the EI items according to the TPB schema of attitudes, norms and behavioral control, most items would fit well into the Perceived Behavioral Control (PBC) domain. In fact, by the factor names of Cognitive Restraint and Disinhibition, one could make a case for all the items in each subscale applying to the TPB Perceived Behavioral Control realm. The Hunger subscale items do not intuitively fit into any of the TPB domains per se, and the EI does not seem to address attitudes or social norms to any significant degree. Looking at individual EI items, some fit within the important behavioral antecedent of *Intent* within the TPB (e.g., "How frequently do you avoid stocking up on tempting foods?").

Perceived Behavioral Control (PBC) and Intent

Many research applications of the TPB have focused on the aspects of PBC and Intention in relation to weight loss and exercise behavior (Ajzen, 2001; Ajzen, 2002; Conner, Norman, & Bell, 2002; Gardner et al., 2005; Schifter et al., 1985; Smith, 2004). In addressing conceptual and methodological ambiguities regarding PBC, Ajzen (2002) illustrated a hierarchical model of PBC containing two sub-components: 1) perceived self-efficacy and 2) perceived controllability. In this view of PBC, it is then possible to interpret the EI subscale of Cognitive Restraint as a form of self-efficacy based on past behavior that can be applied to intent. Additionally, the perceived controllability component in terms of lack of control might relate to the EI subscales of Disinhibition and Hunger which serve to disrupt intent. Again it must be emphasized that while the EI does not fit the TPB model, other group differences that are not necessarily being captured by the EI will highlight areas for possible expansion of the EI. Or at least, supplemental measures of attitudes and norms regarding weight control will help paint a more complete picture of differences between groups whether they are different genders, ages, or cultural origins.

The research implications of the present study highlight several areas of focus. First, due to lack of availability of more comprehensive measures of obesity risk, careful selection of a group of theory-based instruments should be assembled and tested across groups as a standard panel. From this, fewer, more comprehensive assessments can be further distilled within a broader theoretical framework to create practical tools for both research and clinical practice. The continued used of differing combinations of questionnaires and other measures will delay the progress of solutions to the obesity epidemic.

Second, researchers should avoid focus on factor analytic studies that result in semantic arguments over constructs and what they should be called. By developing and testing newer instruments with a strong theoretical foundation, using better psychometric techniques for item development that result in high reliability, validity and internal consistency, researchers can attempt to increase the effect sizes of the variables being observed between groups. This has the practical effect of reducing the sample sizes needed to generate results with confidence.

Finally, in the development of these newer instruments, consideration needs to be given to the most effective ways to deploy these measures (e.g., Internet and personal communication devices) while still providing high rates of independence of observations and face validity. This approach will aid in extending the reach of researchers to large population samples. The small effect sizes observed in most psychosocial research need to be addressed in order to advance the research in this and many other areas.

This study represents a first step towards determining if racial group norms are needed for the Eating Inventory. If so, such normative information would have immediate clinical application for individual assessment and treatment. This effort is critical to address the health disparities of obesity and its associated comorbidities.

Limitations

While the design and validity of this study has advantages over several others cited, aspects of it limit the interpretation of some results. Specifically, to have greater confidence in the question of experimenter/participant race effects, participants should have been randomly assigned to one condition or the other in equal numbers. Additionally, a social desirability measure designed to detect more eating-related attitudes might have revealed different levels of influence on the primary outcome variables. In the spirit of true informed consent, participants were informed that the purpose of the study was to evaluate an eating questionnaire in the context of racial differences in obesity risk. This may have influenced response patterns based on participants' own views on the reasons for the problem.

As for external reliability and generalizability, this study needs to be expanded and replicated between groups in other geographic areas to account for possible local differences in obesity risk, e.g., local dietary preferences and the built environment. The metropolitan area that served as the source for recruitment is very automobile-oriented. Other areas of the United States provide easier opportunities for physical activity, which may or may not affect these two groups differentially.

Regarding statistical analysis and power concerns, this study sample was at the lower end of the minimum recommendation for factor analysis, e.g., at least 5 cases for each parameter being estimated (Kline, 2005). Having no a priori information on effect size for this study, few options exist. SEM involves an additional level of difficulty when attempting to estimate power and effect sizes. Rather than at the parameter level, as is simply done with univariate statistics, attempts to define power for an overall structural equation model are difficult. A parameter's power depends on the complexity of its model and where that parameter is in relation to other parameters in the model. Few SEM software programs offer a power estimation module, but such tools are available.

To estimate statistical power in SEM, particularly with EQS, the Lagrange Multiplier and Wald tests can be used for individual parameters (Kline, 2005). For samples with unequal group sizes, lack of rejection of the null hypothesis may be due to unequal sample sizes, lack of large mean differences or both (Kline, 2005). This was likely of little effect in this study due to very nearly equal number per group. According to Hancock & Freeman (2001) for a RMSEA value of 0.00, with 45 degrees of freedom (the most complex model in this study) at a .8 power level, 275 participants would be needed per group in this study. Larger numbers would be needed for RMSEA values up to the .05 level (Hancock & Freeman, 2001). At best, of all models tested in this study, power was .26 per these guidelines.

Steps were taken to improve measurement compared to prior studies. Height and weight were directly measured rather than obtained by self report. Screening methods were used to rule out pregnancy and diabetes, eliminating the influence of those potentially unknown conditions on measured variables. Additional screening for other medical conditions increased the confidence that body composition was not strongly influenced by a medical condition, more so than other factors being examined.

Future Directions

Despite the limitations previously discussed, this study provides useful data to inform future studies and clinical practice. Future replications and expansions of this study will benefit from knowledge gained in this effort regarding procedural and statistical methods. Additional efforts to determine where group differences may or may not exist, especially in most the vulnerable populations, will likely improve treatment and prevention of obesity for all.

APPENDIX

TABLES AND FIGURES

Table 1. Descriptive statistics and one-way analysis of variance between groups, N=210

	African American (n=100)		Caucasian (n=110)			
	M	SD	M	SD	*F*(1,209), *p*	Effect size (Cohen's d), Power (η)
Age	35.44	6.68	34.69	7.40	.59, .44	.05, .56
Range	24 – 50		24 – 50			
BMI	29.85	7.16	28.11	7.44	2.94, .09	.12, .73
Range	17.9 – 51.8		17.4 – 52.5			
Education	5.54	0.88	5.97	0.86	12.93, <.001*	.24, .94
Range	4 – 7		3 – 7			
Occupation	5.81	1.87	6.14	1.89	1.59, .21	.09, .65
Range	1 – 9		1 – 9			
Income (% poverty)†	304.16	192.20	452.36	277.04	19.90, <.001*	.30, .95
Range	30.8 – 1017.1		0.0 – 1285.7			
Social Desirability	5.95	2.17	4.94	2.30	10.73, .001*	.22, .92
Range	1 – 10		0 – 10			

* Significant with Bonferroni correction, α= .008, two-tailed

† Groups had unequal variances based on Levene's statistic. Variance for the group with the larger n was used for power calculation.

Table 2. 2008 United States Poverty Guidelines, Department of Health and Human Services

Persons in Family or Household	48 Contiguous States and District of Columbia
1	$10,400
2	$14,000
3	$17,600
4	$21,200
5	$24,000
6	$28,000
7	$32,000
8	$35,600

Table 3. Correlations (Pearson's *r*, *p*), all participants, N=210

	Body Mass Index
Education	-.280, p <.001
Occupation	-.183, p = .008
Income (% poverty)	-.230, p = .001
Cognitive Restraint	.040, p = .561
Disinhibition	.450, p < .001
Hunger	.261, p < .001
Social Desirability	-.069, p = .321

Table 4. Eating Inventory items to parcels organization used to test Hypothesis #1

Parcel	Item Numbers
Cognitive Restraint Parcel 1	14, 35, 51
Cognitive Restraint Parcel 2	6, 33, 37, 44
Cognitive Restraint Parcel 3	28, 30, 32, 42
Disinhibition Parcel 1	11, 20, 50
Disinhibition Parcel 2	9, 13, 45, 49
Disinhibition Parcel 3	15, 27, 36
Hunger Parcel 1	3, 5, 12
Hunger Parcel 2	24, 26, 34

Table 5. Between factor correlations for overall and group samples

Hypothesis/Factor	Overall Sample N=210	African American n=100	Caucasian n=110
Hypothesis 1			
Cognitive Restraint, Disinhibition	.166	.026	.172
Cognitive Restraint, Hunger	-.158	-.255	-.092
Disinhibition, Hunger	.570	.650	.475
Hypothesis 2a			
Flexible Restraint, Rigid Restraint	.977	1.000	.887
Hypothesis 2b			
Flexible Restraint, Rigid Restraint	.169	.093	.285
Hypothesis 3 – Cognitive Restraint			
Strategic Dieting Behavior, Attitude to Self Regulation	.862	1.000	.795
Strategic Dieting Behavior, Avoidance of Fattening Foods	.601	.607	.550
Attitude to Self Regulation, Avoidance of Fattening Foods	.890	1.000	.843
Hypothesis 4 - Disinhibition			
Habitual, Emotional	.660	.553	.651
Habitual, Situational	.841	.927	.808
Emotional, Situational	.705	.666	.681
Hypothesis 5 - Hunger			
Internal Cues, External Cues	.839	.993	.684

Table 6. Factor loadings for overall and group samples with no covariate

Hypothesis/Factor/Parcel		Overall Sample N=210	African American n=100	Caucasian n=110
Hypothesis 1	Cognitive Restraint Parcel 1	.787	.753	.840
	Cognitive Restraint Parcel 2	.799	.757	.791
	Cognitive Restraint Parcel 3	.822	.810	.808
	Disinhibition Parcel 1	.794	.787	.778
	Disinhibition Parcel 2	.837	.788	.860
	Disinhibition Parcel 3	.859	.798	.909
	Hunger Parcel 1	.738	.900	.592
	Hunger Parcel 2	.712	.638	.867
Hypothesis 2a	Flexible Restraint 1	.611	.554	.648
	Flexible Restraint 2	.734	.716	.785
	Rigid Restraint 1	.679	.609	.720
	Rigid Restraint 2	.734	.718	.752
Hypothesis 2b	Flexible Restraint 1	.596	.759	.592
	Flexible Restraint 2	.990	.756	1.000
	Rigid Restraint 1	.603	.594	.597
	Rigid Restraint 2	.651	.695	.642
	Rigid Restraint 3	.538	.492	.569
Hypothesis 3 – Cognitive Restraint	Parcel 1	.550	.580	.508
	Parcel 2	.720	.719	.746
	Parcel 3	.529	.443	.544
	Parcel 4	.680	.534	.764
	Parcel 5	.710	.805	.609
	Parcel 6	.671	.580	.714
Hypothesis 4 – Disinhibition	Parcel 1	.719	.463	.871
	Parcel 2	.661	.677	.680
	Parcel 3	.611	.616	.562
	Parcel 4	.962	1.000	.970
	Parcel 5	.675	.677	.654
	Parcel 6	.598	.579	.585
Hypothesis 5 – Hunger	Parcel 1	.722	.734	.719
	Parcel 2	.726	.777	.639
	Parcel 3	.664	.651	.737
	Parcel 4	.542	.528	.503
	Parcel 5	.591	.578	.601
	Parcel 6	.343	.352	.341

Table 7. Factor loadings for overall and group samples with body mass index as covariate

Hypothesis/Factor/Parcel		Overall Sample N=210	African American n=100	Caucasian n=110
Hypothesis 1	Cognitive Restraint Parcel 1	.783	.759	.831
	Cognitive Restraint Parcel 2	.797	.761	.783
	Cognitive Restraint Parcel 3	.827	.814	.810
	Disinhibition Parcel 1	.785	.770	.769
	Disinhibition Parcel 2	.837	.797	.857
	Disinhibition Parcel 3	.870	.823	.912
	Hunger Parcel 1	.752	.888	.602
	Hunger Parcel 2	.717	.634	.895
Hypothesis 2a	Flexible Restraint 1	.608	.554	.684
	Flexible Restraint 2	.756	.726	.772
	Rigid Restraint 1	.724	.611	.735
	Rigid Restraint 2	.672	.740	.742
Hypothesis 2b	Flexible Restraint 1	.632	.795	.574
	Flexible Restraint 2	.933	.754	1.000
	Rigid Restraint 1	.609	.612	.596
	Rigid Restraint 2	.645	.663	.646
	Rigid Restraint 3	.553	.526	.568
Hypothesis 3 – Cognitive Restraint	Parcel 1	.553	.565	.533
	Parcel 2	.729	.731	.746
	Parcel 3	.539	.464	.566
	Parcel 4	.664	.547	.725
	Parcel 5	.722	.827	.622
	Parcel 6	.677	.594	.719
Hypothesis 4 – Disinhibition	Parcel 1	.725	.467	.865
	Parcel 2	.653	.659	.676
	Parcel 3	.591	.592	.559
	Parcel 4	.957	1.000	.951
	Parcel 5	.670	.672	.650
	Parcel 6	.571	.553	.571
Hypothesis 5 – Hunger	Parcel 1	.711	.732	.712
	Parcel 2	.725	.804	.626
	Parcel 3	.670	.673	.709
	Parcel 4	.530	.526	.491
	Parcel 5	.604	.603	.629
	Parcel 6	.336	.383	.292

Table 8. Factor loadings for overall and group samples with combined covariate*

Hypothesis/Factor/Parcel		Overall Sample N=210	African American n=100	Caucasian n=110
Hypothesis 1	Cognitive Restraint Parcel 1	.793	.775	.837
	Cognitive Restraint Parcel 2	.801	.759	.792
	Cognitive Restraint Parcel 3	.813	.785	.807
	Disinhibition Parcel 1	.791	.773	.779
	Disinhibition Parcel 2	.834	.791	.853
	Disinhibition Parcel 3	.867	.821	.913
	Hunger Parcel 1	.759	.881	.623
	Hunger Parcel 2	.719	.634	.886
Hypothesis 2a	Flexible Restraint 1	.600	.539	.655
	Flexible Restraint 2	.743	.722	.767
	Rigid Restraint 1	.720	.602	.731
	Rigid Restraint 2	.667	.726	.752
Hypothesis 2b	Flexible Restraint 1	.578	.866	.577
	Flexible Restraint 2	1.000	.656	1.000
	Rigid Restraint 1	.614	.597	.623
	Rigid Restraint 2	.652	.717	.622
	Rigid Restraint 3	.545	.488	.579
Hypothesis 3 – Cognitive Restraint	Parcel 1	.547	.535	.533
	Parcel 2	.714	.741	.724
	Parcel 3	.536	.449	.567
	Parcel 4	.669	.535	.741
	Parcel 5	.711	.825	.606
	Parcel 6	.673	.572	.724
Hypothesis 4 – Disinhibition	Parcel 1	.724	.472	.865
	Parcel 2	.679	.717	.691
	Parcel 3	.600	.588	.566
	Parcel 4	.957	1.000	.962
	Parcel 5	.668	.691	.631
	Parcel 6	.598	.591	.583
Hypothesis 5 – Hunger	Parcel 1	.713	.727	.716
	Parcel 2	.724	.783	.631
	Parcel 3	.670	.668	.732
	Parcel 4	.537	.530	.500
	Parcel 5	.586	.589	.594
	Parcel 6	.328	.332	.328

* Combined covariate – summed z-scores of body mass index (BMI), education level and social desirability. Tested separately from BMI alone as covariate due to software limitations.

Table 9. Hypothesis 1 model testing summary

	Scaled χ^2 (df)	p	MFI	CFI	RMSEA	90% C.I.
No covariates						
Overall Model	23.20 (17)	.1430	.985	.991	.042	.000 - .080
Between Groups Constrained Model	61.41 (45)	.0522	.962	.974	.059	.000 - .093
Between Groups Unconstrained Model	43.92 (34)	.1187	.977	.985	.053	.000 - .093
χ^2 Difference	17.49 (11)	.0942				
BMI as covariate						
Overall Model	23.01 (17)	.1490	.986	.990	.040	.000 - .080
Between Groups Constrained Model	60.35 (45)	.0627	.964	.975	.057	.000 - .092
Between Groups Unconstrained Model	44.58 (34)	.1058	.975	.982	.055	.000 - .095
χ^2 Difference	15.76 (11)	.1503				
Combined covariate*						
Overall Model	20.81 (17)	.2347	.991	.994	.033	.000 - .074
Between Groups Constrained Model	56.04 (45)	.1252	.974	.981	.049	.000 - .085
Between Groups Unconstrained Model	39.88 (34)	.2251	.986	.990	.041	.000 - .085
χ^2 Difference	16.16 (11)	.1353				

MFI – McDonald's fit index, CFI – Comparative fit index, RMSEA – Root-mean squared error of approximation, 90% C.I. – 90 percent confidence interval of the RMSEA value

*Combined covariate = Summed z-scores for body mass index, Social Desirability and Education rank

Table 10. Hypothesis 2a model testing summary – original Cognitive Restraint items only

	Scaled χ^2 (df)	p	MFI	CFI	RMSEA	90% C.I.
No covariates						
Overall Model	5.33 (4)	.2550	.996	.994	.040	.000 - .118
Between Groups Constrained Model	8.20 (7)	.3156	.997	.994	.041	.000 - .131
Between Groups Unconstrained Model	3.37 (2)	.1851	.997	.993	.081	.000 - .226
χ^2 Difference	4.83 (5)	.4370				
BMI as covariate						
Overall Model	6.73 (4)	.1507	.994	.987	.057	.000 - .129
Between Groups Constrained Model	9.45 (7)	.2221	.994	.987	.058	.000 - .141
Between Groups Unconstrained Model	3.59 (2)	.1660	.996	.992	.087	.000 - .230
χ^2 Difference	5.86 (5)	.3201				
Combined covariate*						
Overall Model	7.90 (4)	.0954	.991	.980	.068	.000 - .138
**Between Groups Constrained Model	7.99 (6)	.2386	.995	.989	.057	.000 - .147
Between Groups Unconstrained Model	4.31 (2)	.1159	.995	.988	.105	.000 - .244
χ^2 Difference	3.68 (4)	.4510				

MFI – McDonald’s fit index, CFI – Comparative fit index, RMSEA – Root-mean squared error of approximation, 90% C.I. – 90 percent confidence interval of the RMSEA value

*Combined covariate = Summed z-scores for body mass index, Social Desirability and Education rank

** Only factor loadings were constrained to be equal. Model with equal factors failed to converge.

Table 11. Hypothesis 2b model testing summary – supplemental Cognitive Restraint items only

	Scaled χ^2 (df)	p	MFI	CFI	RMSEA	90% C.I.
No covariates						
Overall Model	8.61 (4)	.0716	.989	.973	.074	.000 - .143
Between Groups Constrained Model	16.73 (14)	.2711	.994	.984	.043	.000 - .108
Between Groups Unconstrained Model	12.38 (8)	.1350	.990	.974	.073	.000 - 147
χ^2 Difference	4.35 (6)	.6294				
BMI as covariate						
Overall Model	5.07 (4)	.2804	.997	.994	.036	.000 - .115
Between Groups Constrained Model	12.96 (14)	.5294	1.002	1.000	.000	.000 - .088
Between Groups Unconstrained Model	8.85 (8)	.3548	.998	.995	.032	.000 - .122
χ^2 Difference	4.11 (6)	.6618				
Combined covariate*						
Overall Model	4.12 (4)	.3898	1.000	.999	.012	.000 - .106
Between Groups Constrained Model	13.53 (14)	.4852	1.001	1.000	.000	.000 - .092
Between Groups Unconstrained Model	7.90 (8)	.4437	1.000	1.000	.000	.000 - .113
χ^2 Difference	5.63 (6)	.4659				

MFI – McDonald's fit index, CFI – Comparative fit index, RMSEA – Root-mean squared error of approximation, 90% C.I. – 90 percent confidence interval of the RMSEA value

*Combined covariate = Summed z-scores for body mass index, Social Desirability and Education rank

Table 12. Hypothesis 3 model testing summary – Cognitive Restraint subfactors

	Scaled χ^2 (df)	p	MFI	CFI	RMSEA	90% C.I.
No covariates						
Overall Model	2.94 (6)	.8164	1.007	1.000	.000	.000 - .055
Between Groups Constrained Model	20.31 (21)	.5020	1.002	1.000	.000	.000 - .080
Between Groups Unconstrained Model	8.01 (12)	.7843	1.010	1.000	.000	.000 - .067
χ^2 Difference	12.30 (9)	.1969				
BMI as covariate						
Overall Model	3.03 (6)	.8055	1.007	1.000	.000	.000 -.057
Between Groups Constrained Model	21.23 (21)	.4450	.999	.999	.010	.000 -.083
Between Groups Unconstrained Model	8.43 (12)	.7510	1.009	1.000	.000	.000 - .071
χ^2 Difference	12.80 (9)	.1719				
Combined covariate*						
Overall Model	3.12 (6)	.7932	1.007	1.000	.000	.000 -.058
Between Groups Constrained Model	18.43 (21)	.6219	1.006	1.000	.000	.000 - .071
Between Groups Unconstrained Model	7.80 (12)	.8003	1.010	1.000	.000	.000 -.065
χ^2 Difference	10.63 (9)	.3019				

MFI – McDonald's fit index, CFI – Comparative fit index, RMSEA – Root-mean squared error of approximation, 90% C.I. – 90 percent confidence interval of the RMSEA value

*Combined covariate = Summed z-scores for body mass index, Social Desirability and Education rank

Table 13. Hypothesis 4 model testing summary – Disinhibition subfactors

	Scaled χ^2 (df)	p	MFI	CFI	RMSEA	90% C.I.
No covariates						
Overall Model	8.16 (6)	.2267	.995	.994	.041	.000 -.105
Between Groups Constrained Model	29.54 (21)	.1017	.980	.972	.063	.000 - .111
Between Groups Unconstrained Model	15.90 (12)	.1960	.991	.987	.056	.000 - .121
χ^2 Difference	13.64 (9)	.1357				
BMI as covariate						
Overall Model	9.67 (6)	.1394	.991	.988	.054	.000 -.114
Between Groups Constrained Model	28.12 (21)	.1369	.983	.975	.057	.000 -.107
Between Groups Unconstrained Model	15.19 (12)	.2310	.992	.989	.051	.000 - .117
χ^2 Difference	12.92 (9)	.1663				
Combined covariate*						
Overall Model	7.93 (6)	.2429	.995	.994	.039	.000 - .103
Between Groups Constrained Model	28.97 (21)	.0883	.979	.970	.066	.000 - .114
Between Groups Unconstrained Model	13.42 (12)	.2620	.994	.992	.046	.000 - .103
χ^2 Difference	15.55 (9)	.0769				

MFI – McDonald's fit index, CFI – Comparative fit index, RMSEA – Root-mean squared error of approximation, 90% C.I. – 90 percent confidence interval of the RMSEA value

*Combined covariate = Summed z-scores for body mass index, Social Desirability and Education rank

Table 14. Hypothesis 5 model testing summary – Hunger subfactors

	Scaled χ^2 (df)	p	MFI	CFI	RMSEA	90% C.I.
No covariates						
Overall Model	6.60 (8)	.5807	1.003	1.00	.000	.000 - .071
**Between Groups Constrained Model	33.45 (22)	.0558	.973	.950	.071	.000 - .116
Between Groups Unconstrained Model	28.56 (16)	.0271†	.957	.945	.087	.029 - .137
χ^2 Difference	4.89 (8)	.4335				
BMI as covariate						
Overall Model	6.25 (8)	.6195	1.004	1.000	.000	.000 - .068
Between Groups Constrained Model	34.00 (23)	.0521	.972	.945	.071	.000 - .115
Between Groups Unconstrained Model	27.54 (16)	.0359†	.973	.948	.083	.021 -.134
χ^2 Difference	7.46 (7)	.3826				
Combined covariate*						
Overall Model	5.93 (8)	.6549	1.005	1.000	.000	.000 - .066
Between Groups Constrained Model	32.51 (23)	.0901	.978	.956	.063	.000 - .109
Between Groups Unconstrained Model	25.29 (16)	.0649	.978	.957	.075	.000 - .127
χ^2 Difference	7.22 (7)	.4063				

MFI – McDonald's fit index, CFI – Comparative fit index, RMSEA – Root-mean squared error of approximation, 90% C.I. – 90 percent confidence interval of the RMSEA value

*Combined covariate = Summed z-scores for body mass index, Social Desirability and Education rank

** Only factor loadings constrained to be equal. Model with equal factor variances indicated χ^2 (23) $p = .0357$.

† Significant at α=.05.

Table 15. Hypothesis 5 Hunger subfactors with additional single covariates

	Scaled χ^2 (df)	p	MFI	CFI	RMSEA	90% C.I.
Education as covariate						
Overall Model	4.48 (8)	.8118	1.008	1.000	.000	.000 - .051
Between Groups Constrained Model	26.26 (23)	.2887	.992	.980	.037	.000 - .091
*Between Groups Unconstrained Model	16.59 (16)	.4123	.999	.996	.019	.000 - .093
χ^2 Difference	9.67 (7)	.2081				
Social desirability as covariate						
Overall Model	8.08 (8)	.4255	1.000	1.000	.007	.000 - .081
Between Groups Constrained Model	34.49 (23)	.0585	.973	.935	.069	.000 - .114
Between Groups Unconstrained Model	29.34 (16)	.0217†	.969	.925	.090	.021 -.134
χ^2 Difference	5.14 (7)	.6429				

MFI – McDonald's fit index, CFI – Comparative fit index, RMSEA – Root-mean squared error of approximation, 90% C.I. – 90 percent confidence interval of the RMSEA value

* Factor 1 and Factor 2 constrained at upper limit of 1.0 in African American group. Correlation between factors in Caucasian group = .615.

† Significant at α=.05.

Figure 1. Overweight and obesity prevalence rates in percentages by race and gender (NHANES - National Health and Nutrition Examination Survey by Centers for Disease Control)

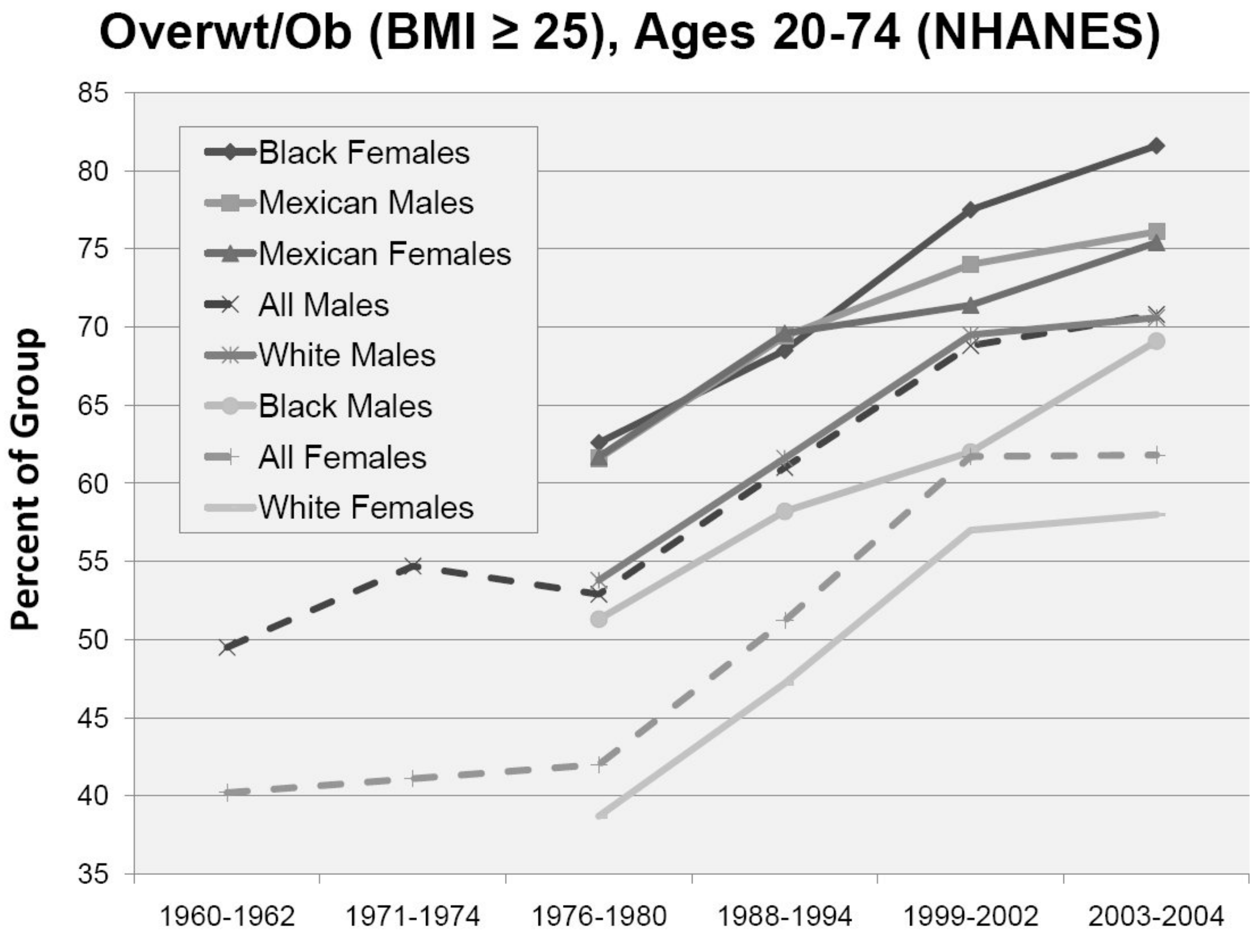


Figure 2. Theory of Planned Behavior (Drawing by author; general model copyright, Icek Azjen, 2006)

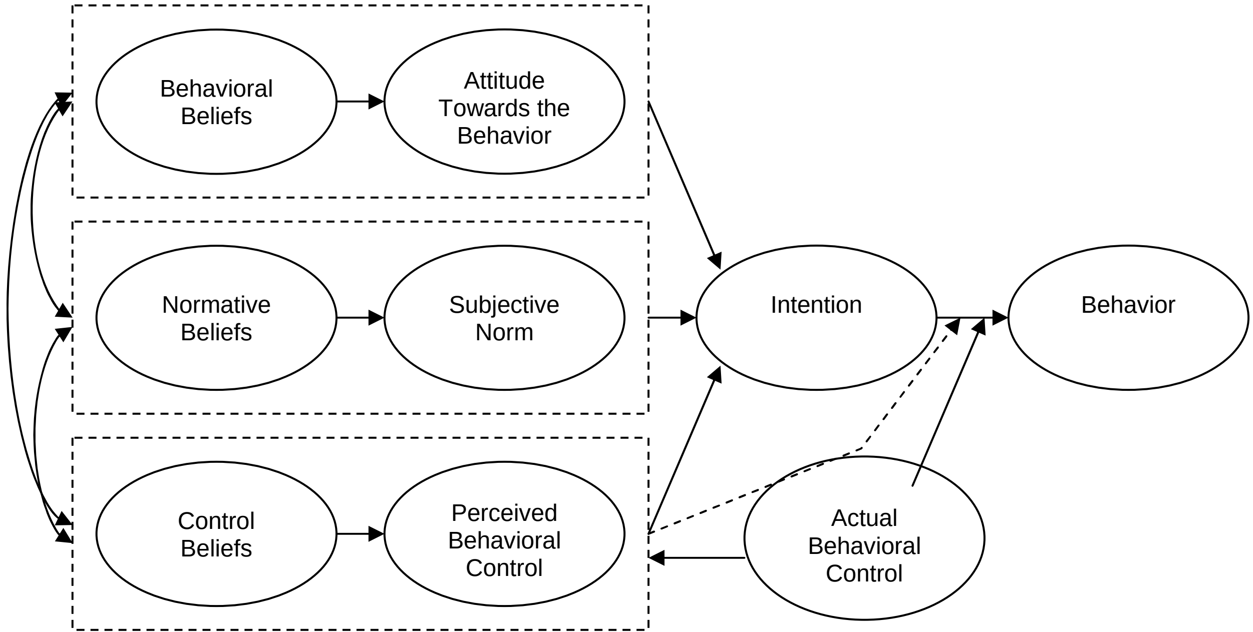


Figure 3. Simplified Structural Model of the Eating Inventory

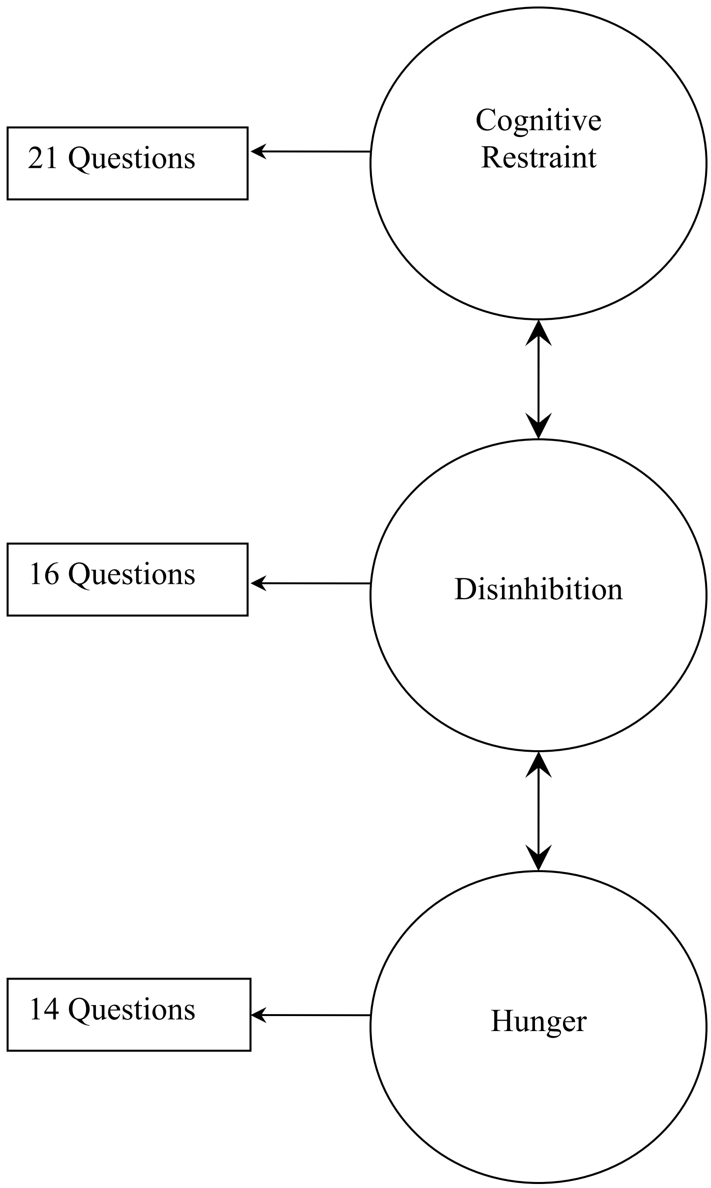
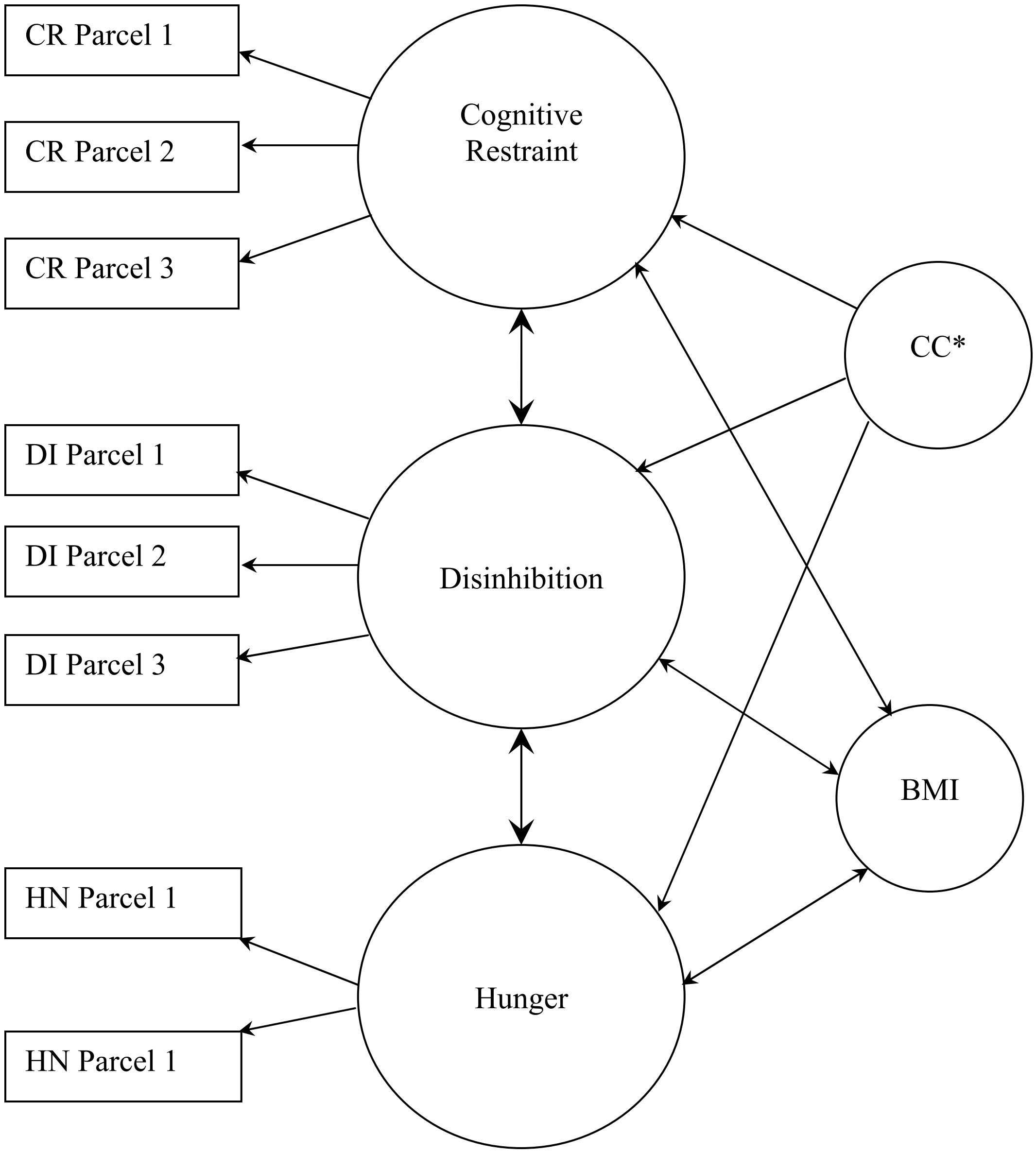
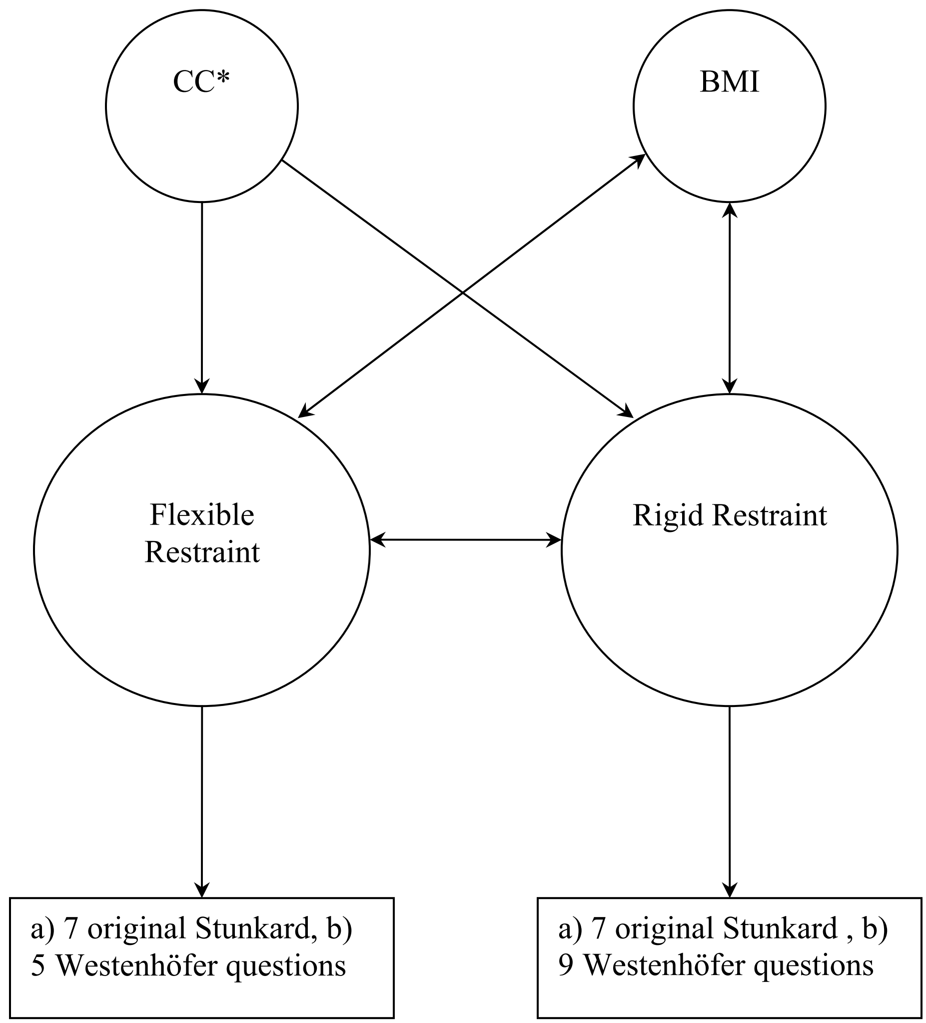


Figure 4. Structural model of the Eating Inventory tested in Hypothesis 1



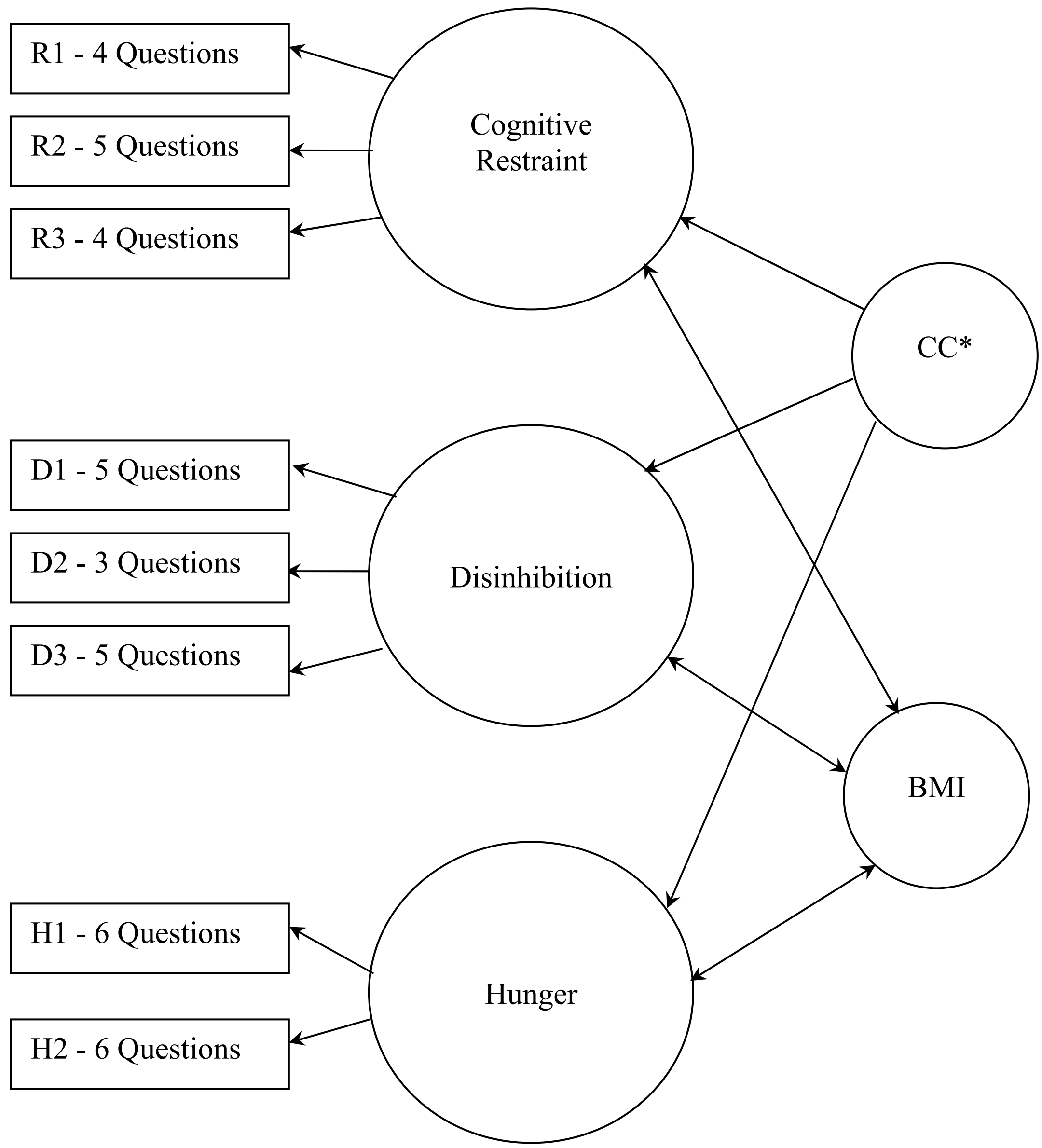
*Combined covariate = Summed z-scores for body mass index, Social Desirability and Education rank

Figure 5. Structural model of the Restraint subfactors (Westenhöfer et al., 1999) tested in hypotheses 2a and 2b



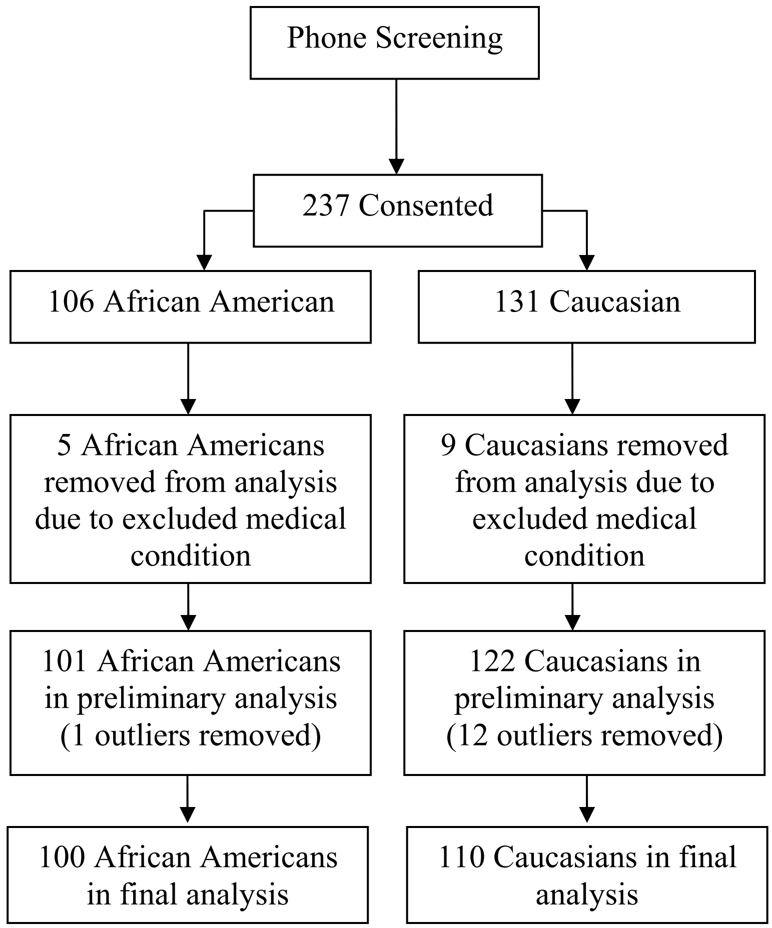
*Combined covariate = Summed z-scores for body mass index, Social Desirability and Education rank

Figure 6. Structural model of the proposed subfactors (Bond, et al., 2001) tested in hypotheses 3 - 5



*Combined covariate = Summed z-scores for BMI, Social Desirability and Education rank

Figure 7. CONSORT Diagram



REFERENCES

Ajzen, I. (1988). *Attitudes, personality and behavior*. Buckingham, England: Open University Press.

Ajzen, I. (1991). The theory of planned behavior. *Organizational behavior and human decision processes, 50,* 179-211.

Ajzen, I. (2001). Nature and operation of attitudes. *Annual Review of Psychology, 52,* 27-58.

Ajzen, I. (2002). Perceived behavioral control, self-efficacy, locus of control, and the theory of planned behavior. *Journal of Applied Social Psychology, 32,* 665-683.

Ajzen, I. & Fishbein, M. (1980). *Understanding attitudes and predicting social behavior*. Englewood Cliffs, NJ: Prentice-Hall.

American Psychiatric Association (2000). *Diagnostic and statistical manual of mental disorders (4th ed.)*. Washington, D.C.: Author.

Anderson, J.L., Crawford, C.B., Nadeau, J. & Lindberg, T. (1992). Was the Duchess of Windsor right? A cross-cultural review of the socioecology of ideals of female body shape. *Ethology and Sociobiology, 13,* 197-227.

Atlas, J.G., Smith, G.T., Hohlstein, L.A., McCarthy, D.M. & Kroll, L.S. (2002). Similarities and differences between Caucasian and African American college women on eating and dieting expectancies, bulimic symptoms, dietary restraint, and disinhibition. *International Journal of Eating Disorders, 32,* 326-334.

Bagozzi, R.P. & Heatherton, T.F. (1994). A general approach to representing multifacted personality constructs: Application to state self-esteem. *Structural Equation Modeling, 1,* 35-67.

Baranowski, T., Cullen, K.W. & Baranowski, J. (1999). Psychosocial correlates of dietary intake: Advancing dietary intervention. *Annual Review of Nutrition, 19,* 17-40.

Bentler, P. M. (2006). *EQS 6 Structural Equations Program Manual*. Encino, CA: Multivariate Software, Inc.

Berridge, K.C. (2004). Motivation concepts in behavioral neuroscience. *Physiology and Behavior, 81,* 179-209.

Björvell, H., Rush, A.J. & Stunkard, A.J. (1986). Obesity, weight loss and dietary restraint. *Internation Journal of Eating Disorders, 5,* 727-734.

Boerner, L.M., Spillane, N.S., Anderson, K.G. & Smith, G.T. (2004). Similarities and differences between women and men on eating disorder risk factors and symptom measures. *Eating Behaviors, 5,* 209-222.

Bond, M.J., McDowell, A.J. & Wilkinson, J.Y. (2001). The measurement of dietary restraint, disinhibition and hunger: An examination of the factor structure of the Three Factor Eating Questionnaire (TFEQ). *International Journal of Obesity and Related Metabolic Disorders, 25,* 900-906.

Bouchard, C. (1996). Genetics of obesity in humans: current issues. In D.J.Chadwick & G. Cardew (Eds.), *The origins and consequences of obesity* (pp. 108-117). John Wiley & Sons.

Braveman, P.A., Cubbin, C., Egerter, S., Chideya, S., Marchi, K.S., Metzler, M. et al. (2005). Socioeconomic status in health research: One size does not fit all. *Journal of the American Medical Association, 294,* 2879-2888.

Bray, G.A., Nielsen, S.J. & Popkin, B.M. (2004). Consumption of high-fructose corn syrup in beverages may play a role in the epidemic of obesity. *American Journal of Clinical Nutrition, 79,* 537-543.

Browman, C.L. (1996). The health consequences of racial discrimination: A study of African Americans. *Ethnicity and Disease, 6,* 148-153.

Browne, M. W. & Cudek, R. (1993). Alternative ways of assessing model fit. In Bollen.K.A. & J. S. Long (Eds.), *Testing structural equations models* (pp. 136-162). Newbury Park, CA: Sage.

Brownell, K.D. & Wadden, T.A. (1992). Etiology and treatment of obesity: Understanding a serious, prevalent, and refractory disorder. *Journal of Consulting and Clinical Psychology, 60,* 505-517.

Cachelin, F.M., Rebeck, R.M., Chung, G.H. & Pelayo, E. (2002). Does ethnicity influence body-size preference? A comparison of body image and body size. *Obesity Research, 10,* 158-166.

Caldwell, M.B., Brownell, K.D. & Wilfley, D.E. (1997). Relationship of weight, body dissatisfaction, and self-esteem in African American and White female dieters. *International Journal of Eating Disorders, 22,* 127-130.

Centers for Disease Control (2006). Health, United States, 2005: With chartbook on trends in the health of Americans. Retrieved March 20, 2007 from: http://www.ncbi.nlm.nih.gov.proxy.hsc.unt.edu/books/bv.fcgi?rid=healthus05.table.421

Cheung, G.W. & Rensvold, R.B. (2002). Evaluating goodness-of-fit indices for testing measurement invariance. *Structural Equation Modeling, 9,* 233-255.

Clark, R., Anderson, N.B., Clark, V.R. & Williams, D.R. (1999). Racism as a stressor for African Americans: A biopsychosocial model. *American Psychologist, 54,* 805-816.

Clark, V.R. & Harrell, J.P. (1982). The relationship among type A behavior, styles used in coping with racism, and blood pressure. *Journal of Black Psychology, 8,* 89-99.

Conner, M., Norman, P. & Bell, R. (2002). The theory of planned behavior and healthy eating. *Health Psychology, 21,* 194-201.

Devaney, B. & Moffitt, R. (1991). Dietary effects of the food stamp program. *American Journal of Agricultural Economics, 73,* 202-211.

Drewnowksi, A., Riskey, D. & Desor, J.A. (1982). Feeding fat yet unconcerned: Self-reported overweight and the restraint scale. *Appetite: Journal for Intake Research, 3,* 273-279.

Dunn, G., Everitt, B., & Pickles, A. (1993). *Modeling covariances and latent variables using EQS*. Boca Raton, FL: Chapman & Hall/CRC Press.

Esparza, J., Fox, C., Harper, I.T., Bennett, P.H., Schulz, L.O., Valencia, M.E. et al. (2000). Daily energy expenditure in Mexican and USA Pima indians: Low physical activity as a possible cause of obesity. *International Journal of Obesity and Related Metabolic Disorders, 24,* 55-59.

Fernando, S. (1984). Racism as a cause of depression. *International Journal of Social Psychiatry, 30,* 41-49.

First, M. B., Spitzer, R. L., Gibbon, M., & Williams, J. B. W. (1997). *Structure Clinical Interview for DSM-IV Axis I Disorders - Clinician Version (SCID-CV)*. Washington, DC: American Psychiatric Press.

Fishbein, M. & Ajzen, I. (1975). *Belief, attitude, intention, and behavior*. New York: Wiley.

Ganley, R.M. (1988). Emotional eating and how it relates to dietary restraint, disinhibition and perceived hunger. *International Journal of Eating Disorders, 7,* 635-647.

Gardner, R.E. & Hausenblas, H.A. (2005). Exercise and diet determinants of overweight women participating in an exercise and diet program: a prospective examination of the theory of planned behavior. *Women & Health, 42,* 37-62.

Gibson, D. (2003). Food stamp participation is positively associated to obesity in low income women. *Journal of Nutrition, 133,* 2225-2231.

Goldstone, A.P. (2006). The hypothalamus, hormones, and hunger: Alterations in human obesity and illness. *Progress in Brain Research, 153,* 57-73.

Grilo, C. M. (2006). *Eating and weight disorders*. New York: Psychology Press.

Hancock, G.R. & Freeman, M.J. (2001). Power and sample size for the root mean square error of approximation test of not close fit in structural equation modeling. *Educational and Psychological Measurement, 61,* 741-758.

Herman, C.P. & Mack, D. (1975). Restrained and unrestrained eating. *Journal of Personality, 43,* 647-660.

Herman, C.P. & Polivy, J. (1975). Anxiety, restraint and eating behavior. *Journal of Abnormal Psychology, 84,* 666-672.

Herman, C. P. & Polivy, J. (1980). Restrained eating. In A.J.Stunkard (Ed.), *Obesity* (pp. 208-225). Philadelphia: Saunders.

Hibscher, J.A. & Herman, C.P. (1977). Obesity, dieting, and the expression of "obese" characteristics. *Journal of Comparative and Physiological Psychology, 91,* 374-380.

Hollingshead, A. B. (1975). Four factor index of social status. Department of Sociology, Yale University, New Haven, CT: http://www.yale.edu/sociology/faculty/docs/hollingshead_socStat4factor.pdf

Hoyle, R. H. (1995). Writing about structural equation models. In R.H.Hoyle (Ed.), *Structural equation modeling concepts, issues, and applications* (pp. 158-176). Thousand Oaks: Sage Publications.

Hyland, M.E., Irvine, S.H., Thacker, C., Dann, P.L. & Dennis, I. (1989). Psychometric analysis of the Stunkard-Messick Eating Questionnaire (SMEQ) and comparison with the Dutch

Eating Behavior Questionnaire (DEBQ). *Current Psychology: Research and Reviews, 8,* 233.

Johnson, E.H. & Browman, C.L. (1987). The relationship of anger expression to health problems among Black Americans in a national survey. *Journal of Behavioral Medicine, 10,* 103-116.

Kaiser, K.A. (2009). A comparison of Eating Inventory Scores to BMI between weight groups and genders. *Unpublished data*.

Kaplan, H.I. & Kaplan, H.S. (1957). The psychosomatic concept of obesity. *Journal of Nervous and Mental Disease, 125,* 181-201.

Karlsson, J., Persson, L.-O., Sjostrom, L. & Sullivan, M. (2000). Psychometric properties and factor structure of the Three-Factor Eating Questionnaire (TFEQ) in obese men and women: Results from the Swedish Obese Subjects (SOS) study. *International Journal of Obesity, 24,* 1715-1725.

Keesey, R.E. & Powley, T.L. (1986). The regulation of body weight. *Annual Review of Psychology, 37,* 109-133.

Kline, R. B. (2005). *Principles and practice of structural equation modeling*. (2nd ed ed.) New York: Guilford Press.

Kopelman, P. G., Caterson, I. D., & Dietz, W. H. (2005). *Clinical obesity in adults and children*. (2nd ed.) Oxford, UK: Blackwell Publishing.

Kremers, S.P., Visscher, V.L., Seidell, J.C., van Mechlen, W. & Brug, J. (2009). Cognitive determinants of energy balance-related behaviours: measurement issues. *Sports Medicine, 35,* 923-933.

Kumanyika, S. (2002). The minority factor in the obesity epidemic. *Ethnicity and Disease, 12,* 316-319.

Kumanyika, S.K. (1993). Special issues regarding obesity in minority populations. *Annals of Internal Medicine, 119,* 650-654.

Kumanyika, S.K. (1994). Obesity in minority populations: An epidemiologic assessment. *Obesity Research, 2,* 166-182.

Kumanyika, S.K. (2008). Environmental influences on childhood obesity: Ethnic and cultural influences in context. *Physiology and Behavior, 94,* 61-70.

Kumanyika, S.K., Gary, T.L., Lancaster, K.J., Samuel-Hodge, C.D., Banks-Wallace, J., Beech, B.M. et al. (2005). Achieving healthy weight in African-American communities: Research perspectives and priorities. *Obesity Research, 13,* 2037-2047.

Levitsky, D.A. (2005). The future of school feeding programs. *Food Nutrition Bulletin, 26,* S286-287.

Levitsky, D.A., Obarzanek, E., Mrdjenovic, G. & Strupp, B.J. (2005). Imprecise control of energy intake: Absence of a reduction in food intake following overfeeding in young adults. *Physiology and Behavior, 84,* 669-695.

Levitsky, D.A. & Youn, T. (2004). The more food young adults are served, the more they overeat. *Journal of Nutrition, 134,* 2546-2549.

Little, T.D., Cunningham, W.A. & Shahar, G. (2002). To parcel or not to parcel: Exploring the question, weighing the merits. *Structural Equation Modeling, 9,* 151-173.

Lopez-Moreno, J.A., Gonzalez-Cuevas, G. & Navarro, M. (2006). The CB1 cannabinoid receptor antagonist rimonabant chronically prevents the nicotine-induced relapse to alcohol. *Neurobiology of Disease, 25,* 275-283.

Ma, L., Tataranni, P.A., Hanson, R.L., Infante, A.M., Kobes, S., Bogardus, C. et al. (2005). Variations in peptide YY and Y2 receptor genes are associated with severe obesity in Pima Indian men. *Diabetes, 54,* 1598-1602.

MacCallum, R.C., Widaman, K.F., Zhang, S. & Hong, S. (1999). Sample size in factor analysis. *Psychological Methods, 4,* 99.

Malik, V.S., Schulze, M.B. & Hu, F.B. (2006). Intake of sugar-sweetened beverages and weight gain: a systematic review. *American Journal of Clinical Nutrition, 84,* 274-278.

Marcus, M. & Wing, R. R. (1983). Binge eating and dietary restraint. In *Annual Meeting of the Society for Behavioral Medicine. Baltimore, MD.*

Mardia, K.V. (1970). Measures of multivariate skewness and kurtosis with applications. *Biometrika, 57,* 519-530.

Mazzeo, S.E., Aggen, S.H., Anderson, C., Tozzi, F. & Bulik, C.M. (2003). Investigating the structure of the Eating Inventory (Three-Factor Eating Questionnaire): A confirmatory approach. *Internation Journal of Eating Disorders, 34,* 255-264.

McDonald, R.P. (1989). An index of goodness-of-fit based on non-centrality. *Journal of Classification, 6,* 97-103.

Moore, L.V. & Diez Roux, A.V. (2006). Associations of neighborhood characteristics with the location and type of food stores. *American Journal of Public Health, 96,* 325-331.

Mrdjenovic, G. & Levitsky, D.A. (2005). Children eat what they are served: The imprecise regulation of energy intake. *Appetite, 43,* 273-282.

National Research Council Panel (2004). *Understanding racial and ethnic differences in health in late life: A research agenda*. Washington, D.C: National Academies Press.

Neumark-Sztainer, D. (1999). The social environments of adolescents: associations between socioenvironmental factors and health behaviors during adolescence. *Adolescent Medicine, 10,* 41-55.

Ogden, C.L., Carroll, M.D., Curtin, L.R., McDowell, M.A., Tabak, C.J. & Flegal, K.M. (2006). Prevalence of overweight and obesity in the United States, 1999-2004. *Journal of the American Medical Association, 295,* 1549-1555.

Ogden, C.L., Yanovski, S.Z., Carroll, M.D. & Flegal, K.M. (2007). The epidemiology of obesity. *Gastroenterology, 132,* 2087-2102.

Paykel, E.S. (1977). Depression and appetite. *Journal of Psychosomatic Research, 21,* 401-407.

Perusse, L., Rankinen, T., Zuberi, A., Chagnon, Y.C., Weisnagel, S.J., Argyropoulos, G. et al. (2005). The human obesity gene map: the 2004 update. *Obesity Research, 13,* 381-490.

Powell, L.M., Slater, S., Mirtcheva, D., Bao, Y. & Chaloupka, F.J. (2007). Food store availability and neighborhood characteristics in the United States. *Preventive Medicine, 44,* 189-195.

Powley, T.L., Chi, M.M., Schier, L.A. & Phillips, R.J. (2005). Obesity: should treatments target visceral afferents? *Physiology and Behavior, 86,* 698-708.

Pudel, V., Metzdorff, M. & Oetting, M. (1975). [The personality of obese persons in psychological tests with special consideration on latent obesity]. *Zeitschrift für Psychosomatische Medizin und Psychoanalyse, 21,* 345-361.

Ravussin, E., Valencia, M.E., Esparza, J., Bennett, P.H. & Schulz, L.O. (1994). Effects of a traditional lifestyle on obesity in Pima Indians. *Diabetes Care, 17,* 1067-1074.

Ricciardelli, L.A. & Williams, R.J. (1997). A two-factor model of dietary restraint. *Journal of Clinical Psychology, 53,* 123-131.

Rodin, J. (1981). Current status of the internal-external hypothesis for obesity: What went wrong? *American Psychologist, 36,* 361-372.

Rosenberger, P.H., Henderson, K.E. & Grilo, C.M. (2006). Correlates of body image dissatisfaction in extremely obese female bariatric surgery candidates. *Obesity Surgery, 1,* 1331-1336.

Rotter, J.B. (1975). Some problems and misconceptions related to the construct of internal versus external control of reinforcement. *Journal of Consulting and Clinical Psychology, 43,* 56-67.

Satorra, A. & Bentler, P.M. (1990). Model conditions for asymptotic robustness in the analysis of linear relations. *Computational Statistics and Data Analysis, 19,* 235-249.

Schacter, S. & Rodin, J. (1974). *Obese humans and rats*. Washington, D.C.: Erlbaum/Wiley.

Schifter, D.E. & Ajzen, I. (1985). Intention, perceived control, and weight loss: An application of the theory of planned behavior. *Journal of Personality and Social Psychology, 49,* 843-851.

Schwartz, M.W., Woods, S.C., Porte, D., Jr., Seeley, R.J. & Baskin, D.G. (2000). Central nervous system control of food intake. *Nature, 404,* 661-671.

Sharma, A.M. & Kushner, R.F. (2009). A proposed clinical staging system for obesity. *International Journal of Obesity, 33,* 289-295.

Siegrist, J. (2000). Place, social exchange and health: proposed sociological framework. *Social Science and Medicine, 51,* 1283-1293.

Smith, J.L. (2004). Food, health, and psychology: Competing recipes for research and understanding. *Journal of Health Psychology, 9,* 483-496.

Sobal, J. & Stunkard, A.J. (1989). Socioeconomic status and obesity: a review of the literature. *Psychological Bulletin, 105,* 260-275.

Steiger, J. H. (1989). *EzPATH: Causal modeling*. Evanston, IL: SYSTAT.

Stunkard, A. J. (1980). *Obesity*. Philadelphia: W.B. Saunders Company.

Stunkard, A. J. (1996). Socioeconomic status and obesity. In D.J.Chadwick & G. Cardew (Eds.), *The origins and consequences of obesity* (pp. 174-182). Hoboken, NJ: John Wiley & Sons.

Stunkard, A.J. & Messick, S. (1985). The three-factor eating questionnaire to measure dietary restraint, disinhibition and hunger. *Journal of Psychosomatic Research, 29,* 71-83.

Stunkard, A. J. & Messick, S. (1988). *Eating inventory manual*. San Antonio, Texas: Brace & Company.

Stunkard, A.J. & Sorensen, T.I. (1993). Obesity and socioeconomic status--a complex relation. *New England Journal of Medicine, 329,* 1036-1037.

Tabachnick, B. G. & Fidell, L. S. (2001). *Using multivariate statistics*. (4th ed.) Boston: Allyn and Bacon.

Thompson, B. (2003). Basic concepts in score reliability. In B.Thompson (Ed.), *Score reliability: Contemporary thinking on reliability issues* (pp. 3-23). Thousand Oaks, CA: Sage Publications.

U.S.Department of Health and Human Services (2009). The 2008 HHS Poverty Guidelines. Department of Health and Human Services Web Page: http://aspe.hhs.gov/poverty/08poverty.shtml

van Prooijen, J.W. & van der Kloot, W.A. (2001). Confirmatory analysis of exploratively obtained factor structures. *Educational and Psychological Measurement, 61,* 777-792.

van Strien, T., Frijters, J.E.R., Bergers, G.P.A. & Defares, P.B. (1986). The Dutch Eating Behavior Questionnaire (DBEQ) for assessment of restrained, emotional, and external eating behavior. *International Journal of Eating Disorders, 5,* 295-315.

Ver Ploeg, M., Mancino, L., & Lin, B. H. (2006). Food stamps and obesity: Ironic twist or complex puzzle? Retrieved March 20, 2007 from Amber Waves: http://www.ers.usda.gov/AmberWaves/February06/Features/feature4.htm

Weinsier, R.L., Hunter, G.R., Zuckerman, P.A. & Darnell, B.E. (2003). Low resting and sleeping energy expenditure and fat use do not contribute to obesity in women. *Obesity Research, 11,* 937-944.

Weinsier, R.L., Nelson, K.M., Hensrud, D.D., Darnell, B.E., Hunter, G.R. & Schutz, Y. (1995). Metabolic predictors of obesity: Contribution of resting energy expenditure, thermic effect of food, and fuel utilization to four-year weight gain of post-obese and never-obese women. *Journal of Clinical Investigation, 95,* 980-985.

Weissenburger, J., Rush, A.J., Giles, D.E. & Stunkard, A.J. (1986). Weight change in depression. *Psychiatry Research, 17,* 275-283.

West, S. G., Finch, J. F., & Curran, P. J. (1995). Structural equation models with non-normal variables. In R.H.Hoyle (Ed.), *Structural equation modeling: Concepts, issues, and applications* (pp. 56-75). Thousand Oaks, CA: Sage Publications.

Westenhöfer, J. (2005). Age and gender dependent profile of food choice. *Forum of Nutrition, 57,* 44-51.

Westenhöfer, J., Stunkard, A.J. & Pudel, V. (1999). Validation of the flexible and rigid control dimensions of dietary restraint. *International Journal of Eating Disorders, 26,* 53-64.

White, M.A., Whisenhunt, B.L., Williamson, D.A., Greenway, F.L. & Netemeyer, R.G. (2002). Development and validation of the food-craving inventory. *Obesity Research, 10,* 107-114.

Williams, L. B. & Considine, R. V. (2001). Etiology of Obesity. In B.Gumbiner (Ed.), *Obesity* Philadelphia: American College of Physicians.

Williamson, D.A., Martin, C.K., York-Crowe, E., Anton, S.D., Redman, L.M., Han, H. et al. (2007). Measurement of dietary restraint: validity tests of four questionnaires. *Appetite, 48,* 183-192.

World Health Organization (2009). BMI Classification. http://www.who.int/bmi/index.jsp?introPage=intro_3.html

Yancey, A.K., Kumanyika, S.K., Ponce, N.A., McCarthy, W.J., Fielding, J.E., Leslie, J.P. et al. (2004). Population-based interventions engaging communities of color in healthy eating and active living: a review. *Preventing chronic disease, 1,* A09.

Zenk, S.N., Schulz, A.J., Israel, B.A., James, S.A., Bao, S. & Wilson, M.L. (2005). Neighborhood racial composition, neighborhood poverty, and the spatial accessibility of supermarkets in metropolitan Detroit. *American Journal of Public Health, 95,* 660-667.

Zenk, S.N., Schulz, A.J., Israel, B.A., James, S.A., Bao, S. & Wilson, M.L. (2006). Fruit and vegetable access differs by community racial composition and socioeconomic position in Detroit, Michigan. *Ethnicity and Disease, 16,* 275-280.

Zhang, Q. & Wang, Y. (2004a). Socioeconomic inequality of obesity in the United States: Do gender, age, and ethnicity matter? *Social Science & Medicine, 58,* 1171-1180.

Zhang, Q. & Wang, Y. (2004b). Trends in the association between obesity and socioeconomic status in U.S. adults: 1971 to 2000. *Obesity Research, 12,* 1622-1632.

Zuckerman, M., Kuhlman, D.M., Joireman, J., Teta, P. & Kraft, M. (1993). A comparison of three structural models for personality: The big three, the big five, and the alternate five. *Journal of Personality and Social Psychology, 65,* 757-768.