THE IMPACT OF DEPRESSIVE SYMPTOMATOLOGY ON THE EFFICACY OF A WEIGHT LOSS INTERVENTION PROGRAM IN AFRICAN-AMERICAN CHILDREN AND ADOLESCENTS

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DEDICATION

For my parents, whose sacrifices and unconditional support have allowed me to pursue my dreams.

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by

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DISSERTATION

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Obesity is a widespread issue that has gradually increased in prevalence over the years. Individuals within certain racial or ethnic groups, minority children and adolescents, or youth living within low-income neighborhoods, are at a particularly high risk for the disease. Current research denotes a relationship between obesity and psychopathology, even though the directionality and the factors contributing to this interplay remain debatable. However, in order to develop viable solutions for solving this epidemic, additional factors impacting weight outcomes need to be examined. This study conducted secondary analyses using previously

collected data from 206 African-American youth ages 7-18 involved in a weight loss program within a major urban area. It explored the impact of various psychosocial elements, psychological factors, and treatment adherence issues, as potential predictors of weight outcomes. It was aimed to supplement the existing literature by attempting to identify the impact of various factors for African-American youth involved in a weight management program, which could potentially assist in the future development of more specific intervention strategies. Although most of the variables within the primary aims were not found to be predictive of weight loss success, certain sex differences between subjects were observed. Generally, males had overall higher hazard ratios in comparison to female subjects, and were thus more likely to drop out at any time during the study. Additional analyses suggested that older subjects and those with greater average food monitoring lost more weight. A specified target weight loss of 5% or 10% was also examined, and although no factors were predictive of this achievement, approximately 8.7 and 10.6 months were necessary to reach the 5% or 10 % weight loss, with female subjects being more successful in this task. These findings suggest that gender and engagement in food monitoring practices may be important factors for weight loss success for this unique population. In addition, early success in losing weight could also serve an important function, as this might have increased self-efficacy and motivation. However, further research is necessary in this area and the exploration of more culturally relevant interventions could prove to be increasingly beneficial.

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LIST OF ABBREVIATIONS

17OH	Progesterone Test
AAP	American Academy of Pediatrics
ACSM	American College of Sports Medicine
AMA	American Medical Association
AND	Academy of Nutrition and Dietetics
APA	American Psychiatric Association
AR1	First Order Autoregression
BED	Binge Eating Disorder
BMI; z-BMI	Body Mass Index; z-score
BS	Binge Scale
CBCL; CBCL-RPL	Child Behavior Checklist; Revised Problems List
CBQ; CBQ-SF	Conflict Behavior Questionnaire; Short Form
CBT	Cognitive Behavior Therapy
CDC	Centers for Disease Control and Prevention
CDI	Children's Depression Inventory
CES	Current Exercise Survey
CHD	Chronic Heart Disease
CI	Confidence Interval
CIDI	Composite International Diagnostic Interviews

DALY	Disability Adjusted Life Years
DHEAS	Dehydroepiandrosterone
DSM-V	Diagnostic and Statistical Manual of Mental Disorders 5th ed.
DXA	Dual Energy X-Ray Absorptiometry
EBI	Eating Behavior Inventory
ECT	Electroconvulsive Therapy
ESES	Eating Self-Efficacy Scale
FDA	Food and Drug Administration
GBD	Global Burden of Disease
HDL; LDL	High Density Lipoprotein Cholesterol; Low Density
HGB A1C	Hemoglobin Test
HPA	Hypothalamic-Pituitary-Adrenal Axis
HR	Hazard Ratio
HSFFQ	Harvard Service Food Frequency Questionnaire
IPT	Interpersonal Therapy
IRB	Institutional Review Board
IRS	Inflammatory Response System
KBIT	Kaufman Brief Intelligence Test
LCD	Low Calorie Diets
LMS	Lambda, Mu, Sigma Procedure

MAOI	Monoamine Oxidase Inhibitor
MDD	Major Depressive Disorder
MDE	Major Depressive Episode
MRI	Magnetic Resonance Imaging
NCS; NCS-R	National Comorbidity Surveys
NHNES; NHANES	National Health and Nutrition Examination Survey
NIH	National Institutes of Health
NLSY79	National Longitudinal Survey of Youth 1979
NPHS	Canadian National Population Health Survey
NWCR	National Weight Control Registry
PedsQL	Pediatric Quality of Life Inventory
PHEN/TPM	Phentermine and Topiramate
RCT	Randomized Controlled Trial
SES	Socioeconomic Status
SNRI	Selective Norepinephrine Reuptake Inhibitor
SPPC; SPPA	Self-Perception Profile for Children; Adolescents
SPSS	Statistical Package for Social Sciences
SSD	Subsyndromal Symptoms of Depression
SSRI	Selective Serotonin Reuptake Inhibitor
TCA	Tricyclic

TSH	Thyroid Stimulating Hormone
USDA	U.S. Department of Agriculture
USDHHS	U.S. Department of Health and Human Services
VLCD	Very Low Calorie Diets
WCHS	Weight Control Habits Survey
WHO	World Health Organization
YLD	Years Lived with Disability
YLL	Years Lost due to Premature Mortality
YSR; YSR-RPL	Youth Self-Report; Revised Problems List

CHAPTER ONE
Introduction

BACKGROUND

Magnitude of Obesity and Depression

Currently reported rates indicate that approximately 11% of adults, or nearly 500 million individuals (Finnucane et al., 2011) are affected by obesity worldwide (WHO, 2013). Overall, these rates have also gradually increased over the years (Kelly, Yang, Reynolds & He, 2008; Malik, Willett & Hu, 2013). Within the Unites States, an estimated 35% of adults (Ogden, Carroll, Kit & Flegal, 2012) and 17% of American children and adolescents suffer from obesity (CDC, 2013). In addition, it appears that certain populations are at a particularly high risk for obesity, especially within minority and low-income groups (CDC, 2013). Due to these alarmingly high levels of obesity and observed increases in prevalence over the years, the American Medical Association (AMA) has taken action to address this issue. Earlier this year it recognized obesity as a disease and recommended that various interventions be implemented for treatment and prevention of obesity (Pollack, 2013).

Depression is another concerning disorder, as it is now classified as the 3rd highest cause of global burden (Collins, Insel, Chockalingham, Daar & Maddox, 2013). While reported prevalence rates vary, a summary of the available data indicates that approximately 3% to 6% of adults could potentially be classified as depressed worldwide (Bromet et al., 2011; Funk et al., 2010). Nationally, adult rates are reported to be close to 7% over a 12-month period, with adult

females being 1.5 to 3 times more likely than men to suffer from depression (American Psychiatric Association, 2013). For children, it seems that prior to the onset of puberty, rates are estimated to be between 0.9% and 2% (McDonnell & Glod, 2003; Wichstrom et al., 2012; Egger & Angold, 2006). However, upon reaching adolescence, the prevalence rates of depression escalate to a range between 3.3% and 5.8% (Garrison et al,. 1997; Lewinsohn, Hops, Roberts, Seely & Andrews, 1993).

Both obesity and depression seem to pose significant difficulties for individuals suffering from these diseases. Obese individuals are reported to develop various health complications in their lifetime, including heart disease, type-2 diabetes, breathing difficulties, sleep problems and even certain cancers (Malnick & Knobler, 2006), which can lead to added health care costs (Tsai et al., 2011). Depressed adults may be more prone to comorbidities such as anxiety and eating disorders (Toups et al., 2013), while youth with these diseases could also be subject to disruptive behavior disorders and substance use problems (Garber, 2006; Maughan, Collishaw & Stringaris, 2012). In addition, increased mortality rates (Luppa, Heirich, Angermeyer, Konig & Riedel-Heller, 2007) and treatment related expenses (Greenberg et al., 2003) further contribute to a high economic burden. Given the extent of these problems, it may be necessary to simultaneously tackle both depression and obesity not only at a national, but a global level.

Relationship of Obesity and Depression

While depression and obesity appear to be connected, the directionality (bidirectional or unidirectional) of their relationship remains a topic of debate. Evidence from Luppino et al. (2010) and Blaine (2008) presents evidence for a mutual interplay, while other studies have concluded that this relationship is unidirectional, suggesting that depressed individuals may be at a higher risk for the development of obesity (Roberts & Duong, 2013; Eremis et al., 2004; Pine et al., 2001). Certain pathways for the development of this link have been assessed, and factors such as the hypothalamic-pituitary-adrenal (HPA) axis, the inflammatory response system (IRS) and serotonin delivery, have all been theorized to play a significant part in mediating the obesity-depression relationship (Bornstein, Schuppenies, Wong & Licinio, 2006; Reeves, Postolache & Snitker, 2008; Bastard et al., 2006). Various psychosocial factors have also been shown to play an important role as both mediating and moderating variables. Faith, Matz and Jorge (2002), as well as Stunkard, Faith and Allison (2003) described several demographic characteristics (e.g. gender, socioeconomic status, gene-environment interactions), psychiatric issues (negative childhood experiences or trauma, eating disorders) and lifestyle influences (diet, physical activity, stress) that could potentially affect this connection.

PROBLEM STATEMENT

Although research has acknowledged a significant relationship between depression and obesity, there is a scarcity of data on effective intervention methods for individuals concurrently diagnosed with both diseases. While certain contributing pathways have been identified, the directionality and implications of this relationship are not fully understood at this time.

Significantly less information is available for the treatment of children and adolescents, particularly minority and low-income youth that present with these comorbidities. The factors involved in the delivery of weight management and depression treatments in a culturally relevant fashion are also not well known within ethnically diverse youth and their families.

STUDY PURPOSE AND IMPORTANCE

The current study attempted to further examine the depression-obesity relationship within a weight management intervention program for African-American children and adolescents. It assessed the impact of known contributors such as psychosocial variables, depressive symptomatology and various adherence factors in relation to weight. It also examined the presence of individual depressive symptoms using the Children's Depression Inventory (CDI) subscales, in an effort to determine if certain aspects of depression are more likely to predict weight loss. The current study sought to provide a better understanding of the interventions that may be necessary for the simultaneous treatment of depression and obesity within minority and low-income youth. It was anticipated that it would add to a much-needed area of research, by providing clinically useful information on obese children and adolescent who may suffer from subsyndromal depressive symptoms, for whom treatment with psychotherapy and psychotropic medications may not be available.

CHAPTER TWO
Review of the Literature

OBESITY

Defining the Problem

Obesity, or above normal weight generally results from an imbalance between caloric consumption and energy expenditure (Macfarlane & Thomas, 2009), leading to an accumulation of excess adipose tissue. This inequity is then evaluated through the measurement of an individual's Body Mass Index (BMI), which estimates body fat percentage. When measuring BMI in adults, an individual's body weight is assessed in kilograms, and is divided by their height in meters squared. The resulting numbers from this formula represent various categories of body fatness that range from underweight (BMI below 18.5 kg/m^2), to healthy (BMI of 18.5 kg/m^2 to 24.9 kg/m^2), overweight (BMI of 25 kg/m^2 to 29.9 kg/m^2) and obese (BMI of 30 kg/m^2 or higher).

Although some debate exists in using BMI as an estimate for healthy weight, as it is not a direct measure of body fatness (only a correlate), it is the most commonly accepted method of estimating an individual's body fat percentage and health status, even in healthcare settings. Current research indicates that correlations among BMI and body fat percentage vary from .72 to .79 for males, while for females they range from approximately .72 to .84 (Flegal et al., 2009). Recent evidence from Stenholm et al., (2008), DiMonaco et al., (2011), Ruiz et al., (2008), as

well as Shah and Braverman, (2012), suggests that current BMI measurements might actually underestimate obesity levels due to the formula's failure to explain additional factors and gender differences when examining lean muscle mass, or loss of muscle mass over time. A thorough evaluation of BMI accuracy conducted by Shah and Braverman (2012) drew a comparison between BMI and dual energy x-ray absorptiometry (DXA) tests, which can concurrently measure bone, muscle mass and adipose tissue. Despite the limitations noted, the authors acknowledged that BMI measurements remain a low-cost and useful method of estimating an individual's body fatness. However, they felt that due to the underestimations discovered, lower cutoffs of 24 kg/m^2 and 28 kg/m^2 for females and males respectively, are warranted (Shah & Braverman 2012). Other more costly and time-consuming practices for measuring body fat percentage involve procedures such as caliper skinfold measurements, underwater weighing and even magnetic resonance imaging (MRI) (CDC, 2013). While these additional measures are more accurate, it is unlikely that they will replace the current BMI formula for estimating body fatness. From a practical perspective, BMI remains the most useful and accepted screening tool at this time.

While most people are aware that with increased food intake and a sedentary lifestyle, or decreased physical activity, individuals become more prone to the storage of excess weight, other risk factors might not be as well known. Some studies have suggested that evidence for increasing obesity actually extends beyond a model that simply addresses the relationships between food and exercise habits. A recent article by Chaput et al. (2013) indicates that a

combination of alternative risk factors such as decreased sleep, disinhibited eating patterns, and low calcium intake actually had a greater contribution to the risks of becoming overweight or obese, when compared to the more traditional "Big Two" model of food intake and exercise. Another extensive study examining this link also proposed factors related to various genetic components, inadequate sleep, modified endocrine functioning due to chemicals and toxins, increased use of pharmaceuticals, increased use of climate control, birth weight and maternal nutrition as notable contributors (McAllister et al., 2009). While it would be impossible to come up with an exhaustive list of all risk factors involved, perhaps the most important information to derive from this evidence is that the pathways for obesity are not very clear, and they involve multiple relationships that extend beyond the commonly assumed food and exercise connections.

A Global Concern

Obesity is a major health concern that has continued to significantly increase in prevalence worldwide since the 1980's. Although it is easily measured and assessed, obesity has become increasingly difficult to manage and prevent. The World Health Organization (WHO) currently classifies obesity as a chronic disease indicator that affects approximately 11% of the world's adult population age 20 and over (WHO Mental Health, 2013). A recent worldwide examination of obesity prevalence indicated that rates of obesity in 2008 were approximately 10% in men and 14% in women, which are close to two times greater than the observed numbers in 1980 (of approximately 5% and 8% in men and women respectively) (Finucane et al., 2011). The authors also reported that approximately 500 million individuals were suffering from obesity globally in

2008 (Finnucane et al., 2011). These numbers are estimated to greatly increase within the next 20 years (by 2030), particularly for low and middle-income countries (Kelly, Yang, Reynolds & He, 2008; Malik, Willett & Hu, 2013).

In continuing to predict future trends for obesity, Malik et al., (2013) suggest that it might also be important to take into account factors related to increased globalization, more abundant or more readily available food sources, and continued population increases. These variables were hypothesized to eventually lead to a greater number of individuals becoming obese, even if actual prevalence rates remained stagnant (Malik, Willett & Hu 2013). Similar global trends and increases were reported by Stevens et al. (2012) in a study that examined adult overweight and obesity trends from 1980 to 2008 in 199 countries and regions worldwide. While their analyses also indicated that the global prevalence of obesity resulted in approximately 508 million obese individuals in 2008, they noted that an additional 1.5 billion adults were also reported to be overweight at the time. However, their focus was also aimed at better understanding the effects of obesity on individual countries and regions around the world. They used estimated mean BMI measures to evaluate the prevalence and possible causes of the disease. As a result, the authors discovered various disparities between countries/regions and even between males and females. This evidence further suggests that in some areas of the world obesity could also be related to other factors such as culture and history, geographic location and even gender (Stevens et al. 2013), adding to the list of the reported contributors noted by Malik et al. (2013).

A National Burden

Within the Unites States, the Centers for Disease Control and Prevention (CDC) reports that approximately 35% percent of adults (age 20 and over) are currently obese (according to the 2010 census data) (CDC, 2013), which translates into about 78 million individuals (based on 2009-10 NHNES data) (Ogden, Carroll, Kit & Flegal, 2012). In fact, overall, the United States seems to have one of the highest mean BMI levels among the more prominent nations (Finucane et al., 2011). Additional results from the National Health and Nutrition Examination Survey (NHNES) from 2009-10 indicate that those over the age of 60 have an even higher chance of being obese, and that men and women now appear to have nearly identical rates of obesity. This is a change from earlier estimates in 1999, when men had lower obesity rates than women (Ogden, Carroll, Kit & Flegal, 2012).

Although recent trends might suggest that obesity percentages have not been significantly increasing since 2003 (Flagal, Carroll, Kit & Ogden, 2012), no decreases have actually been noted at this time, making predictions on future trajectories difficult to ascertain. However, by further examining the cohort of obese individuals within the United States, various differences between morbidly obese, or the super morbidly obese may become evident. Current research indicates that rates of extreme obesity have actually undergone much greater increases compared to the overweight, or just obese categories (Sturm, 2007; Sturm & Hattori, 2013). As reported by Flegal, Carroll, Kit and Ogden (2012), data based on the 2009-2010 CDC statistics suggest that national prevalence levels reached approximately 6.3% for these upper obesity ranges (BMI over

40 kg/m^2), in which individuals are generally 100 pounds or more overweight. This information is also consistent with prior estimates by Flegal et al. (2010) based on 2007 to 2008 NHNES data, in which approximately 5.7% of individuals were identified to have had a BMI of 40 kg/m^2 of above. More recent estimates report that as many as 15.5 million, or 6.6% of our nation's population were in the morbidly obese range, or higher, in 2010 (Sturm & Hattori, 2013).

It is also assumed that with higher increases in weight, the chance of additional health problems will also proportionally increase, and the resulting complications could be even more severe. This may be especially relevant for individuals in the upper obesity ranges, as they are more likely to necessitate additional resources or accommodations within the health care setting, due to existing limitations of standard medical equipment (Sturm & Hattori, 2013). As a result, certain measures are now in place to address issues that arise when working with patients suffering from extreme obesity. While various hospital and clinics are attempting to tackle issues related to weight limits, The National Institute for Occupational Safety and Health has developed a plan to reduce work related injuries when caring for these patients (Galinsky, Hudock & Streit, 2010). Although all of these additional precautions and safety standards are necessary, they also seem to result in additional costs not only for patients, but also for the healthcare system as a whole.

Between the years of 1987 to 2001, medical costs for obese individuals accounted for 27% of the total healthcare cost increases (Thorpe, Florence, Howard & Joski, 2004). This staggering

number reflects the direction of healthcare spending, as obese individuals will require continued medical interventions and follow-up. Perhaps in the midst of acknowledging the magnitude of this financial problem, it is also necessary to assess the specific factors contributing to the high levels of obesity within the United States. Adults with a BMI above 30 kg/m^2 are not only considered obese, but they also develop a greater number of health risks and diseases over their lifetime in comparison to those individuals with lower BMI measurements. They are more likely to develop complications such as heart disease, type-2 diabetes, breathing difficulties, cancers, and sleep problems (Malnick & Knobler, 2006). This increase in health issues can simultaneously translate into an augmented economic burden, particularly due to the added health care costs associated with obese individuals. In a recent review of studies that outlined the costs of US overweight and obese individuals, Tsai et al. (2011) found significantly higher expenses associated with persons at or above a BMI of 30 kg/m^2. The authors noted an approximate cost increase of 10% for overweight individuals and a staggering 43% cost increase for obese individuals when compared to those of normal weight (Tsai, Williamson, Glick, 2011). The consequences become even more severe for those with BMI classifications above 40 kg/m^2, or above 50 kg/m^2, as these individuals represent two additional subgroups of obesity known as the morbidly obese and super morbidly obese.

Children and Adolescents

In children, the obesity epidemic represents an even more immediate concern due to the early onset of health problems and their continuation into adulthood. The CDC reports that

approximately 17% of children and adolescents (2-19 years old) in the United States are currently obese (CDC, 2013). In children, weight standards are classified according to percentiles, which are adjusted for age and sex, while also accounting for the child's continued growth. The percentile rankings therefore allow us to understand where a particular child stands in relation to other children of the same age and sex, and to assess if they meet projected developmental expectations. Weight status categories for children and adolescents are classified as underweight (under the 5th percentile), healthy (from the 5th to < the 85th percentile) overweight (from the 85th to < the 95th percentile), and obese (95th or > percentile) (CDC, 2013). Children classified as having a BMI above the 85th and 95th percentiles have higher risks of comorbidities, including type-2 diabetes, high blood pressure, high cholesterol, breathing difficulties and joint problems (CDC, 2013; Dietz, 1998; Freedman, Mei, Srinivasan, Breneson & Deitz, 2007). Freedman et al., (2007) noted that children and adolescents in the 99th percentile are estimated to have about a 33% chance of having three or more identifiable risk factors. Research also indicates that a large number of overweight children eventually become obese and are more susceptible to Chronic Heart Disease (CHD) and other risks that can extend into adulthood (Biro & Wein, 2010).

Within minority populations, the observed rates of childhood obesity are even more alarming (Hedley et al., 2004). Current studies reveal that African-American and Mexican American children and adolescents have higher rates of obesity when compared to Caucasian children. The latest National Health and Nutrition Examination Survey (NHNES) data from 2007-2008 shows

that that 27% of Mexican-American adolescent males, 20% of African-American adolescent males and 17% of Caucasian adolescent males are currently obese (CDC, 2013). Simultaneously, the same survey denotes that 29% of African-American adolescent girls, 17% of Mexican American girls and close to 15% of Caucasian girls are obese (CDC, 2013). When observing youth within low-income households recent evidence suggests that both minority and low-income children are exposed to more screen time, which perpetuates a sedentary lifestyle and subjects children to a greater amount of food advertising. A lack of access to healthy food choices, an overabundance of fast food restaurants, and lack of exercise facilities, including properly maintained parks, are hypothesized to pose additional barriers for youth within low-income neighborhoods (Kumanyika & Grier, 2006).

When examining obesity risk factors for children and adolescents, additional contributors that are normally associated with adult obesity have also been identified. Sleep is a contributor that seems to be of great importance for future research, due to its potential relationships with obesity in younger populations. A recent study in Australian children evaluated the longitudinal relationship between BMI and sleep length, which included specific analyses for obesity (Magee, Caputi & Iverson, 2013). Although the conclusions drawn were somewhat limited, there nonetheless appeared to be an inverse relationship between decreased sleep and BMI, specifically for children with early onset obesity (Magee, Caputi & Iverson, 2013). This may be a result of physiological changes (hormones such as leptin and ghrelin), decreased physical activity due to fatigue, and increased chances for food consumption due to being awake longer

(Knutson, Spiegel, Penev & Van Cauter, 2007; Taheri, 2006; Spiegel et al., 2004). While some of the additional literature in this area supports the hypothesis of higher obesity risks for children that are sleep deprived (Chen, Beydoun & Wang, 2008), it also points out that the link may not be as obvious as in adult populations and there is a continued need for future exploration in this area (Lytle, Pasch & Farbakhsh, 2011).

DEPRESSION

Major Depressive Disorder

Definition

Defined by a specific set of qualifying criteria, major depressive disorder (MDD) is generally accompanied either by significant distress, and/or an impairment of daily functioning. According to the Diagnostic and Statistical Manual of Mental Disorders 5th edition (American Psychiatric Association, 2013), clinical significance is reached when a cluster of symptoms that last two or more weeks are present on a daily basis. A hallmark presentation of MDD typically consists of depressed mood, or a loss of interest in previously pleasurable activities for at least a two-week duration (anhedonia). This presentation is also associated with additional symptoms such as feelings of guilt/worthlessness, decreased energy, difficulties concentrating, insomnia/hypersomnia, increased or decreased appetite (or weight loss/gain), psychomotor agitation/retardation and suicidal ideation.

Although MDD onset can occur at any age, the risks seem to be significantly higher post-puberty. Risks are also higher for individuals with general negative affect (neuroticism), significant life stressors, comorbid mental or medical illnesses, and a family history of depression (up to 40% heritability) (American Psychiatric Association, 2013). These factors suggest that both genetic predisposition and environmental influences are significant contributors in the development of depression. Studies that further examined the role of environment and heritability noted a genetic influence ranging anywhere from 31% to 42% (Nes et al., 2013; Sullivan, Neale & Kendler, 2000). In general, the prevalence of MDD is also increased in females, despite males having similar presentation, symptoms, and treatment response rates (DSM-V, 2013). The remaining variance seems to be attributed to a variety of other influences, of which individual environment could be the most notable.

Having had a previous major depressive episode (MDE) seems to further serve as an important factor and indicator for future depression. A recent Canadian longitudinal study examining a community sample of over 5,000 individuals from the Canadian National Population Health Survey (NPHS) over a 16-year period reported a 9% risk increase of having one or more recurrent MDEs after the initial episode (Bulloch, Williams, Lavorato, & Patten, 2013). Other studies that analyzed clinical samples reported even higher rates of MDE recurrence. A 15-year prospective follow-up study of 380 individuals revealed a recurrence of up to 85% for those that initially recovered from their first MDE (Mueller et al., 1999). Another study examining a group of approximately 7900 hospitalized patients in Denmark, noted an average recurrence increase of

about 15% with each subsequent episode (Kessing & Andersen, 1999). This evidence indicates that the rates of recurrence seem to increase with time and with each episode, even when remission is achieved. Individuals with depression appear to be at greater risk of developing additional difficulties and perhaps suffering from ongoing prodromal, or residual symptoms that could eventually manifest into another MDE. Findings by Fava et al., (1990), although somewhat limited due to sample size, suggest that similar prodromal symptoms observed during the initial MDE can also be detected prior to the onset of another episode. Concordantly, Judd et al., (1998) summarize an array of data that describes the relationship between MDE relapse and subsyndromal symptoms of depression (SSD). Their own inquiry into this topic confirms that those individuals failing to achieve full remission of symptoms (despite achieving remission of the MDE) relapsed much more quickly than the patients achieving full recovery. Research on sex specific differences has also shown a greater lifetime prevalence of depression in females, with an approximate 2:1 female to male ratio for MDD (Bebbington et al., 1998; Bertakis et al., 2001; Grigoriadis & Robinson, 2007; Hasin, Goodwin, Stinton & Grant, 2005).

Adult Prevalence and Burden

A review of the Global Burden of Disease (GBD) data from 1990 to 2000, indicates that depressive disorders are now 3rd highest cause of burden and primary result of disability worldwide. These statistics bring to attention the critical nature of depression and the need to alleviate the problems that have occurred in our societies by providing more thorough and integrative treatment approaches (Collins, Insel, Chockalingham, Daar & Maddox, 2013).

Although some debate exists on the actual prevalence and incidence rates of depressive disorders due to issues of heterogeneity and variability in the data collected (Brhlikova, Pollock & Manners, 2011; Ferrari et al., 2013; Weich & Araya, 2004), the 2000 GDB figures noted a 12-month MDE prevalence of approximately 1.6% in males and 2.6% in females (Ustun, Ayuso-Mateos, Chatterji, Mathers, & Murray, 2004).

A review of available European study data for MDD prevalence over a 12-month period, depicted rates of approximately 5%, although the information assembled pertained mainly to Western Europe (Paykel, Brugha & Fryers, 2005). However, this number appears to be more consistent with the recent data that was examined by Bromet et al. (2011), as they addressed the use of the WHO's Composite International Diagnostic Interviews (CIDI) for the assessment of MDEs. From their assessments, it appears that prevalence rates are now estimated to be between 5.5% and 5.9% in high and low to middle income nations, respectively. The WHO's 2010 report on mental health further described that approximately 150 million individuals around the world could be currently depressed. In addition, within low-income countries the issues seem to be even more severe, as a noted 3.2% of the 4% total disease burden seems to be resulting from the effects of depression (Funk et al., 2010).

Within the United States, the 12-month prevalence of MDD has been estimated to be approximately 7%, with higher rates for young adults (ages 18-29) and females (1.5 to 3 times higher) (American Psychiatric Association, 2013). Clinical studies note similar numbers,

indicating that approximately 6.6% (13 million) of individuals will actually meet MDD criteria over a one-year period, while up to 16.2% (32 million) could be diagnosed with MDD over their lifetime (Kessler et al., 2003). Prospective data based on the National Comorbidity Surveys (NCS and NCS-R) described prevalence rates from 16.9% to 19% for those suffering from at least one depressive episode between the ages of 18 to 32 (Moffitt et al., 2010).The resulting economic burden due to the incurred expenses aimed at the prevention and treatment of depressive disorders appears to also have significant consequences. A systematic review of literature on this topic by Luppa et al. (2007) described various increases in costs associated with those individuals that were diagnosed with MDD and sought treatment. For a one-year interval, excess costs ranged from $1000 to $2500, without accounting for additional morbidity and mortality expenses (Luppa, Heirich, Angermeyer, Konig & Riedel-Heller, 2007). In the year 2000, approximately $83 billion dollars were associated with the costs of depression in the United States (Greenberg et al., 2003).

Childhood Classification, Prevalence and Burden

While depression is classified in a similar fashion in children and adolescents as it is in adults, some variabilities in symptoms exist. Current diagnostic data indicates that children and adolescents are more likely to experience irritable mood instead of depressed mood and could also experience a failure to meet expected weight requirements (developmental milestones) due to changes in appetite (American Psychiatric Association, 2013). Potential risk factors, treatments and the disease course of depression could also vary as a result of the growth-related

processes that occur during this critical time. Perhaps one important principle to uphold when further examining this population, is that children are not simply "little adults." Therefore, overall management of the disease in this age group cannot be based solely on the information and evidence collected from adult studies.

Although the current literature acknowledges the limited data available on the epidemiology of major depressive disorder in children and adolescents, efforts to better understand the gravity and prevalence of this illness, including its prevention, are currently underway (Sims, Nottlemann, Koretz & Pearson, 2006). Recent research denotes that childhood depression rates prior to the onset of puberty can range from 0.9% (McDonnell & Glod, 2003) to 2% (Wichstrom et al., 2012; Egger & Angold, 2006). However, as adolescence is reached the prevalence increases, resulting in rates of approximately 4% (Thapar, Collishaw, Pine & Thapar, 2012) to 5.7% (Jane-Costello, Erkanli & Angold, 2006), over a one-year period. In addition, being of female gender poses additional risks for adolescents, as depression rates for young girls are two to three times higher than for boys of similar age, an observation consistent with adult estimates (Hankin et al., 1998; Essau, Lewinsohn, Seeley & Sasagawa, 2010). Within the United States, a longitudinal study conducted by Costello et al., (2003) indicated that up to 36.7% of subjects 9 to 13 years old experienced one or more psychiatric disorders by the age of 16. Of these study participants, approximately 9.5% specifically met the DSM criteria for a depressive disorder (Castello, Mustillo, Erkanli, Keeler & Angold, 2003). Previous US based studies also reported approximate

incidence rates at 12-month intervals to be between 3.3% and 5.8% for adolescents (Garrison et al., 1997; Lewinsohn, Hops, Roberts, Seely & Andrews, 1993).

In a quest to explore global data, a recent GDB analysis initiated by the WHO (based on 2010 information) examined degree of burden for a several nations (Gore et al., 2011). Generally, when exploring GDB, health summaries are provided in terms of disability adjusted life years or DALYs, which are comprised of years lost due to premature mortality (YLL) and years lived with disability (YLD). The sum of these two factors is thought to reflect a population's health in relation to existing norms. According to Murray et al. (2012), DALYs represent "an absolute measure of health loss…, [reflecting] the number of individuals who are ill or die in each age-sex group and location" (pg. 2199). The study from Gore et al. (2011) suggested that for individuals between 10 to 24 years old, neuropsychiatric disorders resulted in a high degree of burden for nations with all levels of income. However, the greatest burden was actually noted in the highest income countries. The data also shows that when measuring DALYs, children, adolescents, and young adults (10 to 24 years old) suffering from unipolar depression accounted for 8.2% of total DALYs. When further broken down into specific age groups, children and adolescents between 10 to 14 years old and 15 to 19 years old had DALY rates of 5.7% and 9.9% respectively (Gore et al., 2011). This means that countries such as the United States are currently facing some of the highest burden levels in the world due to the significant amount of years lost as a result of illness and premature mortality within this 10 to 24 age group. Nonetheless, for children the economic burden extends beyond the individual, due to their involvement in various environments and

settings. Evaluated costs seem to directly influence the family, overall healthcare, as well as the education system (Lynch, Gregory & Clarke, 2006). In addition, children and adolescents with depressive disorders seem to also have significantly greater amounts of psychiatric comorbidities (Garber, 2006; Maughan, Collishaw & Stringaris, 2012), which could result in further complications and increased treatment costs.

Depression and Obesity Link

As noted above, both obesity and depression seem to independently contribute to a significant amount of public concern and economic burden worldwide. Depression has not only been linked to additional psychiatric comorbidities, but it is also theorized to play an important role in regards to weight. An early study on childhood and adolescent obesity by Hammar et al. (1972), which although limited by a small sample size, began to point out that obese subjects had higher depression scores in comparison to their normal weight peers. More recent evidence by Vila et al. (2004), based on clinical interviews, self report measures and parent report questionnaires indicated that within an outpatient hospital setting, up to 58% of obese children had a psychiatric illness. In addition, approximately 12% of their study sample met criteria for either depression, or dysthymia (Villa et al., 2004). Prospective evidence from a US based study of over 9,000 adolescents revealed that initial depression could eventually contribute to an onset of obesity in non-obese subjects over a one year period, as well as an increase in BMI (adjusted for age) in those that were previously obese (Goodman & Whitaker, 2002). Additional research further supports this link, as findings from Erermis et al. (2004), Petry et al. (2008) and Blaine (2008) all

point to increased odds ratios of obesity for depressed adolescents and adults. Although the exact relationship between obesity and depression remains a topic of much debate, recent evidence does generally acknowledge some level of interplay between the two. However, while some studies such as the meta-analyses conducted by Luppino et al. (2010) and Blaine (2008) view this relationship as bidirectional, meaning that either obesity or depression can act as a risk factor for the other, prospective evidence from Roberts and Duong (2013) suggests that this interchange might not necessarily be mutual. In other words, Roberts and Duong (2013) found that only depression increased the risk for future problems with weight, particularly in the development of obesity, and not vice versa. In addition, they also indicated that baseline depressive symptomatology could be predictive of an individual eventually becoming overweight. Other research by Eremis et al. (2004) and Pine et al. (2001) reported similar findings, as they noted that depression could be more common in obese adolescents, and that depression in childhood could also be associated with increased adult BMI measurements.

Due to the notable split in research on this topic, making a distinction in the type of relationship that exists is difficult. Yet in the quest to provide increased clarity on this issue, the present study aligns with the unidirectional model of depression and obesity supported by, Eremis et al. (2004), Pine et al. (2001), as well as Roberts and Duong (2013), in an effort to replicate these findings. In assuming this directionality, additional questions that become relevant to this issue pertain to the contributors and perpetuators that could surround this directional relationship. Assessing the variables or pathways that could lead to depressed individuals becoming more

obese, could provide additional insight into more effective treatments and interventions for this population.

Currently, the factors assumed to play a role in the relationship between depression and obesity seem to be both neurophysiological and psychosocial in nature. Research suggests that pathways such as the hypothalamic-pituitary-adrenal (HPA) axis, the inflammatory response and serotonin delivery systems, are all highly important mediating this relationship. Bornstein, et al. (2006) and Reeves et al. (2008), discuss evidence for HPA axis dysregulation in both depression and obesity, including its implication in the body's inflammation response system (IRS) and appetite regulation. In a more in depth examination of the neurobiology involved between depression and stress response, Gold and Chrousos (2002) delineated various differences between typical and atypical features of depression. In their model, the authors suggested that patients with typical depression generally experience an increased stress response, which could lead to anxiety, loss of appetite, as well as decreased sleep. On the other hand, those with an atypical presentation would be quite the opposite, as they would exhibit fatigue, decreased activity, increased appetite, and increased sleep (Gold and Chrousos (2002).

Other changes in immune system response (IRS) were also observed, which seem to be consistent with various studies that reported associations between immune system dysregulation and an onset of depression (Reeves, Postolache & Snitker, 2008; Kim et al., 2007; Schiepers, Wichers & Maes, 2005). A recent meta-analysis by Dowlati et al. (2010) indicated that specific

cytokine concentrations (TNF and IL-6) were higher in depressed subjects, which was theorized to contribute to immune system dysregulation, and increased activation of the IRS. Schiepers et al. (2005) also noted that specific cytokines could not only lead to increased HPA axis activity, but a reduction in serotonin levels. Similarly, obesity is also considered to be impacted by the increased production of cytokines due to the resulting inflammation from fatty tissue, which in turn increases activation of the IRS, and could eventually lead to insulin resistance (Bastard et al., 2006; Reeves, Postolache & Snitker, 2008). In their review, Bose et al. (2009) further pointed to the possible cortisol and glucocorticoid effects on HPA axis activation, and resulting obesity.

Both depression and obesity seem to work on similar neurobiological pathways, in which the HPA axis appears to be the largest contributor. However, additional psychosocial factors are also shared and should be equally noted. Faith et al. (2002), and Stunkard et al. (2003) listed various moderators and mediators that could be involved in the interaction between obesity and depression. They indicated that characteristics such as gender, SES, gene-environment interactions, negative childhood experiences, binge eating disorder (BED) and severity of the depression and obesity can act as conditional or moderating variables. In addition, factors such as eating, physical activity, teasing, and stress, are likely to play the roles of mediating or linking variables. The isolation of these factors is particularly interesting when examining them individually. For example, from the above noted information it is evident that more women suffer from depression than men (2:1 ratio), and that the severity of depression and obesity has an impact on MDE recovery, symptoms, and health risks. It is also apparent that generally,

excess adipose tissue is a direct result of an imbalance between caloric intake and physical activity (usually a sedentary lifestyle). However, excess weight, particularly within the central area of the body has been linked to chronic stress levels, as well as to subsequently higher levels of cortisol secretion for both males and females (Epel et al., 1999; Epel et al., 2000). Additional evidence from a longitudinal study further suggests that overeating in itself could be predictive of obesity, and could also be associated with depressive symptoms within a sample of US adolescents and young adults (Sonneville et al., 2013). In another study examining the comorbidities of BED, it was discovered that up to 51% of subjects had a simultaneous diagnosis of MDD (Yanovski, Nelson, Dubber & Spitzer, 1993). It is currently known that not all patients with BED are overweight or obese, and that overall prevalence rates according to WHO survey reports are approximately 2% within the population (Kessler et al., 2013). However, it appears that a connection between BED and depression is plausible, which could in turn also theoretically affect an individual's weight. This can be particularly true if we examine individual symptoms of depression, such as appetite.

Based on the evidence noted, a viable theory may be that individuals with increased appetite (atypical depression) would have a higher caloric intake, and may also have decreased amounts of energy, which could result in an overall energy imbalance. This would then allow for an accumulation of excess adipose tissue. When exploring other factors, such as negative childhood experiences, it seems that sexual, or physical abuse and neglect, have also been associated with increased depression and obesity odds, particularly in women (Rohde et al., 2007). An additional

review of various studies assessing interpersonal violence during childhood revealed that approximately 81% of the research analyzed reported an association with obesity (Midei & Matthews, 2010). Perhaps abuse or violence in childhood leads to a general lack of trust and withdrawal for these children. Subsequently, this could result in the development of internalizing symptoms and other depressive features, which would act as maladaptive coping mechanisms. Ultimately, eating could be a way for these children and adolescents to deal with their emotional difficulties.

Lastly, other significant variables noted by Faith et al. (2002), and Stunkard et al. (2003) were related to the self-esteem, negative body image and teasing aspects of obese and depressed individuals. A Canadian longitudinal study examining these relationships, discovered that children who were obese at the onset of the study, were almost two times more likely to have low-self esteem at follow-up (4 years later), which the authors hypothesized also lead to a greater risk of mental health problems (Wang, Kipp, Kuhle & Veugelers, 2009). Strauss (2000) also found evidence for the relationship between obesity and self-esteem, particularly in white and Hispanic female adolescents. Additional research by Storch et al. (2007) further noted positive associations between peer victimizations and depressive symptoms, body image, as well as behavioral issues for overweight or overweight-risk youth. In a study of female adults with a diagnosis of BED, findings indicated that teasing due to appearance was also associated with body dissatisfaction, depression and self-esteem (Jackson, Grilo & Masheb, 2000). Based on this research, it could be theorized that some overweight or obese individuals might be less satisfied

with their appearance and therefore have a more negative view of self. This could be particularly apparent during adolescence, which is an important time for identity formation according to Erikson's psychosocial stages of development - identity vs. role confusion work (Erikson & Erikson, 1998). In addition, since social interactions are also highly valued during this time, individuals that are teased or bullied could internalize their negative emotions and possibly develop various depressive symptoms. While it seems that a link between depression and obesity is certainly plausible, the directionality of the relationship and factors involved remain to be fully understood. In order to further clarify these issues additional research is needed.

INTERVENTIONS

Weight Management

Brief Overview

Some of the more common and highly advertised weight loss options consist of medications (diet pills), commercial diets (such as Weight Watchers) and even bariatric surgery. However, other changes that include lifestyle and behavioral modifications may be necessary for continued success. Appropriate food choices and a maintained level of physical exercise are similarly important in offsetting the balance between energy intake (food) and energy expenditure. Perhaps an appropriate starting point when working with individuals that are obese or overweight is to determine the types of interventions and treatment delivery methods that should

be utilized. Currently, the consensus appears to be in favor of using a combination treatment approach that is composed of dietary and exercise modifications, as well as behavioral therapy practices, prior to the use of pharmacotherapy and bariatric surgery options (NIH, 1998). Research based on the data collected from the NHANES during 2001-2006, has shown that strategies such as decreasing fat intake, increasing exercise levels, and taking prescription weight loss medications, have all resulted in a 10% or greater weight loss for adults attempting to lose weight over a 12-month period (Nicklas, Huskey, Davis & Wee, 2012). This is particularly relevant, since the available data indicates that even a 5% to 10% loss in current body weight, can significantly reduce various health risks (e.g. cardiovascular function) that are associated with being obese (NIH, 1998; Bigornia et al., 2013; Fayh, Lopes, da Silva, Reischak-Oliviera & Friedman, 2013).

In regards to delivery, while internet and telephone phone based interventions have shown some promise in weight loss treatment (Sherwood et al., 2011; Krukowski, Harvey-Berino, Bursac, Ashikaga & West, 2013), their success may be due to the implementation of various self-monitoring components and their predominant focus on weight loss maintenance. A recent review of various web-based weight loss trials indicated that only those internet maintenance programs that were preceded by a face-to-face component, achieved significant weight loss (Arem & Irwin, 2011). As a result, it currently appears that traditional in-person interventions remain the optimal treatment delivery modalities for weight management programs. Once the

content and delivery methods are established, providers can begin to work on assessing and reducing participants' current health risks.

Lastly, it may also be useful for practitioners to examine the ongoing progress of the individuals involved, their motivation, and their weight outcomes over the course of the provided treatments. This may be helpful in determining the aggressiveness of the interventions and in establishing goals and expectations for various programs. Some of the current interventions in adult populations show that a faster rate of initial weight loss could be more effective than a slow and progressive weight loss pace (Nackers, Ross & Perri, 2010; Elfhag & Rossner, 2005; Jeffery, Wing & Meyer, 1998; Astrup & Rossner, 2000). The old notion indicating that faster loss results in faster regain may no longer be applicable at this time, especially if individuals achieving rapid weight losses are provided with adequate support and additional interventions aimed at maintaining the weight lost (Delbridge & Proietto, 2006).

Dietary Modifications

In regards to caloric intake, research indicates that a moderate daily dietary restriction of 500 of more calories (kcals) can be effective in achieving significant weight loss (Carels et al., 2008; Karam & McFarlane, 2010). However, the amount of caloric restrictions recommended should be a direct result of an individual's level of excess weight. For example, an overweight individual with a BMI of 25 kg/m^2 should not be expected to have the same caloric reductions as an obese individual with a BMI of 50 kg/m^2. The 1998 NIH evidence report for the treatment of

overweight and obesity noted that in order to achieve the target goal of a 10% weight reduction, an individual's caloric restriction could vary from 500 kcals up to 1000 kcals per day, depending on the severity of their obesity (NIH, 1998). This means that daily diets with moderate restrictions (low calorie diets, or LCDs) will usually consist of 1,000 to 1,500 total kcals per day (Karam & McFarlane, 2010).

Other dietary options are also available, including the implementation of very low calorie diets (VLCDs, which are between 400 to 800 total kcals per day), low carbohydrate/high protein diets, and low fat diets. However, a review of the literature comparing the efficacy of VLCD and LCD by Saris (2001) revealed that long-term weight loss is nearly identical between the two dietary modalities. In addition, the risks posed by VLCD diets, particularly in the absence of regular medical supervision, may also outweigh their benefits for various individuals (Tsai & Wadden, 2006). Studies specifically examining these risks have discovered potential increases in the development of gallstones and cholelithiasis with diets that were aimed at achieving weight losses of over 1.5kg per week (VLCDs) (Weinsier, Wilson & Lee, 1995). Research has also suggested that for individuals with severe systemic/organ disease, those that are pregnant/lactating, or have been diagnosed with type I diabetes, an eating disorder, or a major psychiatric illness, participation in VLCD regimens is not recommended (Mustajoki & Pekkarinen, 2001). In regards to the remaining options, the weight loss success associated with low carbohydrate/high protein diets versus low fat diets, also seem to present little to no difference in their level of efficacy after one year (Hession, Rolland, Kulkarni, Wise & Broom,

2009). Laddu et al. (2011) indicated that nutrient composition might therefore not be the predominant factor in achieving weight loss, since all diets described involve a significant decrease in overall caloric intake. Regardless of the variations in the noted diet plans, data cited from the National Weight Control Registry (NWCR), a collection of data tracking over 5,000 individuals that achieved significant weight loss and maintenance for at least 12 months, has shown that strategies such as eating breakfast and modifying existing food intake significantly contributed to successful weight loss (NWCR, 2013; Grief & Miranda, 2010).

Based on the overall evidence, it therefore appears that a diet consisting of higher amounts of protein, lower consumption of carbohydrates, and in which fats make up no more that 30% of total intake, seems to be most promising (NIH, 1998). In addition, according to the Academy of Nutrition and Dietetics recommendations for adult weight loss, energy intake should also be evenly distributed through 4 to 5 meals over the course of a day, portion control should be emphasized and nutritional education regarding food labels and preparation should be available to those embarking on a weight loss plan (AND, 2013).

Exercise

While the noted dietary changes are essential in the pursuit of weight loss, increased physical activity is also paramount in reducing the imbalance between energy intake and expenditure. Current recommendations indicate that exercise should be combined with caloric reductions and behavioral interventions for maximized success, as the standalone benefits of physical activity

may not be enough to overcome the large weight gains observed in some individuals (Poirier & Despres, 2001). However, the 2009 position paper by the American College of Sports Medicine (ACSM) indicated that exercise alone could successfully prevent significant weight gain (of over 3%) and could even result in moderate amounts of weight loss, but only when the amount of exercise is relatively high. In other words, it appears that weight loss is directly proportional to the intensity and duration of exercise performed (Donnelly et al., 2009). While this evidence certainly supports exercise as an independent treatment modality, it may also raise questions regarding the suitability and risks of such high amounts of activity. Therefore, within the context of weight loss intervention programs, multifaceted approaches remain optimal, and exercise is to be used as an adjunct treatment.

In examining the makeup of recommended exercise types, the 2008 Physical Activity Guidelines for Americans (presented by the CDC) suggests that both aerobic and muscle strengthening activities are necessary components (CDC, 2013). Other studies have shown similar benefits in combining aerobic and resistance exercise training toward reducing fat loss, improving cardiovascular functioning, and increasing leas muscle mass (Ho, Dhaliwal, Hills & Pal, 2012; Willis et al., 2012). Resistance training appears to be particularly important in reducing the aging effects that result in a loss of muscle mass in older individuals (Avila, Gutierres, Sheehy, Lofgren & Delmonico, 2010). Additional recommendations by the CDC note that adults should perform at least 150 minutes of moderate intensity aerobic activity, or 75 minutes of vigorous intensity activity per week. This should also be paired with two days of resistance training. For

increased benefits, the CDC set a goal of 300 minutes of moderate aerobic activity, or 150 minutes of vigorous intensity activity, in conjunction with two days of resistance training each week (CDC, 2013).

Research studies have consistently discovered similar results. Data has shown that exercise programs of under 150 minutes per week only result in minimal weight loss, while activities between 250 minutes and 420 minutes per week are most effective (Donnelly et al., 2009). National Institutes of Health (NIH) guidelines also suggest initiation of daily exercise regimens consisting of 30 minutes to 45 minutes per day, 3 to 5 times per week (NIH, 1998). This accumulates to a total amount of 90 minutes to 225 minutes per week. However, a critical aspect in maintaining adequate exercise levels and regulating caloric intake is an overall reduction of sedentary behaviors. Owen et al. (2010), describe that sedentary behaviors can lead to premature mortality and other metabolic changes. Matthews et al. (2012) noted that sitting and TV watching of over 7 hours per day is also associated with higher risks of cardiovascular problems and mortality. In addition, even those individuals engaging in 150 minutes or greater weekly physical activities can negate their benefits through prolonged sitting. This is an occurrence that the authors describe as the "Active Couch Potato" (Owen et al., 2010). Ultimately, it seems that individuals that are successful in achieving weight loss maintain an overall active lifestyle, even outside the physical activities performed.

Psychological Interventions

A review of pooled data from various psychological intervention studies aimed at weight management indicated that subjects using cognitive behavioral strategies, or behavioral strategies alone, displayed a greater amount of weight loss, particularly when paired with dietary and exercise modifications (Shaw, O'Rourke, Del Mar & Kenardy, 2005). Results from various randomized controlled trials (RCTs) also noted a weight loss of approximately 11% for studies that implemented various behavior therapy methods (Wadden, Butryn & Wilson, 2007). Poston and Foreyt, (2000) concluded that behavior modification programs should generally be composed of strategies that include self-monitoring, stimulus control, cognitive restructuring and various stress management techniques.

Self-monitoring refers to the regular recording of both food intake and exercise habits in order to become increasingly aware of the contexts and behaviors in which one engages (Berkel, Poston, Reeves & Foreyt, 2005). For example, by becoming aware of the total amount of caloric intake, including nutritional content, patients could begin to modify their diet and exercise levels to better support weight loss. Self-monitoring is also often paired with various goal-setting phases and psychoeducation, in order to establish more concrete guidelines for food and exercise habits, and to teach healthy living principles (Berkel, Poston, Reeves & Foreyt, 2005). It has also been shown that self-monitoring results in more positive treatment outcomes, such as a greater amount of weight lost, and increased overall adherence (Acharya et al., 2009; Baker & Kirschenbaum, 1993; Boutelle & Kirschenbaum, 1998).

Stimulus control refers to the environmental changes that an individual can initiate, which eventually lead to a change in overall behaviors. Cues associated with negative behaviors are assessed and removed, in an effort to reduce chances of relapse (Berkel, Poston, Reeves & Foreyt, 2005; Butryn, Webb & Wadden, 2011). For example, decreasing the availability of high calorie and high fat foods, while simultaneously increasing healthy eating options, could help a patient develop better control over their food intake. Various problems solving methods and cognitive restructuring tools have also been implemented in an effort to identify treatment barriers, improve self-efficacy, and assess and modify existing maladaptive thought patterns that are not conducive to weight loss (Berkel, Poston, Reeves & Foreyt, 2005; Annesi, 2011). This allows individuals to evaluate negative thoughts, improve coping, and develop positive alternatives that are feasible and more adaptive to their current goals. Lastly, stress reduction strategies and relaxation techniques such as diaphragmatic breathing, guided imagery, meditation, and progressive muscle relaxation have also been shown to reduce overeating and relapse rates (Poston & Foreyt, 2000).

Pharmacological Options

Although many changes have occurred in the pharmacological treatment of obesity over the years, this area remains a promising avenue for future weight management programs. National Institutes of Health (NIH) guidelines for the implementation of anti-obesity drugs recommend the use of Food and Drug Administration (FDA) approved medications only in conjunction with dietary, physical activity, and lifestyle modifications. In addition, use of pharmacological

therapies is only advised in patients with no obesity risk factors and a BMI of 30 kg/m^2 or above, or patients with additional risk factors and a BMI of 27 kg/m^2 and above (NIH, 1998). Currently the only three FDA approved medications for weight loss are orlistat, lorcaserin and a combination of phentermine and topiramate (PHEN/TPM), as the other previously used drugs are now discontinued and have been deemed unsafe due to their side effects profiles (Wyatt, 2013).

Bariatric Surgery

Sometimes considered as a last resort, bariatric surgery is currently an option for severely obese patients who may have not been able to achieve control over their obesity using other means of weight loss. The NIH guidelines indicate that surgery is recommended only for patients with a BMI of at least 40 kg/m^2 (morbid obesity), or a BMI of 35 kg/m^2 and above if additional obesity related risks are also present. However, it has also been noted that while both gastric restriction (banding) and gastric bypass (Roux-en Y) can result in significant weight loss, patients can also be subject to various adverse consequences, including malabsorption issues, and various nutrient and vitamin deficiencies (NIH, 1998). A review of various gastric bypass and banding studies indicated that while significant weight loss was achieved by both procedures, gastric bypass stood out as the most successful in reducing weight. The authors of one study reported that 76% of patients lost excess weight after bypass surgery, in comparison to 48% that underwent gastric banding (Tice, Karliner, Walsh, Petersen & Feldman, 2008). Other studies by Nguyen et al., (2013) and Angrisani et al. (2007) provided further support for greater BMI reductions with

gastric bypass surgeries, independent of whether or not the procedures were performed laparoscopically.

Overall, it does appear that weight loss surgeries are viable option for rapid weight loss, particularly with severely obese patients that also possess high amounts of comorbidities. In regards to the procedure type, an important aspect to keep in mind when deciding on the most appropriate methods for a patient would be to thoroughly consider the operative and post operative risk profiles of each.

Depression

Psychotherapy Treatment

There continues to exist some debate on whether psychotherapy alone, or psychotherapy combined with pharmacotherapy is the best treatment option for depression. When examining remission rates for major depression, the data does support the notion that a joint treatment approach is more beneficial, as a difference of 12% was noted between combination treatment and psychotherapy alone, in individuals with moderate, chronic depression (de Maat, Dekker, Schoevers & Jonghe, 2007). Other studies have also depicted more favorable outcomes for combined therapies compared to either treatment alone (Cuijpers, van Straten, Warmerdam & Andersson, 2009; Pampallona, Bollini, Tibaldi Kupelnick & Munizza, 2004). Current research indicates that there is little to no difference between psychological and pharmacological

treatment options when they are examined independently. A study assessing disparities and effects reported equal efficacy between the two treatment modalities for mild to moderate depression, even though the authors noted that most patients preferred psychotherapy alone (99% versus 84%) (de Jonghe et al., 2004). The predominant choice in psychotherapy could perhaps be due to patients' perceptions of the various side effect profiles of anti-depressant medications. Another study examining psychotherapy and second-generation anti-depressants, also reported nearly identical short-term treatment effects, but mentioned that psychotherapy was more effective than medications long term (Spielmans, Berman, & Usitalo, 2011). When examining psychotherapy as a single treatment modality for depression, a recent meta-analysis conducted by Cuijpers, van Straten, Hollon and Andersson (2010b) revealed an average effect size of .68 for all studies included. However, the authors also mentioned that when considering only the highest quality studies, the effect size was only .22 for psychotherapy (Cuijpers, van Straten, Bohlmeijer, Hollon & Andersson, 2010a). This indicates that while psychotherapy serves as an effective treatment, clinicians may have overestimated its standalone benefits.

Perhaps increased focus needs to be brought to the various categories of psychotherapeutic treatments. A 2008 meta-analysis identified seven potential psychotherapies for mild to moderate depression, in an effort to determine if there were any differences in their efficacy. Despite this goal, the results from the analyses suggested similar effect profiles for all of the modalities involved (cognitive behaviors therapy (CBT), nondirective supportive treatment, behavioral activation treatment, psychodynamic treatment, problem solving therapy, interpersonal therapy

(IPT) and social skills training) (Cuijpers, van Straten, Andersson & van Oppen, 2008). Additional research in this area noted that in the treatment of mild or moderate depression, CBT and IPT were presumed to be most beneficial, while the combination of psychotherapy (no specified subtype) and antidepressants were also likely to be helpful and to reduce symptoms (Butler, Hatcher, Price & Von Korff, 2006).

Pharmacotherapy Treatment

Similar to the use of psychotherapy, pharmacotherapy is often used in conjunction with other modalities in the treatment of depression. As stated above, combination treatments are generally thought of as the most effective (Price, Butler, Hatcher, & Von Korff, 2007). Nonetheless, the use of antidepressants still remains a primary action plan for many practitioners. The British Association for Pharmacology (2006 revision) guideline recommends using antidepressants as a "first line treatment" for patients with moderate and severe depression (Anderson et al., 2008). Evidence from a systematic review examining the effects of various medications, indicated that for severe types of depression, antidepressants and electroconvulsive therapy (ECT) were indeed thought to be the most beneficial treatment (Cipriani, Barbui, Butler, Hatcher & Geddes, 2011). A meta-analysis of drug effects and depression severity also reported that the two appear to be directly related. The authors discovered that in the most severely depressed patients, the effects size seemed to be the highest (.47) in the use of antidepressants (Fournier et al., 2010). The American Psychiatric Association (2010 revision) further supports this notion, but also suggests pharmacotherapy should be implemented as an initial treatment even for mild to moderately

depressed patients (APA, 2010). It is possible that as a result of these recommendations, antidepressants have increased in use and distribution over the years. A 2010 study examining U.S. trends in depression from 1998 to 2007, indicated that approximately 75% of individuals receiving outpatient treatment for depression were prescribed an antidepressant over a 12-month period (Marcus & Olfson, 2010).

With increased use of psychotropic medications in the treatment of depression, another important aspect to understand is the efficacy of pharmacological treatments. In their assessment of various MDD treatments, Dimidjian et al. (2006) discovered that pharmacotherapy consisting of an SSRI (paroxetine) resulted in an effect size of approximately .65 for severely depressed patients when compared to placebo. Cuijpers, et al. (2009) noted a .35 increase in effect size, when adding antidepressants to existing psychotherapy. Another .25 increase in effect size was also observed in a second study, when active medication was used within a combined treatment approach (Cuijpers et al., 2010a). However, when describing patient preferences, a study by Kwan et al., (2010) noted that approximately 48% of patients preferred mono-treatment for depression with psychotherapy, while only 18% of individuals preferred pharmacotherapy. This could certainly suggest some hesitation in beginning pharmacological treatment due to possible side effect profiles associated with the medications. Nonetheless, there also appears to be evidence that the use of pharmacotherapy is a necessity in patients suffering from more severe forms of depression.

Commonly known classes of antidepressants generally fall in the selective serotonin reuptake inhibitors (SSRIs – such as fluoxetine, sertraline, paroxetine, fluvoxamine, citalopram and escitalopram), selective norepinephrine reuptake inhibitors (SNRIs - such as venlafaxine, desvenlafaxine and duloxetine), tricyclic (TCAs - such as imipramine and maprotiline), monoamine oxidase inhibitors (MAOIs – such as phenelzine, tranylcypromine, isocarboxazid and selegiline), or "other" category (buproprion, nefezodone, trazodone and mirtazapine). The APA guidelines note that an antidepressant class should only be chosen after a thorough evaluation of risks and benefits in each patient and should be based on the individual effect profiles of the medications (APA, 2010). Various studies have attempted to evaluate the difference between the available classes of medications, but these have yielded mixed results. Although an SSRI is typically the first antidepressant to be prescribed, if there is a lack of response in patients, switching to another SSRI, an SNRI, or a medication in the "other" category (such a buproprion), results in equivocal treatment impact rates (Rush et al., 2006). Within primary care settings, a 2009 review indicated both SSRIs and TCAs are commonly prescribed and have been found to be similarly effective in treating depression, although the authors reported an occurrence of decreased side effects with the use of SSRIs (Arroll et al., 2009). Similarly, although as effective, MAIOs are also seen in a more negative light due to their negative side effects (such as hypertensive crisis and serotonin syndrome), high drug interactions and necessary adherence to a restricted diet. As a result, they are now thought of as "third, or fourth line" treatments for depression (Thase, 2012).

Children and Adolescents

Overview

While there is no specific classification for successful weight loss in children, a weight loss of 5 to 10% will significantly reduce cardiovascular health risks in adulthood (Blackburn, Beatrice & Kanders, 1987; Wing et al., 2011). For children, a set weight loss amount might be difficult to recommend, particularly due to their continued growth. Reinehr (2011) noted that in overweight youth, maintaining the same weight over 12 months is similar to a 1kg/m^2 to 2kg/m^2 decrease in BMI. Weight stability might therefore be a safer option for children, instead of focusing strictly on a target amount of weight to be lost. Nonetheless, similarly to adults, children also need to modify their diet, exercise and overall behaviors, in order to achieve a healthier weight. Currently, two potential of methods of decreasing the sedentary lifestyles (recommended by the American Academy of Pediatrics – AAP) of children include reductions in screen time exposure (TV, video games, computer) to less than two hours per day and increases in overall activity levels (AAP, 2012). The current CDC recommendation for physical activity in children and adolescents is a minimum of 60 minutes per day of moderate to vigorous aerobic activity, as well as muscle and bone strengthening exercises (CDC, 2013). In regards to dietary needs, similar caloric reductions as in adults are needed, with no greater than 25% to 40% (age dependent) of overall intake being derived from fats (USDA & USDHHS, 2010). The reduction in dietary fats paired with the elimination of sugary drinks and fast food choices may be an important approach to consider. A RCT intervention that promoted increased water consumption for elementary

school children noted a reduction in overweight risk for those that followed the study recommendations (Muckelbauer et al., 2009). Other studies aimed at providing interventions to decrease sugary drink consumption have also described similar success in reducing BMI for children (James, Thomas, Cavan & Kerr, 2004; de Ruyter, Olthof, Seidell & Katan, 2012).

In an assessment of the relationship between fast-food availability and BMI, Galvez et al. (2009) reported higher BMI percentiles for children living in closer proximity to fast food restaurants. Portion control plates may also be helpful in allowing adolescents to be more cognizant of appropriate food portions and selecting of healthier options (Bonhert, Randall, Tharp & Germann, 2011). DiSantis et al. (2013) found that when children were provided with adult sized plates (of 100% increased surface area), they consumed and served themselves a greater caloric intake. In addition, the self-monitoring of food and exercise habits have also shown great benefits in several studies (Germann, Kirschenbaum, Rich & O'Koon, 2006; Germann, Kirschenbaum & Rich, 2007; Mockus et al., 2011).

Behavioral interventions, which include the implementation of cognitive behavioral therapy, problem solving strategies, positive reinforcement programs, and goal setting have further been summarized as effective tools in weight management practices (Stewart, Rilley & Hughes, 2009; Seagle, Strain, Makris & Reeves, 2009). However, parental involvement and support appears to be critical when it comes to children and weight loss. Research suggests that the parents making the necessary changes within the home environment and actively participating in their child's

management of their weight through the modeling of healthy and appropriate behaviors (and losing weight), seem to promote greater weight loss in overweight and obese children (Heinberg et al., 2010; Boutelle, Cafri & Crow, 2012).

However, despite the noted success in using various weight management interventions, high attrition rates and a lack of motivation could pose additional barriers to long-term treatment success. Citing evidence from several long-term follow-up weight loss programs for children, Moens, Braet and Van Winckel (2010) indicated that weight management interventions might only benefit approximately 50% of obese children. In their study, approximately 40% of participants had a BMI reduction of 10% of more (Moens et al., 2010). The low success rates can be partly due to the limited ability to maintain the initial weight loss achieved. Further exploration of an individual's motivation to change using the Transtheoretical Model framework (Prochaska & DiClemente, 1982) could provide some answers in regards to the success of various patients (Johnson et al., 2008), as well as their potential propensity for relapse. In addition, an evaluation of the behaviors and strategies used after outpatient support when the weight control program is ceased could help us understand how some individuals are able to maintain weight loss over time. An examination of parental support, psychopathology, and family SES, may also provide information on the amount of parental involvement necessary to promote successful weight management strategies (Moens et al., 2010).

These future research directions would be particularly relevant for low income and minority populations, as they are not only subject to higher rates of obesity, but are also often faced with additional barriers towards treatment. However, there seems to be a limited amount of review data available in the literature for interventions specific to minority and low-income populations. A meta-analysis by Seo and Sa (2010) that focused on weight management interventions specifically for American minority children, sought to address this research gap and synthesize the available data. The authors noted the importance of providing interventions in three (effect size of .33) or more (effect size of .71) components to both children and parents, in the areas of exercise, diet, sedentary behaviors, and counseling, in order to effectively promote weight loss. They also indicated that further attention should be given to the development of daily routines and provision of culturally relevant interventions for minority youth (Seo & Sa, 2010). Kumanyika & Grier, (2006) suggest intervening at the national level, by implementing various policies to help minority children gain access to improved nutrition (through WIC programs), to obtain better health services and insurance coverage (Medicaid) and to decrease targeted food advertising campaigns.

Depressed and Obese Patients

Although some debate regarding directionality remains, from the above noted evidence, it is apparent that a significant relationship between depression and obesity does exist. This comorbidity certainly presents a new set of difficulties for practitioners attempting to simultaneously manage both conditions. Currently, there is scarcity of data available on the

provision of interventions and treatment to this specific group of individuals. Nonetheless, in a study examining the relationship between overweight, obesity and psychiatric disorders, Petry et al. (2008) indicated that 10% to 20% of obese subjects were noted to have comorbidities consisting of either a mood, or anxiety disorder. The authors also felt that simultaneous treatment of both, the medical issues related to weight and psychiatric diagnoses would be most helpful for these patients (Petry, Barry, Pietrzak & Wagner, 2008). However, some difficulties can arise when attempting to initiate a combined treatment approach. For instance, many of the commonly used antidepressant medications are also known to result in significant amounts of weight gain with long-term use, particularly TCAs and MAOIs (Russ & Ackerman, 1988; Serretti & Mandelli, 2010). Other complications and issues that could perpetuate obesity in these patients are the depression related symptoms. Increases in appetite (atypical depression), decreases in sleep, and lower energy levels or anhedonia, may ultimately result in a greater imbalance between energy intake and expenditure. Despite these issues, some successful interventions have been noted within the treatment of obese and depressed patients.

In a pilot study that treated comorbid obesity and depression through CBT interventions (consisting of weekly group session focused on weight management, self-monitoring, stimulus control and the cognitive model), Faulconbridge, et al. (2011) discovered that not only did study completers lose approximately 11% of their initial weight within a 16-week period, but 66% of them were also no longer depressed. In a second study, similar results were reported, as subjects that participated in the intervention lost approximately 9% of initial weight after 12 months and

simultaneously described a significant decrease in their depressive symptoms (Faulconbrige et al, 2012). In a meta-analysis by Fabricatore et al. (2011) on the relationship of weight loss effects and depressive symptoms, it was discovered that lifestyle modifications (consisting of exercise and diet instructions, as well as behavior therapy) and exercise were the most successful treatment modalities for promoting symptom reductions. The authors hypothesized that perhaps various cognitive and behavioral changes could have resulted in a more positive mood for subjects, and that engaging in a group format, particularly during the exercise interventions, may have also increased social support and resulted in normalization effects (Fabricatore et al., 2011).

The implementation of behavioral activation strategies may be another effective tool. Although limited by sample size, a study by Pagoto et al. (2008) demonstrated that the combination of behavioral activation (though self monitoring, goals setting, problem solving, exercise) and nutritional counseling was an effective treatment for both weight loss and depression. Participants were noted to have had an average weight reduction of 5.55 pounds and 72% of them also achieved remission of depression over a 12-week period (Pagoto et al., 2008). Additional hypotheses for future treatment of obesity and depression also suggest providing interventions focused on the "neuroprogressive pathway" by the use of various anti-inflammatory medications and behavioral changes to increase "neurogenesis" (Lopresti & Drummond, 2013). This approach would certainly fit with the previously stated notion that obesity may be a result of a cytokine induced inflammatory process.

FitMatters Protocol

The FitMatters program was initiated in 2000 at LaRabida Children's Hospital/ University of Chicago, which serves the surrounding community primarily consisting of low-income minority families. Rolling enrollment meant that the program was open at any time any families meeting the inclusion/exclusion criteria. Participants were required to be 8 years or older, and to present with a BMI in the 95th percentile or above for their gender and age. Participation in the concurrent research study was voluntary, and families were not excluded from the program if they chose not to participate in the research study. During the active enrollment period of the protocol from 2000 to 2008, approximately 456 obese (above the 95th percentile) children/adolescents (ages 7- 19) and their families participated in the study.

The program assessed various weight loss predictors through a multidisciplinary approach consisting of group therapy for children and their parents, cognitive-behavioral therapy (CBT), nutritional counseling, exercise therapy, and medical management of obesity-related health conditions (e.g., high cholesterol, high blood pressure, polycystic ovarian syndrome). Families were encouraged to participate in the study for at least one year. More thorough evaluations (e.g. physical exam, intelligence testing) were performed at enrollment, while outcome and process measures (e.g. self-monitoring and weight) were recorded every week. After enrollment in the study, participants were registered for a baseline fitness evaluation with a physical therapist, and subsequently met with a dietitian every month, for ongoing support and education regarding healthy eating habits and the importance of increased physical activity. They were also scheduled

to visit the Metabolic Clinic every six months for medical screening and monitoring (e.g. history and physical assessment, lab draws for cholesterol, insulin, blood glucose and hormone levels) by a pediatric endocrinologist.

Long-term treatment via weekly small group sessions (of 3-6 individuals) was provided to both children and their parents. A need for active encouragement was highlighted, as parental attendance during therapy was required for study participation. Behavioral contracts were also implemented by study team members and were signed by the parents and children at the onset of the study. The CBT treatment strategies were geared towards providing psychoeducation to promote the development of coping skills and other lifestyle changes.

STUDY AIMS

Psychosocial and Psychological Factors

The role of known contributors to weight loss success within a sample of African-American youth was examined. Although both psychological and psychosocial factors have been previously evaluated in obese children, data remains limited for minority and low-income populations.

1. The relationship between various psychosocial factors and weight management outcomes for the current sample was assessed.

2. The unique impact of depressive symptomatology on weight loss success in African-American children and adolescents was examined.

Adherence Factors

3. The relationship of adherence factors (self-monitoring and attendance) and weight outcome was evaluated. This included the following:
    a. An evaluation of psychosocial, depressive symptomatology, and self-monitoring as predictors of attrition.
    b. An evaluation of adherence and depressive symptomatology as predictors of weight outcome.

HYPOTHESES

Psychosocial and Psychological Factors

1. Positive psychosocial factor were expected to predict a higher degree of weight loss, while negative psychosocial factors were hypothesized to result in a lower degree of weight loss, or a weight gain. Positive factors consist of healthy eating, good exercise and weight control habits, low family conflict, higher self-efficacy, higher intelligence scores, lower depressive symptomatology, and higher family SES. Negative factors include poor eating, exercise, and weight control habits; higher family conflict, lower self-efficacy, lower intelligence scores, as

well as increased depressive symptomatology and lower family SES. Age and gender are two additional factors that were explored as potential weight loss contributors.

2. Individuals with higher depressive symptomatology were predicted to have a higher BMI and to exhibit a lower degree of weight loss, or result in a weight gain.
    a. Higher initial CDI total scores were expected to positively correlate with higher baseline z-BMI, and to predict a lower degree of weight loss, or a weight gain.
    b. Higher CDI individual subscale scores were anticipated to be positively correlated with higher baseline z-BMI, and to also predict a lower degree of weight loss, or a weight gain.

Adherence Factors

3. Adherence factors were expected to influence weight loss.
    a. It was expected that psychosocial factors, self-monitoring, and depressive symptomatology would predict attrition.
    b. Adherence factors in conjunction with depressive symptomatology were also expected to predict weight outcomes. Greater adherence and lower depressive symptomatology were predicted to result in a higher degree of weight loss, while poor adherence and higher depressive symptomatology were predicted to result in a lower degree of weight loss, or a weight gain.

CHAPTER THREE
Methodology

DESIGN

General Study Outline

This study was a secondary analysis of previously collected data from a weight control intervention program (FitMatters) for children and adolescents at LaRabida Children's Hospital/University of Chicago from 2000 to 2008. Treatment within the study was focused on the provision of various weight management strategies for low-income minority children and adolescents, and their parents. Data analyzed for the current study was therefore based on the initial FitMatters protocol design.

Current Study

While the current study made use of the data collected and the structure outlined in the FitMatters protocol, it was more specific in its scope, with distinct study aims and analyses. This study assessed the data from the FitMatters program over the entire study course (eight years). The current study focused specifically on African-American participants and their families with available Body Mass Index (BMI) measures and initial Children's Depressive Inventory (CDI) scores. The study protocol outline was provided to the UT Southwestern IRB, as well as the University of Chicago IRB, and was approved under exempt status. A written data sharing and

material transfer agreement was also completed for access to the de-identified study database from the FitMatters program.

PARTICIPANTS

Sample

Current study participants were 206 African-American children and adolescents (with BMIs above the 95th percentile) between the ages of 7 to 18 years old and their parents, for whom baseline Children's Depressive Inventory (CDI) scores and body mass index measures (BMI and z-BMI) were recorded. Only African-American subjects were selected for the current study due to African-American children and adolescents representing approximately 86% of the total FitMatters sample (N=456). The remaining 14% of the sample consisted of Hispanic, Non-Hispanic white, and other (including missing data) ethnicities, which did not leave an adequate amount of subjects in each ethnic group to conduct comparative statistical analyses. The sample was further tapered when including only those African-American participants with initial CDI scores and at least one baseline and one post-intervention BMI measurement (z-BMI score). Other subjects were excluded due to missing data in the above noted categories. The final sample size is therefore comprised of 206 subjects between the ages of 7 and 18 years old (see Figure 1).

Consent

Trained study team members obtained written consent from all FitMatters program participants at the time of their enrollment in the program. They explained that participation was voluntary and noted the various procedures and the extent of involvement once enrolled in the program. Study benefits and risks were explained, confidentiality was thoroughly discussed, and all questions were answered prior to the signing of the consent form documents. The participant's parent or guardian provided consent, while the child provided written assent. The University of Chicago Institutional Review Board (IRB) approved all consent materials and study documents prior to enrollment. Copies of the consent documents were placed in the patient's medical record and were provided to each family enrolled. Since the current study relies on secondary analyses and no new subjects were enrolled, no additional consent form documents were created for this purpose.

Confidentiality

In order to maintain confidentiality, study data were de-identified. Each data set corresponding to study participants was assigned a unique study ID number. The master list (computer document) linking the unique identifiers with the participant's medical records was password protected and was not part of the data transfer agreement. This information remained at La Rabida Children's Hospital Chicago and was accessible only by the study team members on site. The current study data (SPSS file) at UT Southwestern was also de-identified and was restricted to only those researchers directly involved with this study.

MEASURES

Initial Assessment Measures

Demographics

Socioeconomic Status (SES), age, Body Mass Index (BMI & z-BMI), insurance status, family constellation and intellectual functioning (Kaufman Brief Intelligence Test - KBIT) were noted.

Socioeconomic Status (SES) was calculated using the Hollingshead Index of Socioeconomic Status (Hollingshead, 1975). The index uses a four-factor model that examines education, occupation, sex and marital status, in order to calculate a total SES score for each individual within the home. Scores can range from a total of 8 to 66 points. The reference ranges for each category are as follows: 8-19 unskilled laborer, 20-29 machine operators, semiskilled workers, 30-39 skilled craftsmen, clerical, sales workers, 40-54 medium business, minor professional, technical, 55-66 major business and professional.

Body Mass Index (BMI) generally represents a measurement of body fatness and is calculated using the height and weight for each individual. The formula is computed by assessing weight in kilograms and then dividing that amount by a person's height in meters squared. However, for children and adolescents, measuring BMI in this fashion may not be accurate. As a result, BMI measures for children and teens are separated into age and sex specific percentiles. This information is accessible via the CDC BMI-for-age growth charts. For the current study, BMI

was assessed using the 2000 Center for Disease Control (CDC) growth charts for boys and girls between the ages of 2 to 20 years, taking into account age and gender specific differences (CDC, 2013).

Body Mass Index z-Scores (z-BMI) were also used to examine the distribution of the data. A z-score defines how far away a measure is (based on number of standard deviations) from the population mean, as well its direction. A normal standardized distribution z score has a mean (M) of zero and a standard deviation (SD) of 1. However, certain z-scores may use different scales that do not necessarily adhere to these criteria. Since BMI is known to change with age and weight, when comparing BMI measures with known reference values, BMI needs to be transformed or translated in order to more accurately reflect that particular distribution. The 2000 CDC report on US growth charts recommends using z-scores due to standardization of values, ability to make comparisons across age and sex, and ability to evaluate children outside defined percentile ranges. They also describe the z-score calculations and transformations that are necessary to obtain a normal distribution for a BMI value (Kuczmarski et al., 2000). Current procedures involve using the lambda, mu, sigma (LMS) curve-fitting and smoothing method (Box-Cox transformation) to correct skewness (Preedy, 2012). In the present study, the entire sample was comprised of obese children and adolescents with BMIs above the 95th percentile. As a result, the best way to discern weight changes, was to examine BMI z-scores for each individual. The BMI z-scores are therefore calculated by using the national BMI norms for gender, age and ethnicity (Flegal & Ogden, 2011).

Kaufman Brief Intelligence Test (KBIT) is a brief measure of intelligence for individuals between the ages of 4 to 90 years old. It assesses both verbal and non-verbal abilities via two separate subtests (Vocabulary and Matrices). It is individually administered and generally takes between 15 to 30 minutes to complete. The test was first published in 1990 (Kaufman, 1990).

Behavioral, Emotional and Family Functioning Measures

The Self-Perception Profile for Children (SPPC) (Harter, 1985) and The Self-Perception Profile for Adolescents (SPPA) (Harter, 1988) are two self-report measures of a child or adolescent's global self-worth and scholastic self-confidence levels. Initially administered to children 8 years old and above, it is now only given to youth between the ages of 12 and 14. Harder noted an internal reliability of approximately .8, yet the National Longitudinal Survey of Youth 1979 (NLSY79) reports this to be closer to .67 within their samples (NSLY79, 2013).

Child Behavior Checklist Revised Problems List (CBCL-RPL) is based on the original CBCL by Achenbach & Edelbrock (1983). It examines the parent's view of a child's behavioral issues, in areas of social and school functioning. Various behavior categories addressed include: aggression, delinquency, depression, thinking disturbances, somatic complaints, obsessive-compulsive behaviors, hyperactivity, and immaturity. The CBCL items are rated on a 3-point likert scale (0=not true, 2=very true or often true) and responses are normed for children between the ages of 12 to 16 years old. The one-week test-retest reliability of the checklist is .95, while

the three-month test-retest reliability is .84.

Youth Self-Report Revised Problems List (YSR-RPL) is based on the original YSR by Achenbach & Eledbrock (1989). It is a self-report measure for children between the ages of 11 to 19 years old that assess areas of difficulty within the last 6 months for the following categories: depression, unpopularity, somatic complaints, self destructive/identity problems, thought disorders, delinquency and aggression. The reported behavioral difficulties are rated on a 3-point likert scale (0 = not true, 2 = very true or often true). The one-week test-retest reliability is .86 and the eight-month test-retest reliability is .67. The YRS has also been able to note criterion related validity, as the higher scores correspond with adolescents referred for mental health services (Achenbach, 1991).

Pediatric Qualify of Life Inventory (PedsQL) (Varni, Seid, & Kurtin, 2001) is a self-report and parent proxy-report (for those between 2-5 years old) tool for children between that ages of 2 to 18 years old, that measures health related quality of life by also taking into account chronic and acute health conditions. Measures are separated into four dimensions, which consist of: physical, emotional, social and school functioning. The PedsQL has demonstrated adequate validity by being able to discern between healthy children, and children with various health conditions. Its reliability values are .88 for the child self report and .90 for the parent proxy report. Information can also be separated into two core scales (Physical Health Summary and Psychosocial Health Summary) as well as disease-specific modules such as diabetes, cancer, asthma and

rheumatology (Varni, 2013).

Conflict Behavior Questionnaire (CBQ) (Robin & Foster, 1988) is a self-report tool that examines conflicts and interactions between adolescents (10-19 years old) and their parent/guardian over a period of 2 to 3 weeks. Both parents and children complete independent forms assessing each other's behaviors and communication. This allows for an evaluation of the current level of conflict, communication deficits and potential conflict resolution aspects. The internal consistency of the questionnaire was reported to be .90 (only mothers and adolescents), with a test-retest reliability of 6 to 8 weeks. Average interrater reliability for parent-adolescent dyads are estimated to be approximately 67% when distressed and 84% when not distressed. The questionnaire data can be used to judge conflict severity through use of T-scores, which allow comparisons with normative samples. The CBQ is also used to isolate specific problems with the dyads and generate hypotheses regarding family structure. Robin & Foster further developed a CBQ short form version (CBQ-SF) comprised of a 20-item scale, in which only items having the highest correlation with total score and differentiating between families that were distressed versus non-distressed were used.

In the current study, only the data recorded from subjects' perceived conflict with their mothers were used due to the limited father reports that were completed.

Children's Depressive Inventory (CDI) is a 27-item self-report scale for children 7-17 years old that assesses the presence of depressive symptoms. The CDI t-scores are based on normative

samples that account for age and gender. It consists of the following scales: negative mood, interpersonal problems, ineffectiveness, andhedonia and negative self-esteem. It can be used to assess symptoms over time and has been demonstrated to have high ratings in reliability and validity (Kovacs, 1992).

Pre-Treatment Weight Control and Health Behaviors Measures

Weight Control Habits Survey (WCHS) is a self-report questionnaire that examines an adolescent's use of successful behavioral weight loss methods, as well as elements that might interfere with their success. Adolescents report how the items on each scale (11 items total) reflect their view of themselves by using a 1 (never or hardly ever) to 5 (always, or almost always) point likert rating system. The WCHS is based on the Eating Behavior Inventory Scale (EBI), which is also used to assess weight loss behaviors. The EBI has a one-month test-retest reliability of .74 and an odd-even spit-half reliability of .62. It has also been effective in differentiating between obese individuals that received training in eating habits and those that did not (O'Neil et al., 1979; Stalonas & Kirschenbaum, 1985).

Current Exercise Survey (CES) (Paffenbarger, Wing, & Hyde, 1978), is a self-report tool that adolescents can use to describes the types of activities or exercises performed over the last two weeks, the level of exertion (light, moderate, heavy) and duration they remained engaged. It is based on the questionnaire originally developed by Paffenbarger et al. (1978) that explored how physical activity could impact cardiovascular risk.

Harvard Service Food Frequency Questionnaire (HSFFQ) was initially named the Prenatel FFQ and was used to assess prenatal health for pregnant women. Questions were adapted from a FFO developed by Willett et al., (1985). The HSFFQ consists of 3 versions; one for pregnant women, one for children 1-5 years old and another for children 6-18 years old. Its purpose is to assess nutrition over the past month by presenting the responder with choices of the most commonly eaten food and portion sizes. It can be self-administered (Suitor & Gardner, 1992).

Eating Self-Efficacy Scale (ESES) (Glynn & Ruderman, 1986) is a self-report measure that examines the individual's perceived control over food intake within various contexts and settings. Adolescents report their level of food control using a 1 (no difficulty controlling) to 7 (most difficulty in controlling eating) point likert rating scale. The scale reaches two additional areas such as, difficulties in controlling consumption during periods of negative affect, and instances when it is socially acceptable to overeat. Internal consistency reliability for the ESES is .92 for the general scale, while the seven-week test-retest reliability is .70. It further displayed adequate convergent and discriminant validity.

Binge Scale (BS) (Hawkins & Clement, 1980) is a self-report tool (comprised of 19 forced choice items) that assesses binge-eating and self-induced vomiting after a binge. It has a reported internal consistency reliability of .68 and a one-month test-retest reliability of .88. The BS scale is able to accurately discern between control subjects and individuals with bulimia. Its total score

has been positively associated with dietary restraint and negatively related to self-image acceptance.

Readiness to participate and importance of losing weight self-reports were based on a 7-point likert and were used to evaluate overall readiness to change. A response of 1 indicated that participating or losing weight was "not at all," important; while a response of 7 indicated that this was "extremely important."

Fitness Levels Measures

A 6-minute walk test, Pre- and post-, and 5-minute-post- walk test was performed. In addition heart rate and oxygen saturation as measured by a finger probe pulse oximeter was also evaluated.

Physical Health Measures

Co-morbid diagnoses such as diabetes or obstructive sleep apnea, waist circumference, blood pressure, and a lab panel (DHEAS - adrenal hormones, Insulin - pancreatic hormone to regulate sugar, C-Peptide - pancreatic hormone to regulate sugar, HGB A1C - average blood sugar over 3-month period, Testosterone - Free and Total, Fasting Glucose, Lipid Panel – Cholesterol, Triglycerides, HDL Cholesterol, LDL Cholesterol, T4 - thyroid hormone, TSH - thyroid hormone, and 17OH-progesterone), were obtained.

Process Measures

Adherence

Self-monitoring and attendance were recorded at each weekly group meeting. Consistency of parental and child self-reporting was noted in days per week, while attendance was measured by the amount of sessions attended over the treatment period.

Outcome Measures

Weight (BMI, z-BMI) was recorded weekly, and changes were noted over the course of the treatment period. The z-BMI measures at monthly intervals will be of particular importance when examining outcome data, as these time points rather than weekly recordings will be the ones used in the statistical analyses.

DATA ANALYSIS

1. The first hypothesis stated that positive psychosocial factors would predict a higher degree of weight loss, while negative psychosocial factors would predict a lower degree of weight loss, or a weight gain.

A continuous linear mixed-effects repeated measures model was used to test the first hypothesis. Baseline psychosocial factors (gender, age, family SES, initial z-BMI, CDI total score, perceived

conflict with mother, weight control habits, total self-efficacy percentile, KBIT scores, and binge eating scores) and visits (the variable denoting time and comprised of each monthly interval) served as the predictor variables, while z-BMI over time was used as the outcome variable. For each psychosocial factor there is a coefficient to indicate the outcome direction as either positive "+" or negative "-" (meaning that an increase in the factor was associated with weight gain or weight loss in terms of z-BMI).

2. The second hypothesis stated that individuals with higher depressive symptomatology would have higher baseline BMI measures and would have a lower degree of weight loss, or would incur a weight gain. More specifically, it was predicted that:
    a. Higher initial CDI total scores would be positively correlated with higher initial z-BMI, and would predict a lower degree of weight loss, or result in a weight gain.
    b. Higher CDI individual subscale scores would be positively correlated with higher baseline z-BMI, and would predict a lower degree of weight loss, or result in a weight gain.

Three continuous mixed linear repeated measures models were used to examine the relationship between depressive symptomatology and weight change during the entire intervention period (12-month duration). The first continuous model involved the use of CDI total score (t-score) as the predictor variable, while z-BMI over time was used as the outcome variable. This repeated measures analysis examined the relationship between total depressive symptoms at baseline and

weight during each time point of the intervention (at 1,2,3…, or 12 months). A second continuous model was conducted using each CDI subscale score (total of 5 analyses) as a predictor variable, while z-BMI over time was set as the outcome variable. In this model each remaining CDI subscales were also used as covariates. A third continuous model was used to again examine each CDI subscale, but without co-varying for the remaining CDI subscales. Additional predictor variables for each of the three models included gender, age, and baseline z-BMI.

3. The third hypothesis stated adherence factors were anticipated to influence weight loss. More specifically:
    a. It was expected that psychosocial factors, self-monitoring, and depressive symptomatology would predict attrition during the first 3 months and 12 months of the study.
    b. Adherence factors in conjunction with depressive symptomatology were also expected to predict weight outcomes. Greater adherence and lower depressive symptomatology were predicted to result in a higher degree of weight loss, while poor adherence and higher depressive symptomatology were predicted to result in a lower degree of weight loss, or a weight gain.

Two separate models were used to test the third hypothesis. First, survival analysis was conducted to examine attrition over a 3-month and 12-month period through the use of various

Cox Regression Models. The initial Cox Regression analysis assessed the impact of all psychosocial variables, while subsequent analyses examined the impact of self-monitoring and depressive symptomatology in relation to attrition at 3 months and 12 months in the program. Predictor variables consisted of adherence (self-monitoring), depression measures (CDI total and individual subscale scores), baseline z-BMI, age, gender and psychosocial variables. This provided an opportunity to measure the rate at which subjects dropped out of the study and to examine potential attrition contributors. A second analysis was applied through the use of a mixed-effects linear repeated measures model to examine the continuous impact of depressive symptomatology and program adherence on weight outcomes. Initial depression scores (CDI), gender, age, baseline z-BMI, weeks in treatment, and average weekly food monitoring were used as predictor variables, while z-BMI over time was used as the outcome variable.

CHAPTER FOUR
Results

SAMPLE CHARACTERISTICS

Demographics

A total of 206 African-American subjects between the ages 7 to 18 years old (M=12.5) years were included in this study. Almost twice as many female participants were present, compared to male participants. School level ranged from the 2nd to the 12th grade, and body mass ranged from 23 kg/m^2 to 108 kg/m^2 (M= 42 kg/m^2). All 206 participants completed the children's depression inventory at the beginning of the program, yielding an average score of 9 out of 28. Socioeconomic status of the sample was measured by utilizing Hollingshead classifications. Although most parents/guardians were categorized as machine operators or semiskilled workers, the average individual was either a skilled craftsmen, clerical or sales worker. The majority of parents/guardians of each participant were single, divorced, or separated, biological mothers. Only 17 fathers completed demographic questionnaires. In addition, almost four times as many families noted being part of a government funded insurance policy, compared to a private policy. See Table 1 for additional demographic characteristics of the sample.

RESEARCH HYPOTHESES

Aim 1: Psychosocial Factors as Predictors of Weight Outcome

The first hypothesis stated that positive psychosocial factors would result in higher degree of weight loss, while negative psychosocial factors would result in a lower degree of weight loss, or in a weight gain.

Linear Mixed-Effects Repeated Measures Model Analysis Preparation

A linear mixed-effects repeated measures model was used to assess the continuous relationship of psychosocial factors and weight. Each individual's subject number (ID) was used to represent the subjects variable, while visits at monthly intervals for a one-year duration represented the repeated measures variable for models with correlated residuals within the random effects. Each subject's intercept was a random effect. A covariance type of first order auto-regression (AR1) was used to examine repeated effects for the subjects and visits variables. In addition, z-BMI over time (a continuous variable) was used as the dependent variable (outcome); gender and visits were used as categorical factors, while age, baseline z-BMI, CDI scores, maternal conflict ratings, family SES, weight control habits, self-efficacy scores, exercise scores, KBIT composite standard scores, and binge eating scores were chosen as predictor variables. All categorical factors and predictors were assessed as fixed effects using a main effects model.

Mixed-Effects Repeated Measures Model Output

When using a linear mixed-effects repeated measures model to assess changes at each month over a one-year period, 134 subjects were part of the analysis. Type-3 tests of fixed effects was significant for baseline z-BMI, $F(1,139)=3123.85$, $p<.001$ and visits, $F(11,544)=2.57$, $p=.004$

(Table 2). For baseline z-BMI, the direction of the relationship with z-BMI over time was positive, as a higher initial z-BMI corresponded with higher overall z-BMI measures at each month. A decrease was also observed in the marginal means with significant effects between visits (Table 3). No other psychosocial factors were significant.

Aim 2: Depressive Symptomatology as Predictors of Weight Outcome

The second hypothesis stated that higher depressive symptomatology (reflected from the total CDI score) would correspond with higher levels of baseline z-BMI, and would predict a lower degree of weight loss or result in weight gain. .

Correlations

Both, Pearson and Spearman correlations were performed in order to evaluate the relationship between depressive symptomatology and baseline z-BMI. The nonparametric (Spearman) correlation was significant at the 0.05 level (2-tailed) for the two variables, $r(204)=.14$, $p<.05$ (Table 4).

Linear Mixed-Effects Repeated Measures Model Analysis Preparation

A linear mixed-effects repeated measures model was used to assess the continuous relationship of depressive symptomatology and weight outcomes. Each individual's identification number (ID) was used to represent the subjects variable, while visits at each monthly interval for a one-year duration represented the repeated measures variable for models with correlated residuals

within the random effects. Each subject's intercept was a random effect. A covariance type of first order auto-regression (AR1) was used to examine repeated effects for the subjects and visits variables. In the first model, z-BMI over time (a continuous variable) was used as the dependent variable; gender and visits were used as categorical factors, and CDI total score, age, and baseline z-BMI were chosen as predictors. In the second model, the dependent variable and categorical factors remained the same, while age, baseline z-BMI, CDI negative mood scale, CDI interpersonal problems scale, CDI ineffectiveness scale, CDI anhedonia scale, and CDI negative-self esteem scale scores represented the predictor variables. In the third model, the dependent variable and categorical factors were once again unchanged, while age, baseline z-BMI, and each CDI subscale score (one at a time) represented the predictor variables. All categorical factors and predictors were assessed as fixed effects using a main effects model.

Mixed-Effects Repeated Measures Model Output

When using a linear mixed effects model to measure changes at each month over a one-year period, 164 subjects were part of the analysis. Results indicated that depressive symptomatology was not a significant predictor of weight outcome. However, type-3 tests of fixed effects was significant for baseline z-BMI, $F(1,180)=4145.21$, $p<.001$ and visits $F(11,678)=1.81$, $p=.049$ for the first model (Tables 5-6). For the second (Tables 7-8) and third models (Tables 9-13) baseline z-BMI also remained a significant predictor of weight.

Aim 3: Adherence Factors as Predictors of Weight Outcome

It was expected that psychosocial factors, self-monitoring, and depressive symptomatology would predict attrition. In addition, it was expected that adherence factors in conjunction with depressive symptomatology would also predict weight outcomes. Greater adherence and lower depressive symptomatology were predicted to result in a higher degree of weight loss, while poor adherence and higher depressive symptomatology were predicted to result in a lower degree of weight loss, or a weight gain.

Cox Regression Analysis Preparation

First, a Cox Regression model (survival analysis) was used to measure the rate at which subjects dropped out of the study. This provided an estimate of the dropout risks for subjects, while simultaneously allowing for the exploration of several predictor, or factor effects. The time variable was each subject's total weeks in treatment, while the status variable was a binary variable, which indicated whether or not a subject dropped out of the study before 12 months. Predictors used in the analysis were previously noted psychosocial factors, CDI total scores, CDI individual subscale scores, and self-monitoring variables. Variable such as age, baseline z-BMI, and gender, were predictors that were used in all Cox Regression models. Gender was entered as a categorical variable and values were plotted on separate lines for males and females in the final output. The initial Cox Regression analysis assessed the impact of psychosocial variables, while subsequent analyses examined the impact of self-monitoring and depressive symptomatology in relation to attrition during the first 3 months and 12 months in the program.

Cox Regression Output

In regards to the Cox Regression models, none of the hypothesized predictors were significant (Tables 14-17) for the first 3 months of the program, with the exception of gender, which was explored with additional analyses (see Figures 2-9). When evaluating predictors of attrition over the course of 12 months, several predictor combinations were found to be significant. Hazard ratios (HR) along with their confidence intervals (CI) were noted for each survival analysis. Generally, HR is a term used to describe the rate at which an event occurred during a specified time period. The final number derived is a constant ratio that can point out the differences between subject groups (e.g. treatment versus control group) according to the event defined (e.g. dropout). Since the current study consisted of only one treatment group (no controls), hazard ratio results were based on differences between gender (a categorical variable), in relation to overall attrition over the course of 12 months. Available subjects were 126, 206, and 174 for the assessment of psychosocial variables, depressive symptomatology and adherence factors respectively. After adjustment of psychosocial variables, gender was a significant predictor of attrition, with males being approximately 67% more likely to drop out of the study from one time point to the next during the entire 12 month intervention (HR=1.67, (95% CI: 1.03-2.70); p=.038; Table 18; see Figures 10-11). After adjustment of depressive symptomatology variables (either CDI total score or CDI individual subscale scores) as predictors of attrition, gender remained a significant predictor, with males having a 49% increased likelihood of dropping out. More specifically, when using CDI total score, the observed effect was HR=1.49, (95% CI: 1.07-2.09); p=.020 (Table 19; see Figures 12-13). When using individual CDI subscale scores, the

effect was HR=1.52, (95% CI: 1.08-2.13); p=.016, indicating a 52% greater attrition risk for males (Table 20; see Figures 14-15). In addition, a significant difference was also observed in the dropout rates between males and females over time when adjusting for self monitoring as a predictor variable, with an approximate dropout rate increase of 53% for males (HR=1.53, (95% CI: 1.06-2.22); p=.025) in comparison to females (Table 21; see Figures 16-17). While all predictor groups (psychosocial variables, depressive symptomatology and adherence factors) resulted in increased HR for males, the variance between them is due to the combination of variable included in each analysis and the change in sample size.

Linear Mixed-Effects Repeated Measures Model Analysis Preparation

A linear mixed-effects repeated measures model approach was used to assess the continuous relationship of adherence factors (attendance or weeks in treatment, and self-monitoring) and weight, while also taking into account depressive symptomatology. Each individual's identification number (ID) was used to represent the subjects variable, while visits at monthly intervals for a one-year duration represented the repeated measures variable for models with correlated residuals within the random effects. Each subject's intercept was a random effect. A covariance type of first order auto-regression (AR1) was used to examine repeated effects for the subject and visits variables. In this model, z-BMI over time was used as the dependent variable (outcome); gender and visits were used as categorical factors, and age, baseline z-BMI, CDI total score, total weeks in treatment, and weekly average food monitoring, were used as predictor

variables. All categorical factors and predictors were assessed as fixed effects using a main effects model.

Mixed-Effects Repeated Measures Model Output

When evaluating the data using a linear mixed-effects repeated measures model,164 subjects were included in the analysis. Type-3 tests of fixed effects was significant for baseline z-BMI ($F(1,177)=4084.87$, $p<.001$; Tables 22-23). Once again, higher initial z-BMI corresponded with higher overall z-BMI measures over 12 months. Adherence and depressive symptomatology factors were not found to be significant.

Exploratory Analyses

The exploratory analyses below were conducted on basis of the results obtained above and supported by available literature. Due to the differences observed in the marginal means for z-BMI (generally decreasing over time) and the significance of the visits variable in several linear mixed model analyses, it appeared that a time effect was present for weight outcomes when certain predictor variables were implemented. The three-month interval was thought to be an optimal time point for these analyses, as it would allow adequate time for change from baseline to occur. In addition, this time point would also remain early enough in the intervention program to where individuals losing weight more rapidly could be observed prior to attenuating effects taking place over time. Both, z-BMI at three months and average z-BMI over three months were individually used as outcome measures. Some of the existing literature suggests that early time

points for weight loss programs may be of great importance, as various interventions have shown that a faster rate of initial weight loss may be more effective than a gradual and slower pace (Nackers, Ross & Perri, 2010; Elfhag & Rossner, 2005; Jeffery, Wing & Meyer, 1998; Astrup & Rossner, 2000). Additional exploratory analyses were also conducted at the twelve-month time point in order to evaluate individuals that lost a target amount of weight at the conclusion of the study (one year). Current literature suggests that various interventions over the course of one year could lead to successful weight loss (NWCR, 2013; Grief & Miranda, 2010). A recent study by Faulconbrige et al. (2012) indicated that after participation in a 12-month weight loss program, subjects not only lost weight, but also noted a decrease in depressive symptomatology. As such, the inclusion of a one-year analysis may be helpful in assessing whether intervention related behaviors continue over time, leading to improved weight outcomes.

Furthermore, exploratory analyses were also conducted in order to evaluate subjects that reached a weight loss of 5% and 10% during their participation in the one-year intervention program and to examine potential predictors of this weight loss. The decision to conduct these analyses was based on existing literature and the potential clinical importance of this finding. Prior research indicates that a weight loss of 5% to 10% may greatly reduce cardiovascular health risks (Blackburn, Beatrice & Kanders, 1987; Wing et al., 2011) and other health issues in obese or overweight individuals.

Linear Regression – Aim 1:

Using a linear regression analysis to examine the impact of psychosocial factors on weight (z-BMI) at the first three months, baseline z-BMI was noted as a significant predictor (b=.99, t(92)=58.18 , p<.001) of weight (Table 24). Using a linear regression to examine the impact of the same psychosocial factors on the average weight (average z-BMI) over the first three months, baseline z-BMI continued to remain a significant predictor (b=.99 t(123)=74.40, p<.001; Table 25). However, no other significant effects were observed.

Linear Regression – Aim 2:

Using a linear regression analysis to examine the impact of depressive symptomatology on weight, with CDI total scores or CDI individual subscale scores as predictors at 3 months, baseline z-BMI was noted to be a significant predictor of weight b=.99, t(122)=66.04, p<.001 (Table 26) and b=.99, t(118)=64.56, p<.001 (Table 27), for total scores and CDI subscales scores respectively. Using a linear regression to examine the impact of the same depressive symptomatology factors on the average weight (average z-BMI) over 3 months, baseline z-BMI remained the only significant predictor of weight when including CDI total score (Table 28), or CDI individual subscale scores (Table 29) as predictors.

Linear Regression – Aim 3:

Using a linear regression analysis to examine the impact of adherence and depressive symptomatology factors on weight at 3 months, both baseline z-BMI (b=.99, t(120)=66.61, p<.001) and average food monitoring (b=-.033, t(120)=-2.82, p=.024) were significant predictors

of weight (Table 30). This indicated that greater food monitoring was associated with lower z-BMI measures. Using a linear regression to examine the impact of the same adherence and depressive factors on the average weight (average z-BMI) over 3 months, baseline z-BMI remained a significant predictor ($b=.99$ $t(157)=88.55$, $p<.001$; Table 31). When the linear regression was expanded to examine the impact of adherence and depressive symptomatology factors at 12 months, age was a significant predictor of weight ($b=-.139$, $t(36)=-2.33$, $p=.026$; Table 32). This suggests that older subjects had lower z-BMI measurements. However, when examining the impact of the same factors on average weight (average z-BMI) over 12 months, baseline z-BMI was the only significant predictor of weight ($b=.99$, $t(157)=72.76$, $p<.001$; Table 33).

Logistic Regression

Using various logistic regression models, psychosocial, depressive symptomatology, and self-monitoring variables were examined as potential predictors of a 5% or 10% weight loss for the duration of the 12-month intervention program. No significant predictors were found for the noted weight loss targets (Tables 34-39).

Kaplan-Meier Analysis Preparation

A Kaplan-Meier survival analysis was conducted to further examine individuals that achieved a 5% and 10% weight loss during the 12-month intervention program. The time variable was defined as the respective month at which an individual achieved the predefined weight loss. The

status was classified as a binary variable, with 1 indicating that the defined event occurred and 0 indicating that the event did not occur. Gender was also used as a factor, and male and female participants were compared for significant differences.

Kaplan-Meier Analysis Output

Results indicated that out of 164 total participants evaluated, 8 male and 26 female subjects reached a 10% weight loss by 12 months in the program. While the average time for this weight loss was 10.7 months for males and 10.3 months for females, overall, males had an approximate 15% chance of achieving the noted weight loss, while females had a 23% chance. However, although these percentages suggest a difference between males and females in achieving a 10% weight loss, this was not statistically significant (Table 40; see Figure 18). When examining participants for a potential 5% weight loss over the 12-month program, 17 male and 46 female subjects achieved this goal. The average time for this weight loss was 9.2 months for males and 8.4 months for females, with males having an approximate 32% chance of achieving the target weight loss, while females had a 41% chance. The success percentages between males and females were not significant. (Table 41; see Figure 19).

CHAPTER FIVE
Conclusions and Recommendations

DISCUSSION

As obesity rates continue to increase globally, various nations will not only be faced with the stark reality of increased mortality rates (Luppa, Heirich, Angermeyer, Konig & Riedel-Heller, 2007), but also with significant escalations in healthcare related costs (Greenberg et al., 2003; Tsai et al., 2011). For a country such as the United States, where approximately 35% of adults (Ogden, Carroll, Kit & Flegal, 2012) and 17% of children and adolescents are obese (CDC, 2013), these issues remain of great concern. Within our nation, it is apparent that low income and minority populations have even higher incidence rates, as disparities are clearly evident in surveys conducted by the CDC (2013). Various studies have also shown that comorbidities associated with obesity can further complicate treatment and lead to more negative outcomes. Mental health concerns such as depression (Erermis et al., 2004; Petry et al., 2008; Blaine 2008) or eating disorders and various psychosocial factors can play a major role within the development or the maintenance of this disease (Faith, Matz & Jorge, 2002; Stunkard, Faith & Allison, 2003). As a result, with the current understanding of obesity related complications throughout the lifespan, early detection and intervention appears to be critical. This means that in order to curb or even prevent further growth of these problems, focusing on creating more successful treatments for childhood obesity will be a pivotal step. The primary purpose of this

study was to evaluate the impact of depressive symptomatology within a group of African-American children and adolescents that participated in a weight loss intervention program. Additionally, other psychosocial and adherence related factors were also assessed as potential predictors of weight loss success.

Aim 1: Psychosocial Factors as Predictors of Weight Outcome

The first hypothesis indicated that certain psychosocial factors would either predict successful or unsuccessful weight loss outcomes within our sample. These factors consisted of gender, age, baseline z-BMI, CDI total scores, maternal conflict ratings, family SES, weight control habits, self-efficacy scores, exercise scores, binge eating scores and intelligence ratings. Using a mixed linear repeated measures model to examine the continuous relationship of various factors on weight over the 12-month intervention, no psychosocial factors were found to be significant predictors of weight change. Type-3 tests of fixed effects displayed significance only for the baseline z-BMI and visits (the monthly interval factor) variables. This meant that higher initial z-BMI (baseline) for each subject corresponded with higher z-BMI numbers at each month for the duration of the 12-month intervention. This trend was expected, as individuals that started the program with a higher z-BMI were expected to achieve a weight loss that was proportional to subjects starting at a lower initial z-BMI. Generally, when weight loss is achieved, it is measured as a percentage of an individual's initial body weight, and success is therefore assessed in relation to the person's own baseline numbers. In regards to the visits variable, this indicated a significant change over time in marginal means for z-BMI.

These results did not appear to directly support the existing literature, which notes a variety of psychosocial factors that can potentially impact obesity and weight management. Currently, significant differences exist between minority and Caucasian children and adolescents in regards to obesity rates. Similarly, some differences are also evident between male and female minority youth within the US, with 20% of African-American adolescent males versus 29% of African-American females being classified as obese (CDC, 2013). The current study was designed to explore these disparities in greater depth, as it was comprised entirely of African-American subjects, in which male participants represented 35% of the sample, while females represented 65% of the sample. Furthermore, Kumanyika and Grier (2006) reported that low-income minority children appear to have significant barriers that could ultimately prevent them from achieving a healthy lifestyle. These include poor access to healthy food choices in the midst of an oversaturation of fast food restaurants, as well as a general lack of exercise facilities (Kumanyika & Grier, 2006). However, although the current study represents a sample of minority children, adolescents, and their families who are also categorized as low-income, it is important to note that the relationship between minority populations and SES can vary.

The presence of mental health difficulties such as depression and eating disorders may serve as contributors for poor weight outcomes. Depression has been shown to increase the risk for obesity (Duong, 2013), and studies by Eremis et al. (2004) and Pine et al. (2001) suggest that depression in childhood could result in higher BMI measurements into adulthood. Longitudinal

research also indicates that overeating may be associated with various depressive symptoms and may itself be predictive of obesity (Sonneville et al., 2013). With a high rate of comorbidity (up to 51%) noted between major depressive disorder and binge eating disorder in a study by Yanovski, Nelson, Dubber and Spitzer (1993), it is possible that individuals suffering from atypical depression could be particularly sensitive to this connection due to an increase in appetite. Although the existence of one or more of these mental health problems was expected to result in poor weight outcomes, this was not evident within the current analyses. When examining binge eating scores and frequency of binge episodes during a one-week period, 23% of subjects reported clinically significant scores. In addition, only 42% of the subjects that reported clinically significant CDI scores also noted clinically significant binge eating behaviors. It is possible that the lower rate of comorbidity observed between depressive symptomatology and binge-eating behaviors might have led to the lack of significance of mental health factors in relation to weight loss. Additional issues to consider include the possibility of underreported symptoms in self-report measures administered and the inability to discriminate between typical versus atypical depression.

Family conflict and other issues within the family system could also play a role in regards to weight. Within the literature, family comparisons of obese versus normal weight children by Zeller et al. (2007), suggest that mothers with obese children might experience greater levels of distress and family conflict. In addition, a recent systematic review noted a bidirectional relationship between family functioning and obesity, indicating that although we have limited

understanding of this interplay, a link is present (Halliday, Palma, Mellor, Green & Renzaho, 2014). This information was particularly applicable to the current study sample, in which mothers represented nearly 92% of the parental figures that filled out the study questionnaires. However, according to results obtained from the Conflict Behavior Questionnaire (CBQ), child and adolescent self-report scores about maternal conflict did not appear to predict weight outcomes. A lack of significance may be partly due to the low levels of perceived conflict among our sample, as only about 41% reported clinically significant conflict (above the 70th percentile) with mothers. In the evidence provided by Halliday et al. (2014) family conflict was associated with weight outcome in approximately 70% of the reviewed studies. It is once again important to note a potential discrepancy and lack of significance due to a reliance on self-report measures, as children/adolescents in the current study might have had a desire to portray themselves in a more positive light when being evaluated. Differences in parental versus youth self-report problems on the CBCL have been previously observed by Berg-Nielsen, Vika and Dahl (2003) within a sample of mother-adolescent dyads. Their results suggest that agreement regarding externalizing problems is likely to be reached only with older adolescents and with those that report more self-esteem problems (Berg-Nielsen et al., 2003). It is therefore possible that convergence between parents and youth was not reached within this sample, as the mean age of participants was only 12.5 years and observed t-scores for negative self-esteem (a CDI subscale) reached significance for a mere 2% of children/adolescents.

Evidence in the literature also suggests a possible link between intelligence, education, and obesity or weight gain. A study by Halkjaer, Holst, Thorkild and Soresen (2003) noted that intelligence could be related to the onset of obesity, but this relationship is likely mediated by education level. Similar results were discovered by Chandola, Deary, Blane and Batty (2006), in their longitudinal examination of childhood IQ in relation to obesity and weight gain into adulthood. In the present study, KBIT score was used as a potential predictor of weight outcomes, but was not found to be significant. However, with education being a mediator in the relationship between obesity and intelligence, perhaps a reassessment of this variable in adulthood may yield different results for these same subjects. Although it is possible that a lower intellectual quotient could have a negative effect on the comprehension of various interventions and performance of certain study-related tasks, this effect was not likely within the current sample. For the present study, the average scores noted on the KBIT fell within the average range, indicating that no severe impairments were present among participants.

Lastly, poor weight management habits were also thought to play an important role in predicting obesity in the current study. These can include poor eating habits (but not binge eating) and high levels of sedentary behavior, as indicated by the amounts of exercise or physical activity performed. Within the literature, successful weight loss and maintenance has been generally shown to require various modifications in food intake content, or amount consumed (NWCR, 2013; Grief & Miranda, 2010). For children, strategies of decreasing sedentary lifestyles include reductions in screen time exposure and increases in activity levels to at least 60 minutes per day

of moderate to vigorous aerobic, as well as muscle and bone strengthening exercises (AAP, 2012; CDC, 2013). In addition, the development of stimulus control methods and other psychological intervention such as problem solving strategies or cognitive restructuring may increase self-efficacy and decrease various treatment barriers (Berkel, Poston, Reeves, & Foreyt, 2005; Annesi, 2011). However, in the current study, baseline weight control habits, exercise scores, and self-efficacy indicators were not shown to be predictive of weight outcomes, despite an approximate 71% of subjects reporting infrequent use of weight control habits, and an average self-efficacy percentile of 34.5%. It might be possible that the high amount of self-reported calories expended on exercise, in conjunction with the large standard deviations observed in both self-efficacy and caloric expenditure, attenuated some of the above noted factors. With weight gain resulting out of an imbalance between energy intake and expenditure, even a potential increase in exercise could prevent additional weight gains in some individuals (ACSM, 2009). Prior to the commencement of the study, baseline reports for participants showed a daily average of 592 calories expended through exercise over the last 7 days. While at first glance this appears to be a positive indicator, it may not have been enough to offset the large standard deviations observed in exercise (approximately 960 calories per day) and the high caloric intake that might have been present. In addition, the low levels of perceived self-efficacy could have further resulted in a lack of motivation to engage in positive weight loss habits. Poor success within weight management programs for obese children is well known, as Moens et al. (2010) suggested that overall, only 50% of obese youth stood to benefit from participation in various treatment interventions. This evidence is based on a review of several long-term follow-up

studies that examined weight trajectories into adulthood for obese children that participated in a weight loss program. Moens et al. (2010) noted that success for these children is ultimately influenced by a multitude of biological, behavioral, and psychosocial factors.

Aim 2: Depressive Symptomatology as Predictors of Weight Outcome

The second hypothesis indicated that higher depressive symptomatology would correspond to higher baseline BMI and would predict a lower degree of weight loss, or would result in a weight gain. Depressive symptoms were assessed using available CDI data from baseline questionnaires completed by each subject. Both CDI total scores and CDI individual subscale scores were hypothesized to act as potential predictors of weight outcome. Using a linear mixed model to examine the continuous relationship of depression on weight over the 12-month intervention, depressive symptomatology was not found to be a significant predictor of weight change. Similar to the results from the first aim, Type-3 tests of fixed effects displayed significance only for the baseline z-BMI and visits (the monthly interval factor) variables.

These results were somewhat surprising and inconsistent with existing literature, which suggests a mutual interplay between obesity and depression (Luppino et al., 2010; Blaine, 2008). Although the direction of the relationship remains debated (unidirectional versus bidirectional), prospective evidence from Roberts and Doung (2013) points to depression posing an increased risk for the development of weight problems, including obesity. Similar findings were noted by Eremis et al. (2004) and Pine et al. (2001), which indicated that depression could be more

common in obese adolescents compared to those of normal weight. The current study aligned with this unidirectional model in the hypotheses that were posed, predicting that the presence of depressive symptomatology would result in decreased weight loss, or even a weight gain for subjects involved in the intervention program.

Upon participation in the study, various baseline measures were administered, including a CDI assessment for depressive symptoms that may have been present within the last two weeks. Generally, the CDI is a tool used by clinicians in conjunction with a clinical interview and collateral information, in order to make more well-rounded and informed diagnostic and treatment decisions. Its interpretation is based on a generated t-score, which accounts for age and gender. Although CDI cutoffs for raw scores can vary, a t-score of 65 (raw score of 19) or above is considered clinically significant and potentially indicative of depression (Kovacs, 1992). In applying these guidelines to the current study, approximately 6% of our sample had t-scores of 65 or above, while a total of 7% reported raw scores of 19 or above. Despite these percentages being slightly higher than previously reported incidence rates of 3.3% to 5.8% for a depressive disorder within US adolescents (Garrison et al., 1997; Lewinsohn, Hops, Roberts, Seely, & Andrews, 1993), they could still potentially underestimate the amount of depressive disorders present in obese children and adolescents. A more recent study by Vila et al. (2004) discovered that up to 58% of obese children seen in an outpatient setting had a psychiatric illness. In addition, nearly 12% of their study sample was found to meet criteria for depression or dysthymia (Villa et al., 2004). It is possible that the small sample size of the current study

contributed to the smaller rates observed, as well as a lack of significance when examining depressive symptomatology as a predictor of weight change.

Another possibility for this difference could be due to the presence of prodromal symptoms of depression, rather than the actuality of a clinically significant depressive disorder. Various studies point to the increased risk of subsequent MDEs after an initial episode of depression, even when remission is achieved (Bulloch, Williams, Lavorato, & Patten, 2013; Mueller et al., 1999). Furthermore, individuals with a history of depression can also possess various subsyndromal symptoms prior to the onset of another episode (Fava et al., 1990; Judd et al., 1998) that could potentially cause impairment. It is for these additional reasons that the current study also examined individual subscales of the CDI, attempting to assess the relationship of various prodromal symptoms in relation to weight change. Despite the lack of significance at the symptom level, this could be an area of importance from a clinical standpoint, as many obese children/adolescents may not warrant a depressive disorder diagnosis, or may underreport their level of difficulty or impairment.

Aim 3: Adherence and Depressive Symptomatology as Predictors of Weight Outcome

The third hypothesis indicated that psychosocial factors, self-monitoring, and depressive symptomatology would predict attrition. In addition, greater program adherence would result in a higher degree of weight loss, while poor adherence would predict a lower degree or weight loss, or a weight gain. Initially, various Cox Regression models were conducted to evaluate the impact

of psychosocial, self-monitoring, and depressive symptomatology variables as predictors of attrition. It is however, important to recall that age, baseline z-BMI, and gender, were predictors used in each model. This essentially meant that they served as covariates when used in conjunction with other predictor variables. Overall results from the survival analyses indicated that gender might be an important factor to consider. Regardless of the combination of predictor factors used in each model, males had a 49% to 53% greater likelihood of dropping out of the study (up to 1 year) in comparison to female subjects. This result might be partially related to a greater amount of pressure that is being placed on young girls to lose weight, due to the various stereotypes associated with body image that often portray women who are overweight or obese in a more negative light than men.

Subsequently, when using a linear mixed model to examine the continuous relationship of adherence factors and weight, while also accounting for potential depressive symptomatology during the 12-month intervention, type-3 tests of fixed effects were significant only for baseline z-BMI. The lack of significance for overall adherence was also surprising, as previous evidence indicates that self-monitoring can result in greater amounts of weight lost and an increase in adherence. (Acharya et al., 2009; Baker & Kirschenbaum, 1993; Boutelle & Kirschenbaum, 1998). The concept behind this strategy is to increase an individual's awareness regarding current intake and exercise habits, in order to discover the lifestyle factors and behaviors that perpetuate obesity. Additional studies focused on the simultaneous treatment of obesity and depression through CBT modalities, which included the use of self-monitoring principles,

discovered that participants not only lost a significant amount of weight, but also reported a significant decrease in depressive symptoms (Faulconbridge, et al., 2011; Faulconbrige et al, 2012). When examining obese minority youth within a long-term weight intervention program, Germann et al. (2006) noted that more successful subjects engaged in more frequent self-monitoring and attended approximately 50% more sessions. In addition, Germann et al. (2007) further indicated that the frequency of parental self-monitoring was also important in the overall treatment success, as the children whose parents engaged in self-monitoring also lost more weight. This suggests that the modeling of healthy habits and the provision of ongoing support by parents/guardians of obese children and adolescents may be critical in their achievement of successful weight loss.

Within the present sample, the average self-monitoring of food for subjects over 3 months was approximately 3.2 days out of the week, while at 12 months, this decreased to an average of 2.6 days per week. Given these statistics, a lack of significance may be due to the low engagement of these behaviors. For subjects involved in the study, the low engagement in self-monitoring might have been related to a discrepancy between their actual motivation to change and perceived readiness to change. Even though most of the participants that reported their readiness to participate in the weight intervention program also noted a high desire to change, it is possible that within the Transtheoretical Model framework (Prochaska & DiClemente, 1982), these subjects actually remained in the contemplative or pre-contemplative stage of change. However, these results also contrast prior analyses, which indicated that individuals with a medium or high

level of readiness to participate in a weight intervention program lost more weight than those endorsing a low readiness (Pop, Germann, & Littlejohn, 2012).

Exploratory Analyses

Various exploratory analyses were conducted to further analyze the observed changes in marginal means for z-BMI during each monthly interval, and to evaluate whether or not a shorter duration in the program (after three months) would yield any differences in the results already reported. Linear regression analyses were performed for each study aim, as both weight at three months (z-BMI) and average weight after three months were individually used as the outcome variable. While baseline z-BMI was consistently observed to be a significant predictor, when examining the impact of adherence and depressive symptomatology (aim 3) on weight at three months, average food monitoring was also a significant predictor of weight change. This potentially supports that idea that adherence may be of increased importance at an earlier stage of the program, due to higher self-monitoring predicting lower weight at three months. When a mixed linear repeated measures model was initially used to evaluate self-monitoring as a predictor over twelve months, no significance was observed. The difference in results may be due to the presence of certain plateau effects that may occur over time, which could ultimately attenuate the weight loss initially incurred. As such, increased self-efficacy and motivation might have also subsequently increased with early and rapid weight loss, resulting in more success over the initial three months. However, perhaps the nature of the program proved to be too rigorous and demanding for some, in which case engagement and adherence to the provided interventions

might have decreased with time, resulting in a lack of significance in weight change over the entire one-year treatment.

A Kaplan-Meier analysis was also conducted to further evaluate individuals that have lost approximately 5% or 10% of their initial weight during the intervention program. Additional logistic regressions were also performed to examine potential predictors of the target weight loss. Generally, a BMI reduction of 5-10% is considered to be clinically significant in decreasing a variety of risk factors associated with obesity (NIH, 1998; Bigornia et al., 2013; Fayh, Lopes, da Silva, Reischak-Oliviera & Friedman, 2013). A recent study conducted by Nicklas et al. (2012) based on NHANES data also suggested that through a combination of interventions based on food reductions, increases in exercise, and prescription weight loss medications, a 10% or greater weight can be achieved within a 12-month period. Within this sample, a combined average (for males and females) of approximately 10.6 months was necessary for a 10% weight loss to occur, while an approximate average of 8.7 months was needed for a 5% weight loss. Although sex differences were noted, these were not significant. This potentially suggests that with adequate support and provision of structured interventions, a significant reduction of health risks can be achieved in less than one year. Although generalization could be limited due to the current study sample (obese, African-American children and adolescents), results point to an increased need for practitioners to focus more on short-term interventions to increase momentum, self-efficacy and motivation of participants.

Strengths, Limitations, and Future Directions

One of the greatest accomplishments of the current study was its ability to capture such a unique dataset, comprised only of obese African-American children and adolescents, and their parents. Previous studies examining interventions for individuals suffering from depression and obesity have been limited, and a scarcity of data is evident particularly for low-income minority youth with these comorbidities. The existence of parent/guardian measures further allowed for the exploration of additional variables, including the impact of family dynamics, and the examination of potential self-report discrepancies between children and parents. In addition, the assessment of individual depressive symptoms also provided information on what weight outcome predictors could be meaningful for individuals suffering from subsyndromal symptoms of depression, who are unlikely to receive conventional depression treatments such as psychotherapy or pharmacotherapy.

However, the current study also has several limitations that could impact the generalizability of the findings and results. Perhaps most significant are the relatively small sample size (N=206) and the restriction to only African-American children and adolescents. Initially, a total number of 456 subjects were enrolled in the study. However, when including only those participants with available baseline z-BMI scores and CDI scores as per study hypotheses, the numbers of subjects was greatly reduced. In addition, when examining the makeup of the race/ethnicity within the total sample, it was apparent that the majority (approximately 86%) of participants were classified as African-American. The remaining subjects were 6% Hispanic, 5% Caucasian and

3% were unknown or missing this value, all of which were too small in number in order to allow for any comparisons to be drawn between different races/ethnicities. For this type of study, a design that would include a control group (RCT), or one that would consist of various groups receiving different types of interventions may be particularly insightful. This would give an opportunity to compare the progress and efficacy of weight management interventions between several subject groups and could ultimately allow for a better understanding of the mechanisms and specific components involved in successful weight outcomes.

This study was also limited in its use of collected measures, as all psychosocial and depressive symptomatology indicators were a reflection of baseline measurements for each subject, and were assessed prior to the provision of any interventions. Although weight evaluations and adherence factors were continuous variables, the ongoing assessment of all hypothesized predictors would have been optimal. This would allow a more detailed evaluation of each variable, with at least a pre and post measurement data point, which would indicate the change that occurred during the treatment program. As such, the predictors for weight outcome are limited, since they were only collected at baseline. Lastly, the tools used to evaluate certain factors (e.g. CDI for depressive symptomatology) are mainly based on self-report measures from each participant. Within our field, the setbacks in using self-report measures are commonly known and the potential for underreporting or over-reporting may be a real concern. This could be particularly relevant for children and adolescents with whom rapport may be difficult to

establish and difficulties interacting with authority figures (e.g. study team members, doctors) are sometimes evident.

Conclusion

The treatment of obesity and management of weight remain difficult to address even when structured intervention programs are implemented. This is particularly evident within low-income and minority children and adolescents, as they appear to be faced with significant barriers towards achieving a healthier lifestyle. Although various hypotheses have been postulated in regards to why African-American and Hispanic youth may have higher rates of obesity in comparison to Caucasian children and adolescents, no single, definitive answer has been provided at this time. Cultural factors may be important to consider when addressing these disparities. Although results from this study are generally inconsistent with literature findings, they suggest that gender and the amount or frequency of self-monitoring (in regards to food intake) may be important predictors for overall weight outcomes. It also appears that momentum and perhaps enthusiasm of participants might have been higher within the early phase of the program, and that a reduction in significant risk factors associated with obesity was achieved in the first 10 months. Although further research in this area is necessary in order to elucidate the effects of mental health comorbidities for obese minority youth, the current study provided a more in depth understanding of potential factors involved in the prediction of weight outcomes for this unique sample of children and adolescents.

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FIGURES

FIGURE 1. *Flowchart of Included Subjects*

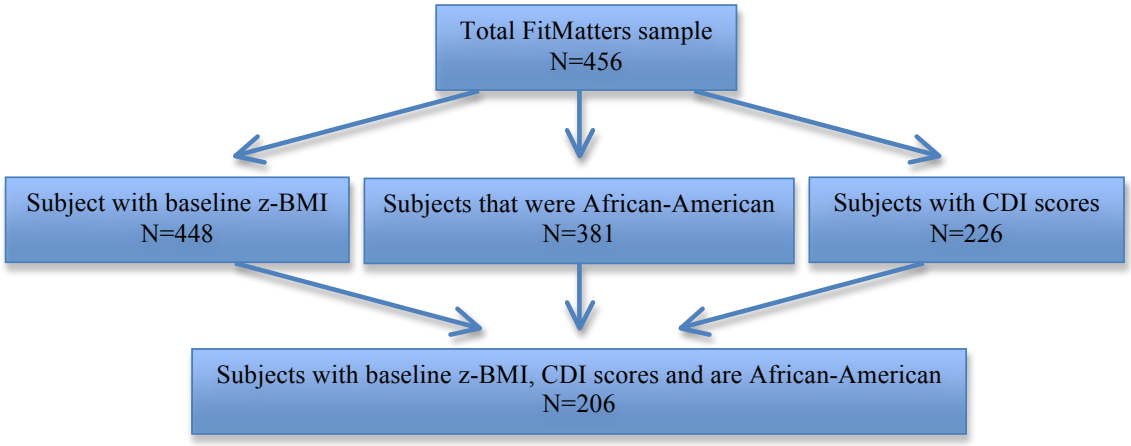


FIGURE 2. *Cox Regression Analysis (3-month) with CDI Total Score*

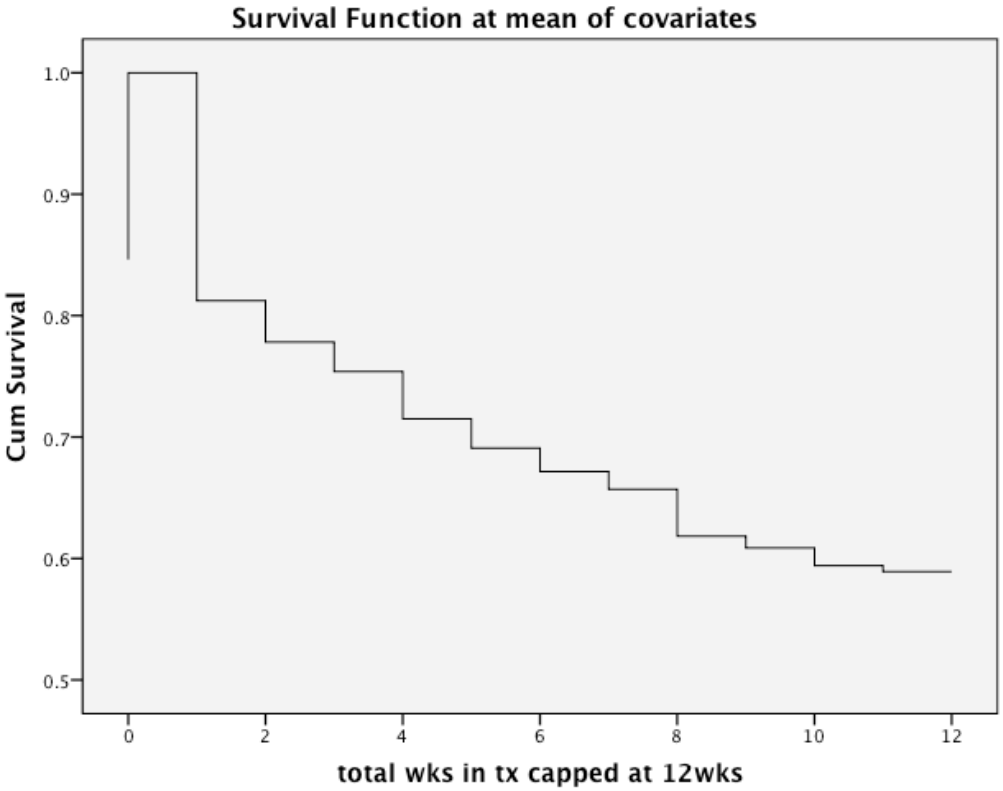


FIGURE 3. *Cox Regression Analysis (3-month) with CDI Total Score by Gender*

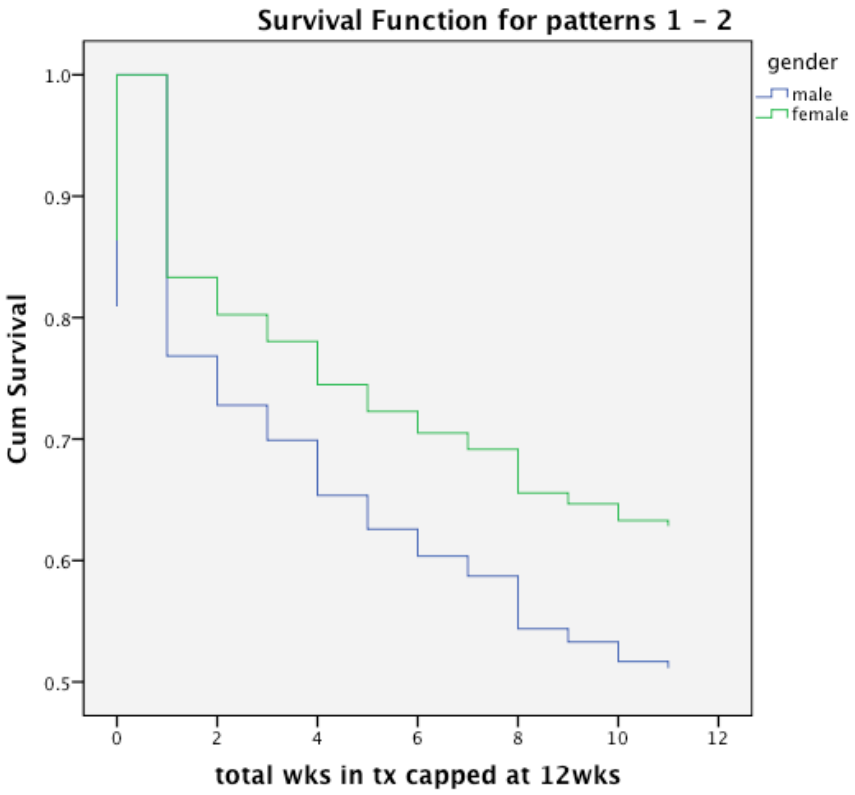


FIGURE 4. *Cox Regression Analysis (3-month) with CDI Individual Subscales*

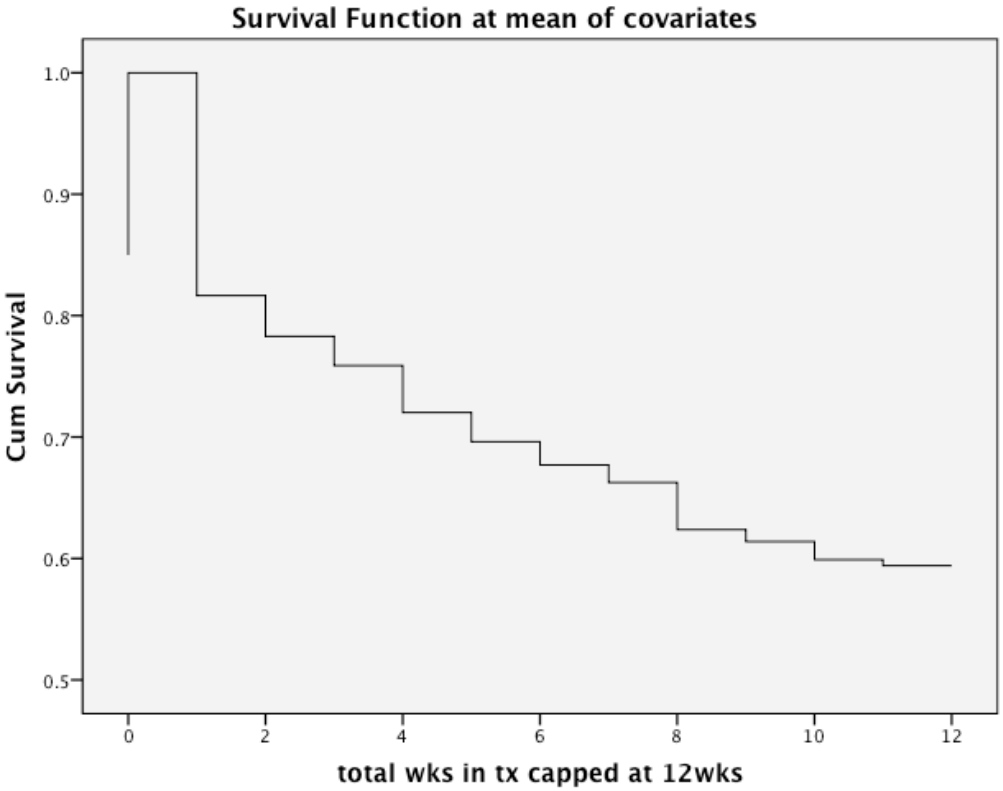


FIGURE 5. *Cox Regression Analysis (3-month) with CDI Individual Subscales by Gender*

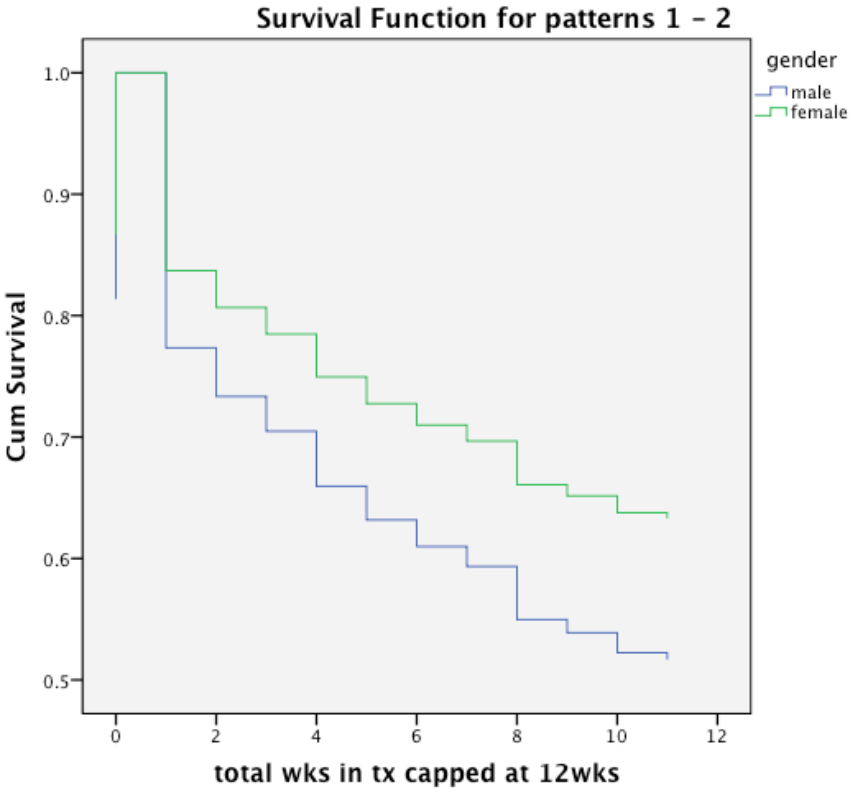


FIGURE 6. *Cox Regression Analysis (3-month) with Psychosocial Variables*

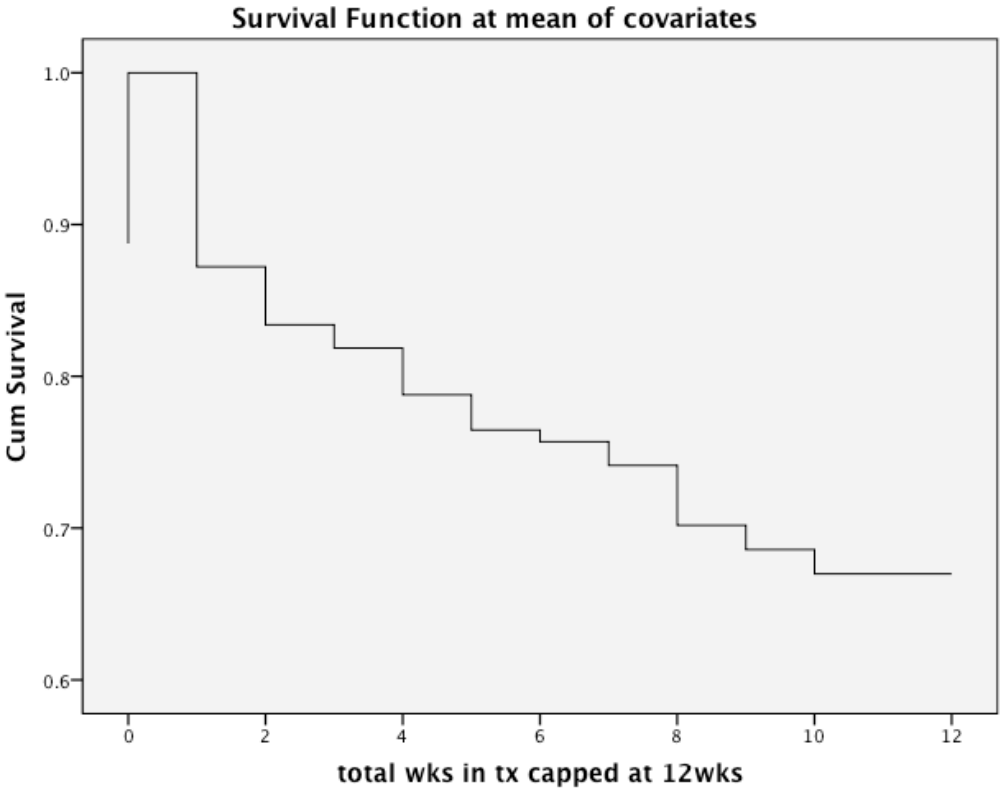


FIGURE 7. *Cox Regression Analysis (3-month) with Psychosocial Variables by Gender*

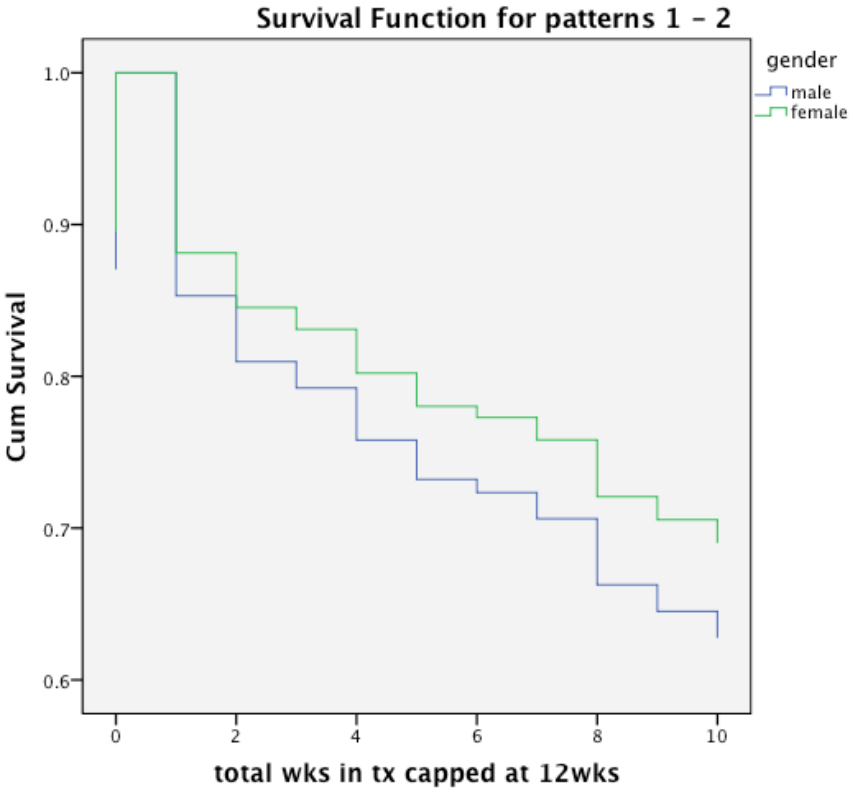


FIGURE 8. *Cox Regression Analysis (3-month) with Self-Monitoring Variables*

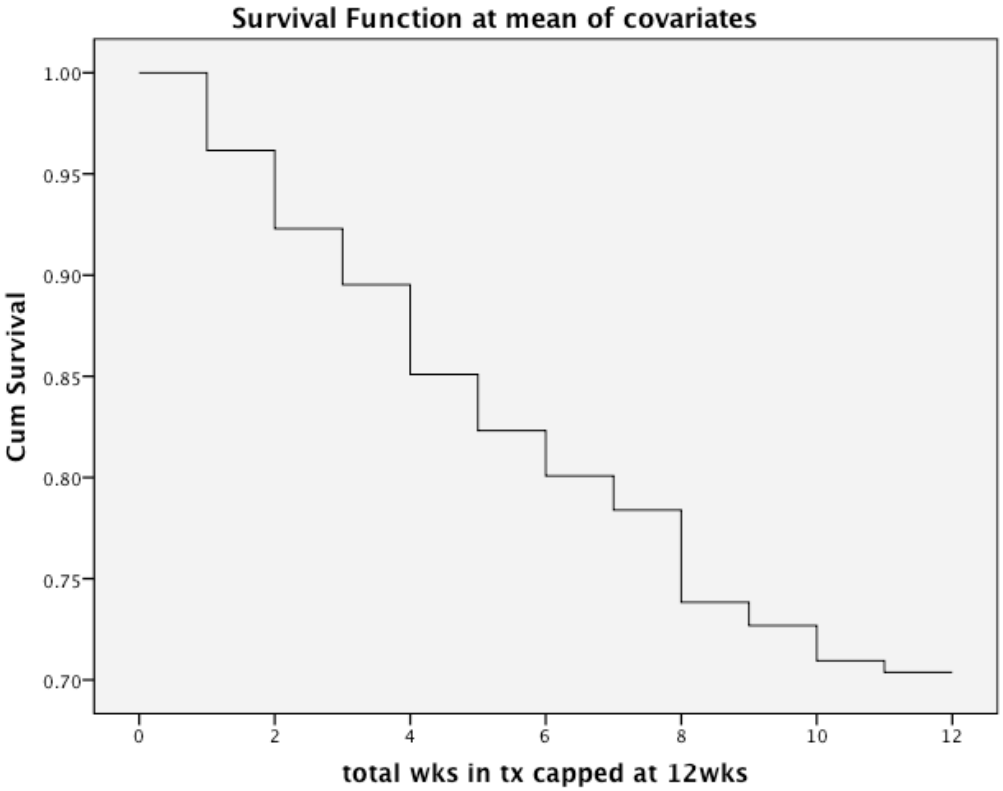


FIGURE 9. *Cox Regression Analysis (3-month) with Self-Monitoring Variables by Gender*

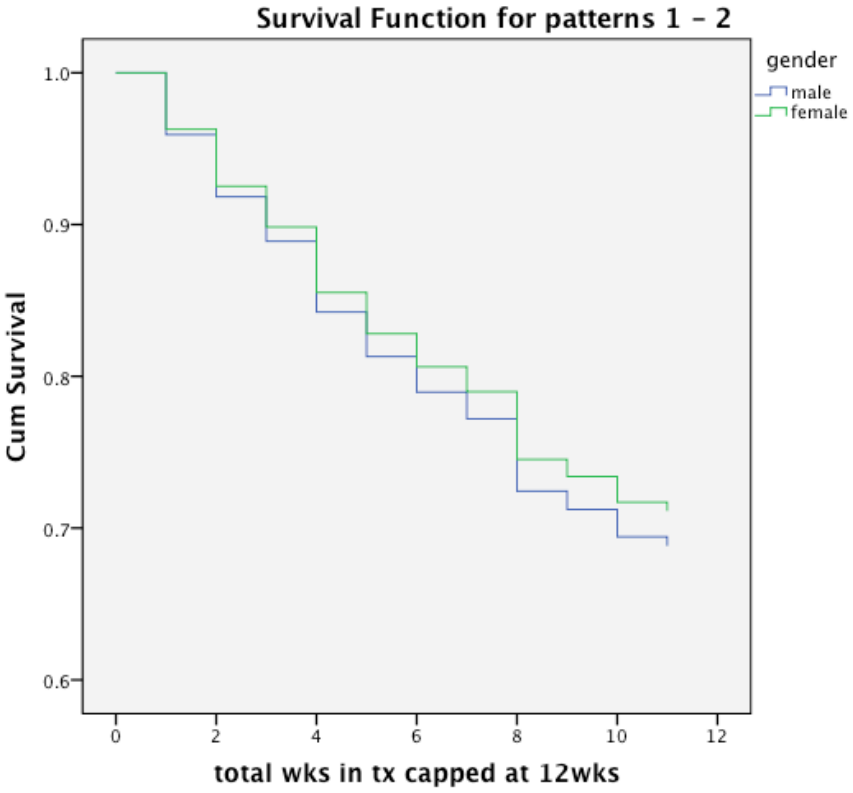


FIGURE 10. *Cox Regression Analysis (12-month) with Psychosocial Variables*

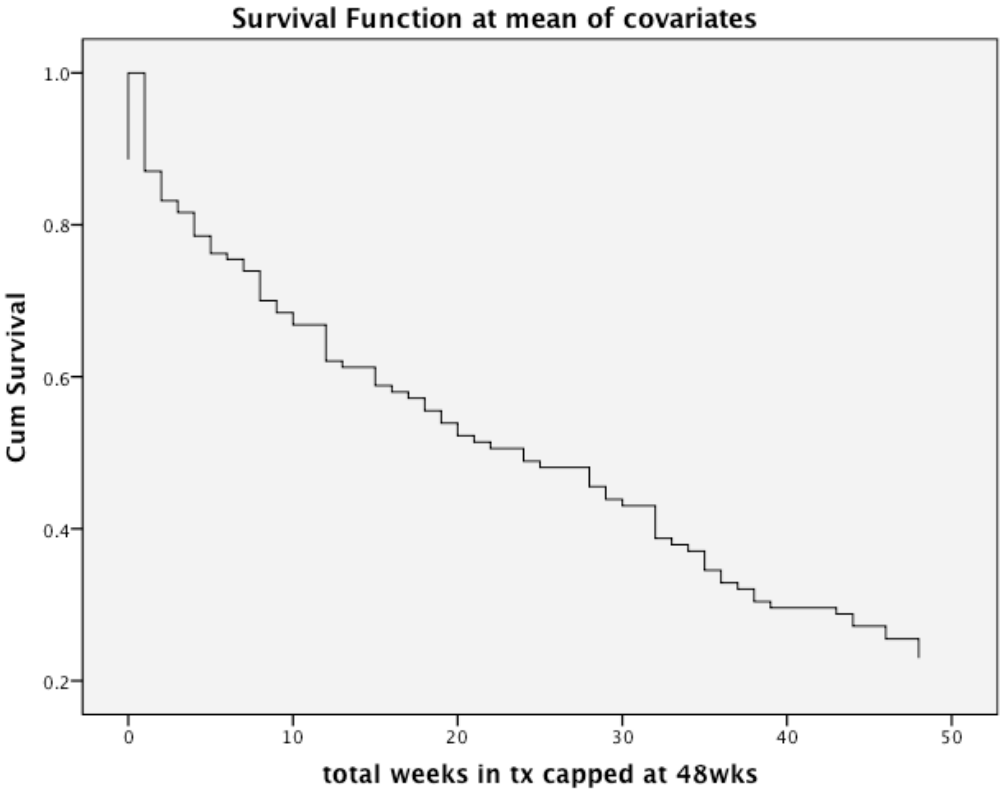


FIGURE 11. *Cox Regression Analysis (12-month) with Psychosocial Variables by Gender*

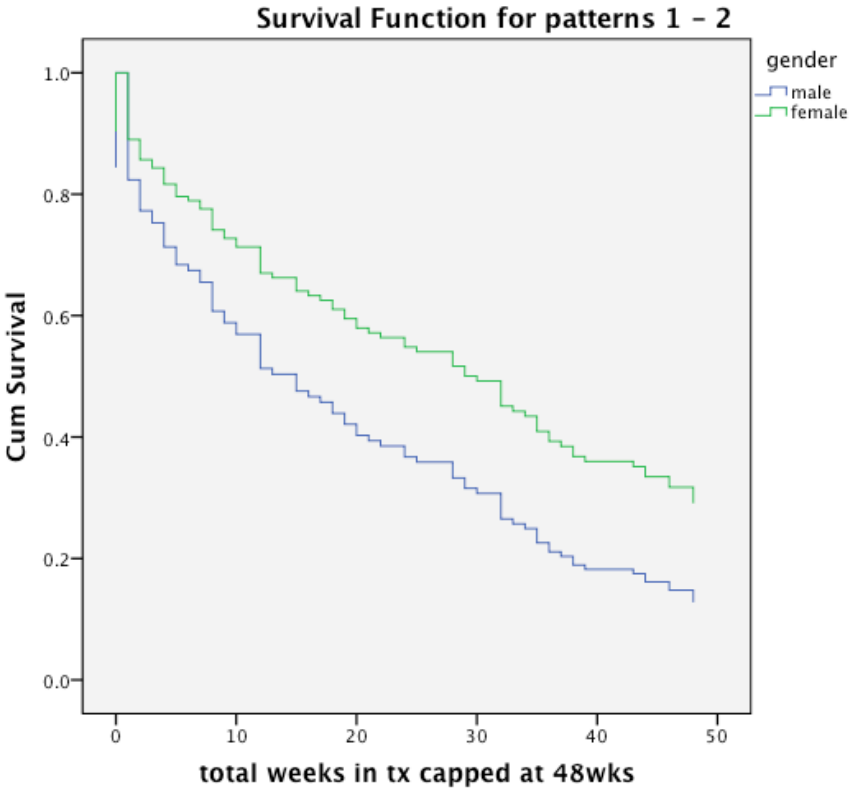


FIGURE 12. *Cox Regression Analysis (12-month) with CDI Total Score*

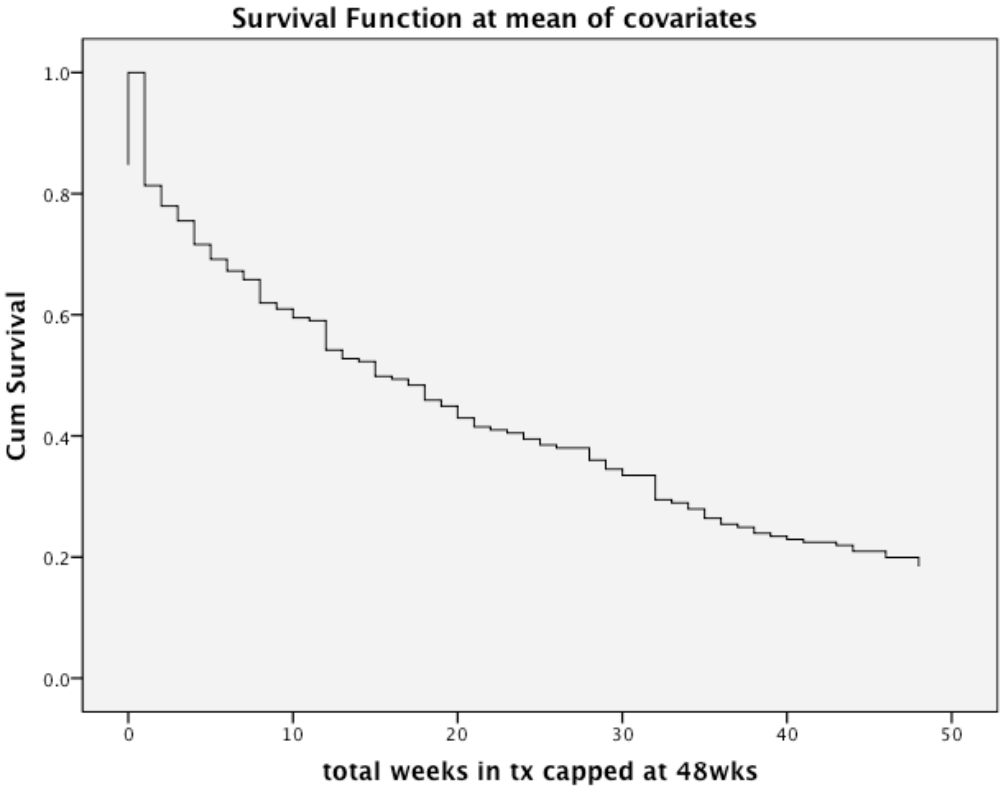


FIGURE 13. *Cox Regression Analysis (12-month) with CDI Total Score by Gender*

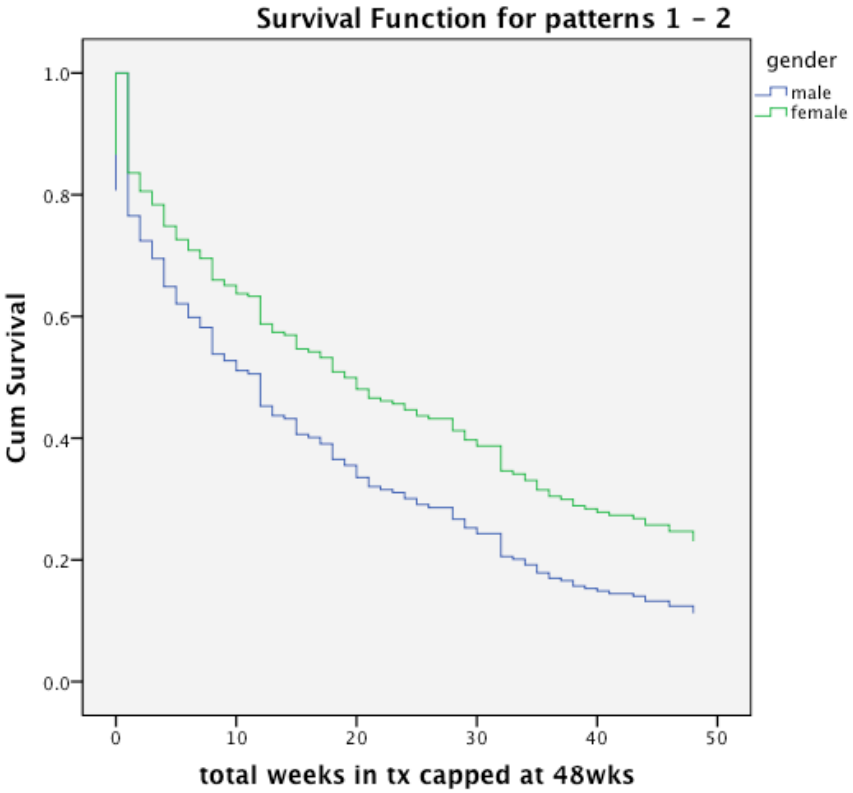


FIGURE 14. *Cox Regression Analysis (12-month) with CDI Individual Subscales*

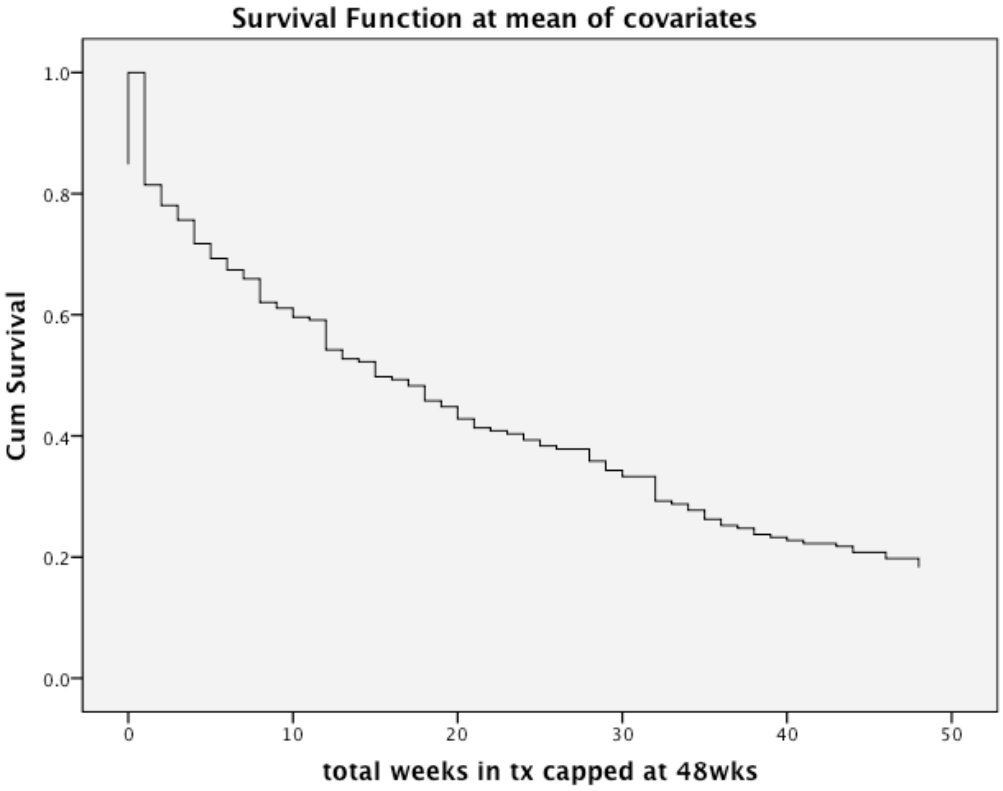


FIGURE 15. *Cox Regression Analysis (12-month) with CDI Individual Subscales by Gender*

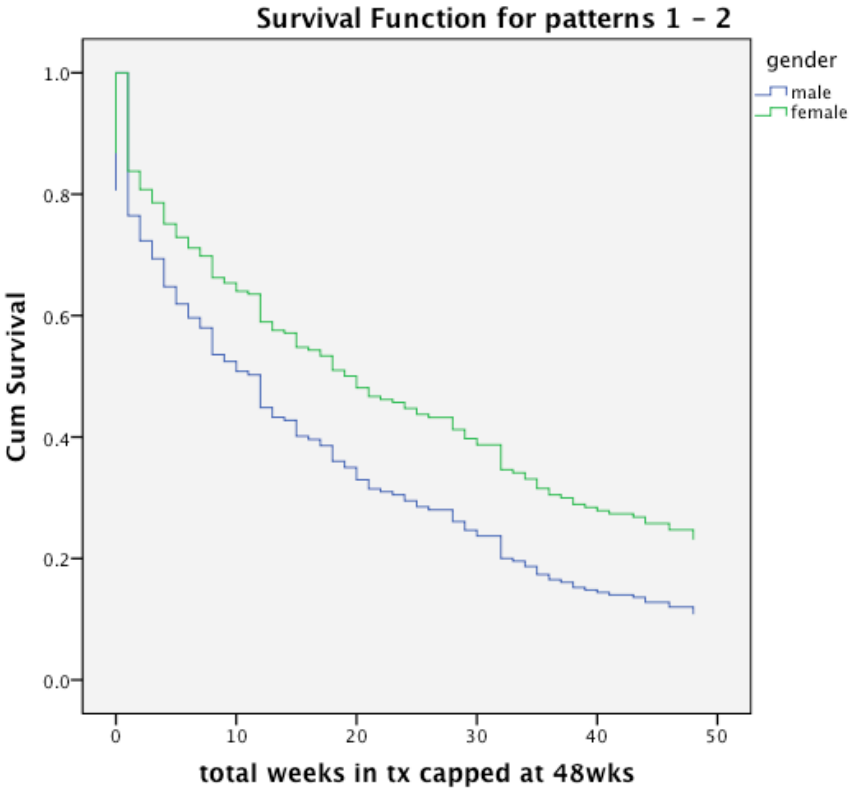


FIGURE 16. *Cox Regression Analysis (12-month) with Self-Monitoring Variables*

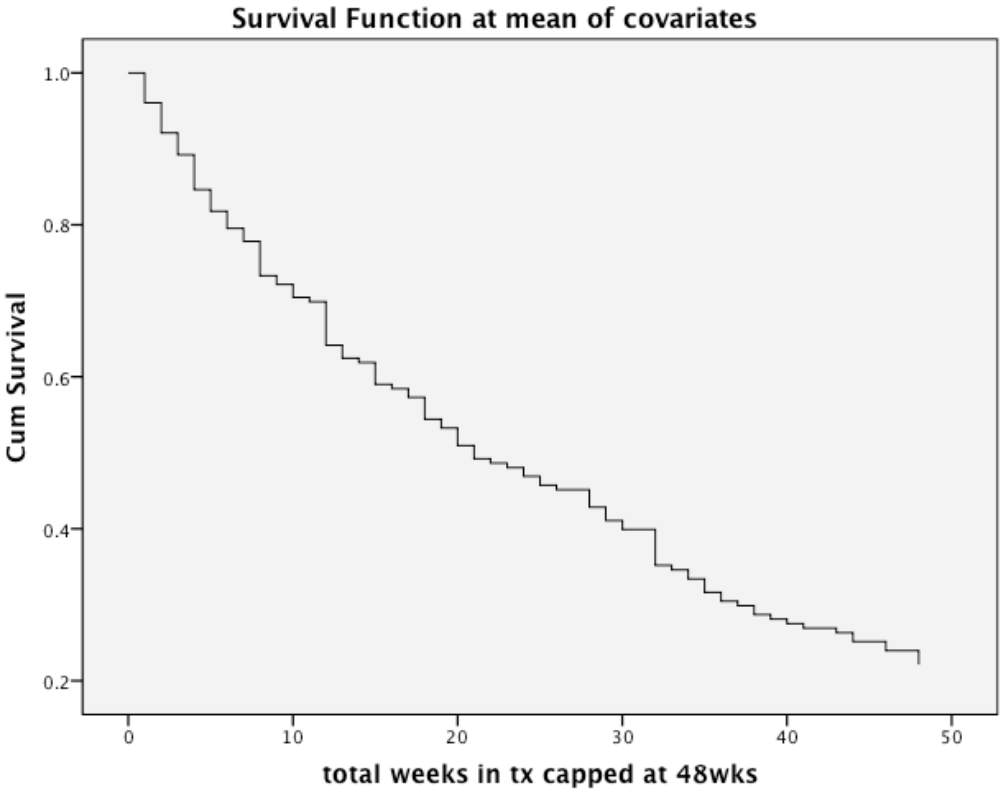


FIGURE 17. *Cox Regression Analysis (12-month) with Self-Monitoring Variables by Gender*

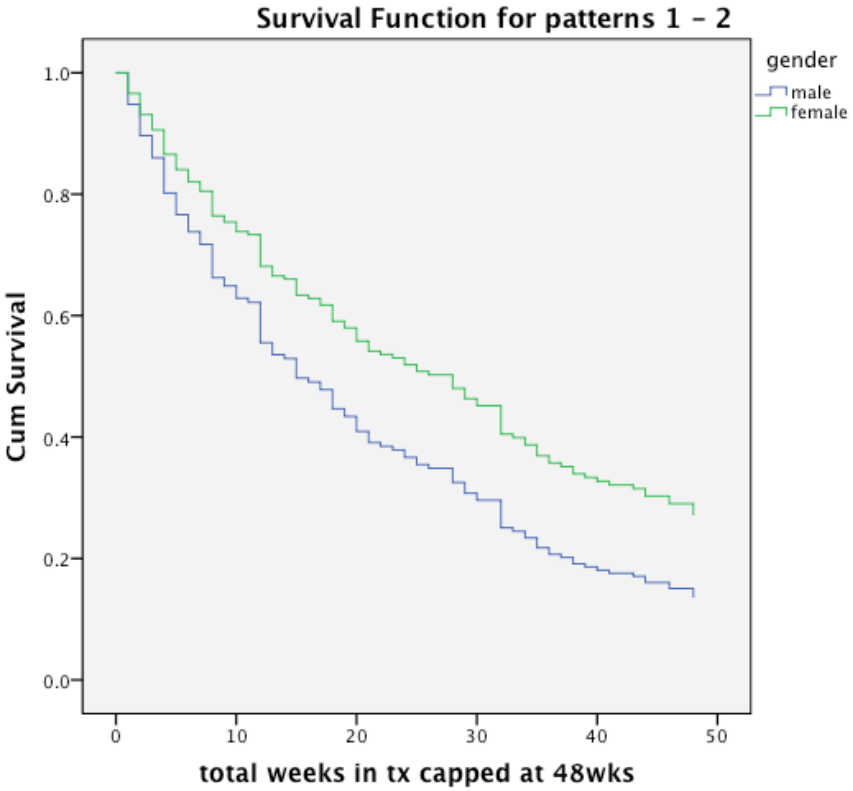


FIGURE 18. *Kaplan Meier Survival Analysis for 10% Weight Loss*

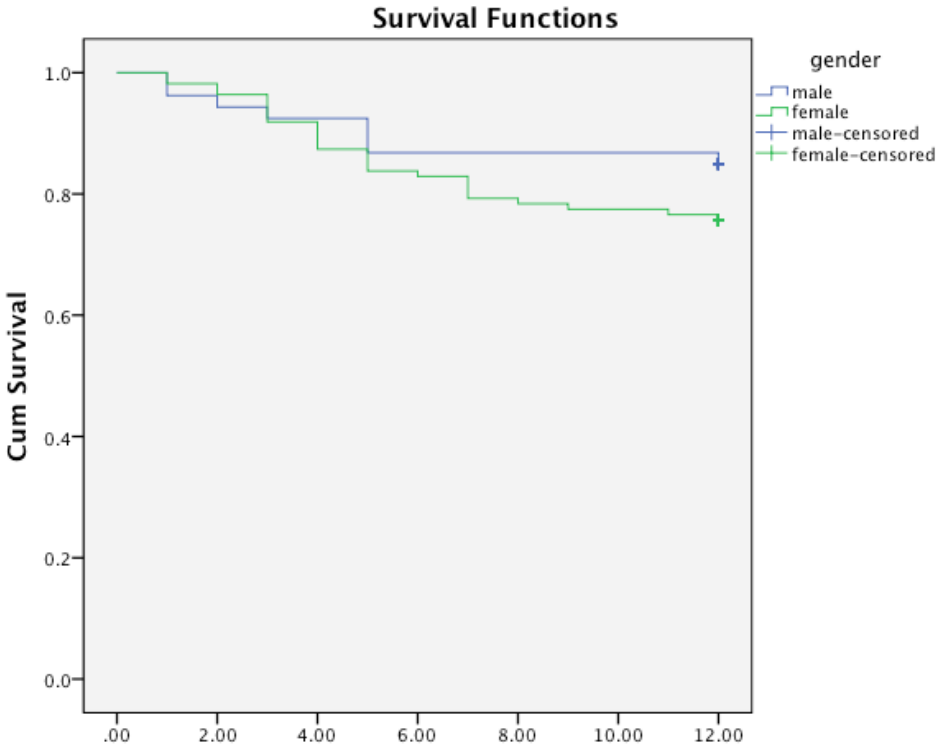
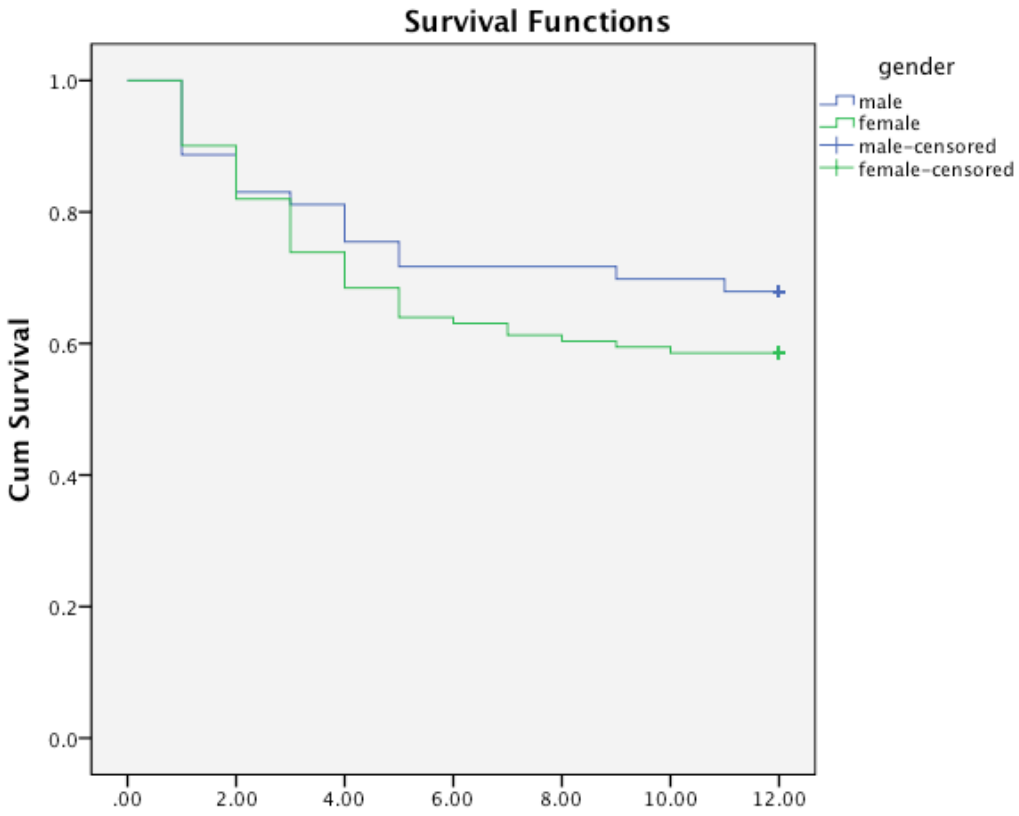


FIGURE 19. *Kaplan Meier Survival Analysis for 5% Weight Loss*



TABLES

TABLE 1. *Characteristics of Sample*

Demographics		Total N=206	N	%	M	SD
Age	7-18 yrs old		206	-	12.5	± 2.5
Sex	Male		73	35%		
	Female		133	65%		
Race/Ethnicity	African-American		206	100%		
BMI	23-108 (range)		206	-	42.3	± 11.9
SES	Hollingshead Scores		171	-	36.2	± 14.7
Parent/Guardian	Relationship to Child		217*	-		
		Father/Adoptive/Grandfather	16	89%		
		Other	2	11%		
		Mother/Adoptive/Grandmother	186	93%		
		Other	13	7%		
	Marital Status - Mothers		189	-		
		Married	62	33%		
		Single/Divorced/Separated	115	61%		
		Widowed	12	6%		
	Marital Status - Fathers		17	-		
		Married	7	41%		
		Single/Divorced	10	59%		
Insurance Status			168	-		
	Medicaid		131	78%		
	HMO/PPO		37	22%		
Psychosocial		Ranges				
	CDI scores	0-28	206	-	9.0	± 5.9
	Perceived conflict with mother		203	-		
		yes	84	41%		
		no	119	59%		
	KBIT composite scores (SS)	M=100, SD=15	160	-	92.7	± 13.5
	Weight control habits scores	11-55	203	-	29.5	± 6.0
	Total self-efficacy percentile	0-100	203	-	34.5	± 31.5
	Total calories expended on exercise (last 7 days)	n/a	206	-	4141.4	± 6717.7
	Total binge eating scores	0-27	201	-	6.1	± 4.6
Adherence	Over 3 months (12 wks)					
		Attendance in weeks	206	-	8.3	±4.9
		Self-monitoring in days/week	174**	84%	3.2	±2.0
	Over 12 months (48 wks)					
		Attendance in weeks	206	-	20.7	±18.2
		Self-monitoring in days/week	174**	84%	2.6	±1.6

* N exceeds 206 total subjects due to certain participants having had both mothers and fathers present at time of assessment

** N at 3 months and 12 months is unchanged due to the averaging out of self-monitoring over the entire treatment period

TABLE 2. *Hypothesis 1 (LMM) – Psychosocial Variables*

Type III Tests of Fixed Effects

Source	Numerator df	Denominator df	F	Sig.
Intercept	1	131.14	0.873	0.352
gender	1	134.411	0.885	0.348
visits	11	543.747	2.567	0.004
age	1	139.325	1.053	0.307
zbmi	1	138.889	3123.846	0.000
cditot	1	133.839	0.467	0.495
clinicalmibq	1	134.461	0.552	0.459
famses	1	132.105	1.886	0.172
habtot	1	136.199	0.298	0.586
seperc	1	135.691	1.717	0.192
bngscore	1	134.355	0.405	0.526
totexer	1	153.254	0.056	0.813

**Dependent Variable: zbmi over time (1yr total)*

TABLE 3. Hypothesis (LMM) 1 – *Estimated Marginal Means*

Estimated Marginal Means

all 12 months	Mean	Std. Error	df	95% Confidence Interval	
				Lower Bound	Upper Bound
zbmi1m	5.797b	0.057	211.892	5.684	5.909
zbmi2m	5.792b	0.058	223.232	5.678	5.906
zbmi3m	5.788b	0.059	243.296	5.67	5.905
zbmi4m	5.765b	0.062	269.759	5.643	5.886
zbmi5m	5.711b	0.064	294.575	5.585	5.838
zbmi6m	5.714b	0.067	318.701	5.583	5.846
zbmi7m	5.654b	0.069	335.525	5.518	5.791
zbmi8m	5.690b	0.071	341.183	5.549	5.83
zbmi9m	5.601b	0.076	381.122	5.452	5.75
zbmi10m	5.619b	0.081	429.284	5.459	5.779
zbmi11m	5.567b	0.08	494.013	5.41	5.724
zbmi12m	5.537b	0.075	496.072	5.389	5.685

a. Dependent Variable: zbmi over time (1yr total)

b. Covariates appearing in the model are evaluated at the following values: age = 12.48, z BMI assessment = 5.8287, CDI Total Score = 9.48, Clinical levels of perceived conflict with mother (percentile>=70) = .48, family ses = 38.38, Weight Control Habits Total = 29.11, Total self-efficacy percentile = 34.69, total binge eating score = 6.33, total exercise = 3141.83

TABLE 4. *Hypothesis 2 – Correlations*

Correlations		z-BMI assessment	CDI Total Score
z-BMI assessment	Pearson Correlation	1	0.076
	Sig. (1-tailed)	-	0.139
	N	206	206
CDI Total Score	Pearson Correlation	0.076	1
	Sig. (1-tailed)	0.139	-
	N	206	206

Correlations			z-BMI assessment	CDI Total Score
Spearman's rho	z-BMI assessment	Correlation Coefficient	1	.142*
		Sig. (1-tailed)	.	0.021
		N	206	206
	CDI Total Score	Correlation Coefficient	.142*	1
		Sig. (1-tailed)	0.021	.
		N	206	206

* Correlation is significant at the 0.05 level (1-tailed).

TABLE 5. *Hypothesis 2 (LMM) – CDI Total Score*

Type III Tests of Fixed Effects

Source	Numerator df	Denominator df	F	Sig.
Intercept	1	177.177	3.09	0.081
gender	1	175.381	0.171	0.679
visits	11	677.459	1.809	0.049
age	1	177.356	1.205	0.274
zbmi	1	179.954	4145.213	0.000
cditot	1	170.328	0.313	0.576

** Dependent Variable: zbmi over time (1yr total)*

TABLE 6. *Hypothesis 2 (LMM) – Estimated Marginal Means*

Estimated Marginal Means

all 12 months	Mean	Std. Error	df	95% Confidence Interval	
				Lower Bound	Upper Bound
zbmi1m	5.622b	0.049	281.137	5.527	5.718
zbmi2m	5.625b	0.05	299.883	5.527	5.722
zbmi3m	5.618b	0.051	327.214	5.517	5.718
zbmi4m	5.585b	0.053	361.398	5.48	5.689
zbmi5m	5.554b	0.055	390.302	5.446	5.663
zbmi6m	5.543b	0.058	424.453	5.429	5.657
zbmi7m	5.493b	0.06	446.339	5.374	5.611
zbmi8m	5.535b	0.062	457.218	5.412	5.658
zbmi9m	5.477b	0.066	498.957	5.347	5.608
zbmi10m	5.496b	0.07	554.503	5.358	5.634
zbmi11m	5.448b	0.069	642.908	5.312	5.584
zbmi12m	5.449b	0.065	647.028	5.322	5.575

a. Dependent Variable: zbmi over time (1yr total)

b. Covariates appearing in the model are evaluated at the following values: age = 12.36, z BMI assessment = 5.6525, CDI Total Score = 9.21

TABLE 7. *Hypothesis 2 (LMM) – CDI Individual Subscales*

Type III Tests of Fixed Effects

Source	Numerator df	Denominator df	F	Sig.
Intercept	1	172.865	3.626	0.059
gender	1	171.241	0.058	0.810
visits	11	674.999	1.799	0.051
age	1	172.494	1.65	0.201
zbmi	1	176.269	4013.422	0.000
negmtot	1	177.058	0.316	0.575
iprobtot	1	160.341	0.381	0.538
ieffetot	1	164.594	1.812	0.180
anhedtot	1	165.22	0.001	0.970
negsetot	1	172.406	0.499	0.481

** Dependent Variable: zbmi over time (1yr total)*

TABLE 8. *Hypothesis 2 (LMM) – Estimated Marginal Means*

Estimated Marginal Means

all 12 months	Mean	Std. Error	df	95% Confidence Interval	
				Lower Bound	Upper Bound
zbmi1m	5.617b	0.049	272.395	5.521	5.714
zbmi2m	5.620b	0.05	289.641	5.522	5.718
zbmi3m	5.614b	0.051	315.879	5.513	5.715
zbmi4m	5.580b	0.053	349.315	5.475	5.685
zbmi5m	5.550b	0.055	378.133	5.441	5.659
zbmi6m	5.538b	0.058	412.219	5.424	5.653
zbmi7m	5.488b	0.061	434.507	5.369	5.607
zbmi8m	5.531b	0.063	445.987	5.408	5.654
zbmi9m	5.473b	0.067	487.618	5.342	5.604
zbmi10m	5.492b	0.071	542.82	5.353	5.631
zbmi11m	5.444b	0.069	627.055	5.308	5.58
zbmi12m	5.444b	0.065	627.331	5.317	5.571

a. Dependent Variable: zbmi over time (1yr total)

b. Covariates appearing in the model are evaluated at the following values: age = 12.36, z BMI assessment = 5.6525, CDI Negative Mood, Scale A Total = 2.01, CDI Interpersonal Problems, Scale B Total = .79, CDI Ineffectiveness, Scale C Total = 1.56, CDI Anhedonia, Scale D Total = 3.63, CDI Negative Self-Esteem, Scale E Total = 1.23

TABLE 9. *Hypothesis 2 (LMM) – Negative Mood Subscale – A*

Type III Tests of Fixed Effects

Source	Numerator df	Denominator df	F	Sig.
Intercept	1	177.578	3.315	0.070
gender	1	176.659	0.096	0.757
visits	11	677.356	1.803	0.050
age	1	175.89	1.019	0.314
zbmi	1	180.367	4149.903	0.000
negmtot	1	184.159	0.035	0.851

** Dependent Variable: zbmi over time (1yr total)*

TABLE 10. *Hypothesis 2 (LMM) – Interpersonal Problems Subscale – B*

Type III Tests of Fixed Effects

Source	Numerator df	Denominator df	F	Sig.
Intercept	1	178.648	3.264	0.073
gender	1	174.603	0.106	0.745
visits	11	676.971	1.797	0.051
age	1	176.028	0.969	0.326
zbmi	1	180.42	4165.894	0.000
iprobtot	1	162.739	0.07	0.792

** Dependent Variable: zbmi over time (1yr total)*

TABLE 11. *Hypothesis 2 (LMM) – Ineffectiveness Subscale – C*

Type III Tests of Fixed Effects

Source	Numerator df	Denominator df	F	Sig.
Intercept	1	178.905	3.659	0.057
gender	1	173.593	0.128	0.721
visits	11	677.541	1.817	0.048
age	1	177.114	1.708	0.193
zbmi	1	179.728	4183.152	0.000
ieffetot	1	167.683	2.068	0.152

** Dependent Variable: zbmi over time (1yr total)*

TABLE 12. *Hypothesis 2 (LMM) – Anhedonia Subscale – D*

Type III Tests of Fixed Effects

Source	Numerator df	Denominator df	F	Sig.
Intercept	1	176.316	2.978	0.086
gender	1	174.537	0.135	0.714
visits	11	677.373	1.805	0.050
age	1	175.809	1.077	0.301
zbmi	1	180.142	4198.754	0.000
anhedtot	1	171.029	0.079	0.780

** Dependent Variable: zbmi over time (1yr total)*

TABLE 13. *Hypothesis 2 (LMM) – Negative Self-Esteem Subscale – E*

Type III Tests of Fixed Effects

Source	Numerator df	Denominator df	F	Sig.
Intercept	1	178.654	3.342	0.069
gender	1	174.34	0.179	0.672
visits	11	677.637	1.808	0.049
age	1	176.073	1.266	0.262
zbmi	1	180.458	4121.163	0.000
negsetot	1	173.025	0.654	0.420

** Dependent Variable: zbmi over time (1yr total)*

TABLE 14. *Hypothesis 3 (Cox Regression – 3 month) – CDI Total Score*

Variables in the Equation

	B	SE	Wald	df	Sig.	Exp(B)	95.0% CI for Exp(B)	
							Lower	Upper
zbmi	-0.019	0.038	0.254	1	0.614	0.981	0.91	1.057
age	0.02	0.046	0.181	1	0.67	1.02	0.932	1.117
gender	0.367	0.236	2.419	1	0.12	1.444	0.909	2.293
cditot	0.008	0.019	0.192	1	0.661	1.008	0.972	1.046

TABLE 15. *Hypothesis 3 (Cox Regression – 3 month) – CDI Individual Subscales*

Variables in the Equation

	B	SE	Wald	df	Sig.	Exp(B)	95.0% CI for Exp(B)	
							Lower	Upper
zbmi	-0.011	0.038	0.082	1	0.774	0.989	0.919	1.065
age	0.033	0.047	0.485	1	0.486	1.033	0.942	1.133
gender	0.367	0.238	2.39	1	0.122	1.444	0.906	2.3
iprobtot	-0.218	0.143	2.323	1	0.127	0.804	0.608	1.064
ieffetot	0.026	0.075	0.123	1	0.725	1.027	0.886	1.189
negsetot	-0.073	0.098	0.566	1	0.452	0.929	0.767	1.125
negmtot	0.092	0.074	1.542	1	0.214	1.097	0.948	1.268
anhedtot	0.024	0.053	0.214	1	0.644	1.025	0.924	1.136

TABLE 16. *Hypothesis 3 (Cox Regression – 3 month) – Psychosocial Variables*

Variables in the Equation

	B	SE	Wald	df	Sig.	Exp(B)	95.0% CI for Exp(B)	
							Lower	Upper
zbmi	-0.043	0.066	0.414	1	0.52	0.958	0.841	1.091
age	-0.071	0.078	0.827	1	0.363	0.932	0.8	1.085
gender	0.228	0.361	0.4	1	0.527	1.257	0.619	2.549
kbitcomp	-0.002	0.013	0.03	1	0.861	0.998	0.974	1.023
cditot	0.003	0.03	0.012	1	0.913	1.003	0.946	1.064
clinicalmibq	-0.156	0.346	0.204	1	0.652	0.855	0.434	1.686
famses	-0.017	0.012	2.152	1	0.142	0.983	0.961	1.006
habtot	0.021	0.031	0.444	1	0.505	1.021	0.961	1.084
seperc	0.002	0.007	0.132	1	0.716	1.002	0.989	1.016
bngscore	0.019	0.044	0.184	1	0.668	1.019	0.935	1.111

TABLE 17. *Hypothesis 3 (Cox Regression – 3 month) – Self-Monitoring Variable*

Variables in the Equation

	B	SE	Wald	df	Sig.	Exp(B)	95.0% CI for Exp(B)	
							Lower	Upper
zbmi	0.02	0.046	0.181	1	0.67	1.02	0.932	1.116
age	-0.024	0.06	0.166	1	0.684	0.976	0.868	1.098
gender	0.093	0.315	0.087	1	0.769	1.097	0.592	2.035
totwklyavgfd12wks	-0.149	0.079	3.546	1	0.06	0.862	0.738	1.006

TABLE 18. *Hypothesis 3 (Cox Regression – 12 month) – Psychosocial Variables*

Variables in the Equation

	B	SE	Wald	df	Sig.	Exp(B)	95.0% CI for Exp(B)	
							Lower	Upper
zbmi	-0.072	0.044	2.662	1	0.103	0.93	0.853	1.015
age	-0.016	0.053	0.093	1	0.761	0.984	0.887	1.092
gender	0.511	0.246	4.324	1	0.038	1.666	1.03	2.696
kbitcomp	-0.005	0.009	0.38	1	0.537	0.995	0.978	1.012
cditot	0	0.022	0	1	0.999	1	0.959	1.043
clinicalmibq	-0.21	0.225	0.872	1	0.35	0.81	0.521	1.26
famses	-0.007	0.008	0.726	1	0.394	0.993	0.978	1.009
habtot	0.024	0.02	1.355	1	0.244	1.024	0.984	1.065
seperc	0	0.005	0.006	1	0.939	1	0.991	1.009
bngscore	0.001	0.031	0.002	1	0.964	1.001	0.942	1.065

TABLE 19. *Hypothesis 3 (Cox Regression – 12 month) – CDI Total Score*

Variables in the Equation

	B	SE	Wald	df	Sig.	Exp(B)	95.0% CI for Exp(B)	
							Lower	Upper
zbmi	-0.018	0.029	0.395	1	0.53	0.982	0.929	1.039
age	0.035	0.034	1.031	1	0.31	1.035	0.968	1.107
gender	0.399	0.171	5.45	1	0.02	1.491	1.066	2.085
cditot	-0.008	0.014	0.327	1	0.567	0.992	0.966	1.019

TABLE 20. *Hypothesis 3 (Cox Regression – 12 month) – CDI Individual Subscales*

Variables in the Equation

	B	SE	Wald	df	Sig.	Exp(B)	95.0% CI for Exp(B)	
							Lower	Upper
zbmi	-0.016	0.029	0.29	1	0.59	0.984	0.93	1.042
age	0.04	0.035	1.338	1	0.247	1.041	0.973	1.114
gender	0.417	0.173	5.821	1	0.016	1.517	1.081	2.129
iprobtot	-0.142	0.096	2.194	1	0.139	0.867	0.718	1.047
ieffetot	-0.007	0.054	0.016	1	0.899	0.993	0.893	1.104
negsetot	-0.012	0.071	0.029	1	0.866	0.988	0.859	1.136
negmtot	0.053	0.057	0.866	1	0.352	1.055	0.943	1.181
anhedtot	-0.013	0.038	0.113	1	0.737	0.987	0.916	1.064

TABLE 21. *Hypothesis 3 (Cox Regression – 12 month) – Self-Monitoring Variable*

Variables in the Equation

	B	SE	Wald	df	Sig.	Exp(B)	95.0% CI for Exp(B)	
							Lower	Upper
zbmi	-0.009	0.031	0.077	1	0.782	0.991	0.933	1.054
age	0.025	0.037	0.456	1	0.499	1.026	0.953	1.104
gender	0.426	0.19	5.027	1	0.025	1.531	1.055	2.222
totwklyavgfd48wks	0.081	0.065	1.574	1	0.21	1.085	0.955	1.231

TABLE 22. *Hypothesis 3 (LMM) – Adherence and Depressive Symptomatology*

Type III Tests of Fixed Effects

Source	Numerator df	Denominator df	F	Sig.
Intercept	1	190.3	3.171	0.077
gender	1	172.537	0.118	0.732
visits	11	659.368	1.54	0.113
age	1	175.923	1.153	0.284
zbmi	1	176.951	4084.868	0.000
cditot	1	168.346	0.408	0.524
totwkstxadj48	1	216.501	1.235	0.268
totwklyavgfd48wks	1	196.391	0.121	0.729

** Dependent Variable: zbmi over time (1yr total)*

TABLE 23. *Hypothesis 3 (LMM) – Estimated Marginal Means*

Estimated Marginal Means

all 12 months	Mean	Std. Error	df	95% Confidence Interval	
				Lower Bound	Upper Bound
zbmi1m	5.592b	0.056	260.168	5.482	5.703
zbmi2m	5.596b	0.056	263.806	5.485	5.707
zbmi3m	5.592b	0.057	271.033	5.48	5.703
zbmi4m	5.562b	0.058	286.13	5.448	5.675
zbmi5m	5.534b	0.059	304.568	5.419	5.649
zbmi6m	5.525b	0.06	333.684	5.406	5.644
zbmi7m	5.477b	0.062	360.913	5.355	5.6
zbmi8m	5.522b	0.064	383.408	5.396	5.647
zbmi9m	5.466b	0.067	437.279	5.333	5.598
zbmi10m	5.483b	0.072	483.311	5.343	5.624
zbmi11m	5.432b	0.071	522.698	5.292	5.571
zbmi12m	5.427b	0.068	494.854	5.294	5.56

a. Dependent Variable: zbmi over time (1yr total)

b. Covariates appearing in the model are evaluated at the following values: age = 12.36, z BMI assessment = 5.6525, CDI Total Score = 9.21, total weeks in tx capped at 48wks = 36.19, total average food monitoring per week, for 48 weeks (12 month), from each fdtot and based on totwkstx = 2.6065

TABLE 24. *Exploratory Analyses (Linear Regression at 3-months) – Aim 1*

Coefficients

	Unstandardized Coefficients		Standardized Coefficients			95.0% Confidence Interval (B)		Correlations		
	B	Std. Error	Beta	t	Sig.	Lower Bound	Upper Bound	Zero-order	Partial	Part
(Constant)	0.257	0.503		0.512	0.61	-0.741	1.256			
gender	0.012	0.116	0.002	0.099	0.921	-0.219	0.242	-0.346	0.01	0.002
age	-0.002	0.023	-0.001	-0.072	0.943	-0.047	0.044	-0.018	-0.007	-0.001
z BMI assessment	0.955	0.016	0.992	58.175	0	0.922	0.987	0.988	0.987	0.908
family ses	0.001	0.004	0.005	0.309	0.758	-0.006	0.008	-0.057	0.032	0.005
CDI Total Score	0.005	0.01	0.011	0.501	0.617	-0.015	0.026	0.087	0.052	0.008
perceived conflict with m	0.012	0.104	0.002	0.114	0.91	-0.195	0.218	-0.065	0.012	0.002
weight control habits tota	-0.007	0.01	-0.013	-0.684	0.496	-0.026	0.013	-0.08	-0.071	-0.011
total self-efficacy percent	-0.002	0.002	-0.019	-0.867	0.388	-0.006	0.002	0.175	-0.09	-0.014
total binge eating score	0.009	0.014	0.013	0.629	0.531	-0.019	0.037	0.007	0.065	0.01
total exercise	8.18E-06	0	0.013	0.797	0.428	0	0	-0.029	0.083	0.012

** Dependent Variable: month 3 zbmi*

TABLE 25. *Exploratory Analyses (Linear Regression over 3-months with Average z-BMI) – Aim 1*

Coefficients

	Unstandardized Coefficients			Standardized Coefficients		95.0% Confidence Interval (B)		Correlations		
	B	Std. Error	Beta	t	Sig.	Lower Bound	Upper Bound	Zero-order	Partial	Part
(Constant)	0.062	0.381		0.162	0.872	-0.693	0.816			
gender	0.075	0.087	0.012	0.858	0.393	-0.097	0.246	-0.365	0.077	0.01
age	-0.006	0.016	-0.004	-0.348	0.728	-0.037	0.026	-0.01	-0.031	-0.004
z BMI assessment	0.965	0.013	0.997	74.403	0	0.94	0.991	0.991	0.989	0.904
family ses	4.02E-05	0.003	0	0.015	0.988	-0.005	0.005	-0.082	0.001	0
CDI Total Score	0.001	0.008	0.002	0.129	0.898	-0.014	0.016	0.094	0.012	0.002
perceived conflict with m	0.02	0.076	0.003	0.259	0.796	-0.131	0.171	-0.08	0.023	0.003
weight control habits total	-0.001	0.007	-0.003	-0.183	0.855	-0.015	0.013	-0.064	-0.017	-0.002
total self-efficacy percent	-0.001	0.002	-0.013	-0.761	0.448	-0.004	0.002	0.123	-0.068	-0.009
total binge eating score	0.012	0.01	0.018	1.173	0.243	-0.008	0.031	0.043	0.105	0.014
total exercise	3.49E-06	0	0.007	0.547	0.585	0	0	-0.069	0.049	0.007

** Dependent Variable: average zbmi over 3 months*

TABLE 26. *Exploratory Analyses (Linear Regression at 3-months) – Aim 2 (CDI Total Score)*

Coefficients

	Unstandardized Coefficients		Standardized Coefficients			95.0% Confidence Interval (B)		Correlations		
	B	Std. Error	Beta	t	Sig.	Lower Bound	Upper Bound	Zero-order	Partial	Part
(Constant)	0.101	0.303		0.334	0.739	-0.498	0.7			
gender	0.052	0.096	0.008	0.539	0.591	-0.138	0.241	-0.362	0.049	0.007
age	-0.006	0.018	-0.005	-0.353	0.725	-0.042	0.029	0.034	-0.032	-0.005
z BMI assessment	0.96	0.015	0.991	66.042	0	0.931	0.988	0.988	0.986	0.911
CDI Total Score	0.006	0.007	0.012	0.823	0.412	-0.008	0.02	0.083	0.074	0.011

** Dependent Variable: month 3 zbmi*

TABLE 27. *Exploratory Analyses (Linear Regression at 3-months) – Aim 2 (CDI Individual Subscales)*

Coefficients

	Unstandardized Coefficients		Standardized Coefficients			95.0% Confidence Interval (B)		Correlations		
	B	Std. Error	Beta	t	Sig.	Lower Bound	Upper Bound	Zero-order	Partial	Part
(Constant)	0.106	0.31		0.341	0.733	-0.509	0.721			
gender	0.076	0.099	0.012	0.773	0.441	-0.119	0.272	-0.362	0.071	0.011
age	-0.01	0.018	-0.008	-0.533	0.595	-0.046	0.027	0.034	-0.049	-0.007
z BMI assessment	0.96	0.015	0.991	64.559	0	0.93	0.989	0.988	0.986	0.894
CDI Negative Mood, Scale A Total	-0.032	0.033	-0.018	-0.954	0.342	-0.098	0.034	0.067	-0.088	-0.013
CDI Interpersonal Problems, Scale B Total	0.073	0.048	0.024	1.515	0.133	-0.022	0.168	0.105	0.138	0.021
CDI Ineffectiveness, Scale C Total	0.015	0.029	0.008	0.53	0.597	-0.042	0.072	0.101	0.049	0.007
CDI Anhedonia, Scale D Total	0.004	0.021	0.003	0.182	0.856	-0.038	0.046	-0.023	0.017	0.003
CDI Negative Self-Esteem, Scale E Total	0.015	0.037	0.007	0.419	0.676	-0.057	0.088	0.128	0.039	0.006

** Dependent Variable: month 3 zbmi*

TABLE 28. *Exploratory Analyses (Linear Regression over 3-months with Average z-BMI) – Aim 2 (CDI Total Score)*

Coefficients

	Unstandardized Coefficients		Standardized Coefficients			95.0% Confidence Interval (B)		Correlations		
	B	Std. Error	Beta	t	Sig.	Lower Bound	Upper Bound	Zero-order	Partial	Part
(Constant)	0.031	0.218		0.14	0.889	-0.4	0.461			
gender	0.092	0.07	0.015	1.311	0.192	-0.047	0.231	-0.364	0.103	0.014
age	-0.007	0.013	-0.006	-0.585	0.56	-0.033	0.018	0.033	-0.046	-0.006
z BMI assessment	0.971	0.011	0.996	86.984	0	0.949	0.993	0.991	0.99	0.914
CDI Total Score	0.004	0.005	0.007	0.691	0.491	-0.007	0.014	0.095	0.055	0.007

** Dependent Variable: average zbmi over 3 months*

TABLE 29. *Exploratory Analyses (Linear Regression over 3-months with Average z-BMI) – Aim 2 (CDI Individual Subscales)*

Coefficients

	Unstandardized Coefficients		Standardized Coefficients			95.0% Confidence Interval (B)		Correlations		
	B	Std. Error	Beta	t	Sig.	Lower Bound	Upper Bound	Zero-order	Partial	Part
(Constant)	0.043	0.223		0.193	0.847	-0.398	0.485			
gender	0.099	0.072	0.016	1.378	0.17	-0.043	0.24	-0.364	0.11	0.015
age	-0.009	0.013	-0.008	-0.705	0.482	-0.035	0.017	0.033	-0.057	-0.007
z BMI assessment	0.971	0.011	0.996	85.48	0	0.948	0.993	0.991	0.99	0.905
CDI Negative Mood, Scale A Total	-0.016	0.022	-0.01	-0.712	0.477	-0.06	0.028	0.054	-0.057	-0.008
CDI Interpersonal Problems, Scale B Total	0.042	0.036	0.014	1.177	0.241	-0.029	0.114	0.08	0.094	0.012
CDI Ineffectiveness, Scale C Total	0.01	0.021	0.006	0.471	0.638	-0.032	0.052	0.138	0.038	0.005
CDI Anhedonia, Scale D Total	0.004	0.015	0.004	0.274	0.785	-0.026	0.034	0.001	0.022	0.003
CDI Negative Self-Esteem, Scale E Total	0.003	0.027	0.001	0.119	0.906	-0.051	0.057	0.118	0.01	0.001

** Dependent Variable: average zbmi over 3 months*

TABLE 30. *Exploratory Analyses (Linear Regression at 3-months) – Aim 3 (Adherence and Depressive Symptomatology)*

Coefficients

	Unstandardized Coefficients		Standardized Coefficients			95.0% Confidence Interval (B)		Correlations		
	B	Std. Error	Beta	t	Sig.	Lower Bound	Upper Bound	Zero-order	Partial	Part
(Constant)	0.228	1.051		0.217	0.828	-1.852	2.309			
gender	0.12	0.102	0.019	1.178	0.241	-0.081	0.321	-0.362	0.107	0.016
age	-0.014	0.018	-0.011	-0.795	0.428	-0.05	0.021	0.034	-0.072	-0.011
z BMI assessment	0.963	0.014	0.994	66.61	0	0.934	0.991	0.988	0.987	0.907
CDI Total Score	0.007	0.007	0.013	0.912	0.364	-0.008	0.021	0.083	0.083	0.012
total wks in treatment (max 12 wks)	0.002	0.088	0	0.018	0.986	-0.172	0.175	-0.025	0.002	0
total avg. food monitoring/wk (over 12wks)	-0.054	0.024	-0.033	-2.282	0.024	-0.101	-0.007	-0.051	-0.204	-0.031

** Dependent Variable: month 3 zbmi*

TABLE 31. *Exploratory Analyses (Linear Regression over 3-months with Average z-BMI) – Aim 3 (Adherence and Depressive Symptomatology)*

Coefficients

	Unstandardized Coefficients		Standardized Coefficients			95.0% Confidence Interval (B)		Correlations		
	B	Std. Error	Beta	t	Sig.	Lower Bound	Upper Bound	Zero-order	Partial	Part
(Constant)	0.221	0.239		0.926	0.356	-0.25	0.692			
gender	0.123	0.072	0.02	1.703	0.091	-0.02	0.266	-0.364	0.135	0.018
age	-0.009	0.013	-0.008	-0.745	0.457	-0.035	0.016	0.033	-0.059	-0.008
z BMI assessment	0.972	0.011	0.998	87.548	0	0.95	0.994	0.991	0.99	0.911
CDI Total Score	0.004	0.005	0.007	0.672	0.502	-0.007	0.014	0.095	0.054	0.007
total wks in treatment (max 12 wks)	-0.012	0.01	-0.014	-1.293	0.198	-0.032	0.007	-0.041	-0.103	-0.013
total avg. food monitoring/wk (over 12wks)	-0.029	0.016	-0.02	-1.799	0.074	-0.061	0.003	-0.031	-0.142	-0.019

** Dependent Variable: average zbmi over 3 months based on number of recordings*

TABLE 32. *Exploratory Analyses (Linear Regression at 12-months) – Aim 3*

Coefficients

	Unstandardized Coefficients		Standardized Coefficients			95.0% Confidence Interval (B)		Correlations		
	B	Std. Error	Beta	t	Sig.	Lower Bound	Upper Bound	Zero-order	Partial	Part
(Constant)	-13.106	17.529		-0.748	0.46	-48.657	22.446			
gender	-0.165	0.354	-0.031	-0.467	0.643	-0.883	0.552	-0.263	-0.078	-0.027
age	-0.152	0.065	-0.139	-2.328	0.026	-0.284	-0.02	-0.196	-0.362	-0.136
z BMI assessment	0.826	0.06	0.897	13.773	0	0.704	0.948	0.922	0.917	0.805
CDI Total Score	-0.008	0.022	-0.022	-0.35	0.728	-0.051	0.036	0.004	-0.058	-0.02
total weeks in tx for 1yr	0.332	0.363	0.062	0.915	0.366	-0.405	1.069	0.297	0.151	0.053
total avg. food monitoring/wk for 1yr	0.046	0.141	0.022	0.323	0.748	-0.24	0.331	-0.191	0.054	0.019

** Dependent Variable: month 12 zbmi*

TABLE 33. *Exploratory Analyses (Linear Regression over 12-months) – Aim 3*

Coefficients

	Unstandardized Coefficients		Standardized Coefficients			95.0% Confidence Interval (B)		Correlations		
	B	Std. Error	Beta	t	Sig.	Lower Bound	Upper Bound	Zero-order	Partial	Part
(Constant)	0.57	0.258		2.21	0.029	0.061	1.079			
gender	-0.008	0.083	-0.001	-0.092	0.926	-0.172	0.157	-0.38	-0.007	-0.001
age	-0.019	0.015	-0.016	-1.255	0.211	-0.048	0.011	0.023	-0.1	-0.015
z BMI assessment	0.945	0.013	0.986	72.755	0	0.919	0.97	0.987	0.985	0.898
CDI Total Score	0.004	0.006	0.009	0.716	0.475	-0.008	0.016	0.092	0.057	0.009
total weeks in tx for 1yr	-0.005	0.002	-0.027	-2.198	0.029	-0.009	0	-0.048	-0.173	-0.027
total avg. food monitoring/wk for 1yr	-0.004	0.023	-0.002	-0.155	0.877	-0.049	0.042	0.039	-0.012	-0.002

** Dependent Variable: average zbmi after 12 months*

TABLE 34. *Exploratory Analyses - Logistic Regression 5% - Psychosocial Variables*

Observed			Predicted		
			zbmifivepctyes_no		% Correct
			0	1	
Step 1	zbmifivepctyes_no	0	67	13	83.8
		1	35	19	35.2
	Overall Percentage				64.2

a. The cut value is .500

Variables in the Equation

Step 1		B	S.E.	Wald	df	Sig.	Exp(B)	95% C.I.for EXP(B)	
								Lower	Upper
	zbmi	0.027	0.065	0.176	1	0.675	1.028	0.905	1.167
	age	0.142	0.082	2.988	1	0.084	1.153	0.981	1.354
	gender(1)	-0.665	0.444	2.238	1	0.135	0.514	0.215	1.229
	cditot	-0.045	0.038	1.344	1	0.246	0.956	0.887	1.031
	clinicalmibq	-0.202	0.385	0.274	1	0.601	0.817	0.384	1.738
	famses	-0.009	0.013	0.481	1	0.488	0.991	0.966	1.017
	habtot	-0.049	0.037	1.762	1	0.184	0.953	0.887	1.023
	seperc	0.006	0.008	0.647	1	0.421	1.006	0.991	1.022
	bngscore	-0.021	0.05	0.173	1	0.678	0.98	0.888	1.08
	Constant	0.081	1.76	0.002	1	0.963	1.084	-	-

a. Variable(s) entered on step 1: zbmi, age, gender, cditot, clinicalmibq, famses, habtot, seperc, bngscore.

TABLE 35. *Exploratory Analyses - Logistic Regression 5% - CDI Total Score*

Step 1	Observed		Predicted		
			zbmifivepctyes_no		% Correct
			0	1	
	zbmifivepctyes_no	0	94	7	93.1
		1	60	3	4.8
	Overall Percentage				59.1

a. The cut value is .500

Variables in the Equation

Step 1		B	S.E.	Wald	df	Sig.	Exp(B)	95% C.I.for EXP(B)	
								Lower	Upper
	zbmi	0.003	0.06	0.002	1	0.964	1.003	0.892	1.127
	age	0.091	0.069	1.768	1	0.184	1.096	0.958	1.254
	gender(1)	-0.42	0.384	1.195	1	0.274	0.657	0.31	1.395
	cditot	-0.009	0.028	0.102	1	0.749	0.991	0.938	1.047
	Constant	-1.408	0.912	2.384	1	0.123	0.245	-	-

a. Variable(s) entered on step 1: zbmi, age, gender, cditot.

TABLE 36. *Exploratory Analyses - Logistic Regression 5% - Self-Monitoring Variable*

Step 1	Observed		Predicted		
			zbmifivepctyes_no		% Correct
			0	1	
	zbmifivepctyes_no	0	95	6	94.1
		1	59	4	6.3
	Overall Percentage				60.4

a. The cut value is .500

Variables in the Equation

Step 1		B	S.E.	Wald	df	Sig.	Exp(B)	95% C.I.for EXP(B)	
								Lower	Upper
	zbmi	0.002	0.06	0.001	1	0.978	1.002	0.891	1.126
	age	0.088	0.068	1.65	1	0.199	1.092	0.955	1.248
	gender(1)	-0.419	0.388	1.168	1	0.28	0.658	0.308	1.406
	totwklyavgfd48wks	-0.018	0.105	0.031	1	0.861	0.982	0.798	1.207
	Constant	-1.389	0.959	2.097	1	0.148	0.249	-	-

a. Variable(s) entered on step 1: zbmi, age, gender, totwklyavgfd48wks.

TABLE 37. *Exploratory Analyses - Logistic Regression 10% - Psychosocial Variables*

Step 1	Observed		Predicted		
			zbmitenpctyes_no		% Correct
			0	1	
	zbmitenpctyes_no	0	103	3	97.2
		1	27	1	3.6
	Overall Percentage				77.6

a. The cut value is .500

Variables in the Equation

Step 1		B	S.E.	Wald	df	Sig.	Exp(B)	95% C.I.for EXP(B)	
								Lower	Upper
	zbmi	0.079	0.074	1.16	1	0.281	1.083	0.937	1.251
	age	0.148	0.097	2.328	1	0.127	1.16	0.959	1.404
	gender(1)	-1.04	0.565	3.386	1	0.066	0.354	0.117	1.07
	cditot	-0.039	0.046	0.71	1	0.399	0.962	0.879	1.053
	clinicalmibq	-0.156	0.468	0.111	1	0.739	0.856	0.342	2.142
	famses	-0.021	0.016	1.74	1	0.187	0.979	0.949	1.01
	habtot	-0.049	0.046	1.18	1	0.277	0.952	0.87	1.041
	seperc	0.005	0.009	0.291	1	0.589	1.005	0.987	1.024
	bngscore	-0.033	0.061	0.283	1	0.594	0.968	0.858	1.092
	Constant	-0.703	2.093	0.113	1	0.737	0.495	-	-

a. Variable(s) entered on step 1: zbmi, age, gender, cditot, clinicalmibq, famses, habtot, seperc, bngscore.

TABLE 38. *Exploratory Analyses - Logistic Regression 10% - CDI Total Score*

Step 1	Observed		Predicted zbmitenpctyes_no		% Correct
			0	1	
	zbmitenpctyes_no	0	130	0	100
		1	34	0	0
	Overall Percentage				79.3

a. The cut value is .500

Variables in the Equation

Step 1		B	S.E.	Wald	df	Sig.	Exp(B)	95% C.I.for EXP(B)	
								Lower	Upper
	zbmi	0.048	0.069	0.499	1	0.48	1.05	0.918	1.2
	age	0.108	0.082	1.736	1	0.188	1.114	0.949	1.307
	gender(1)	-0.68	0.489	1.935	1	0.164	0.507	0.194	1.321
	cditot	-0.016	0.034	0.218	1	0.641	0.984	0.921	1.052
	Constant	-2.625	1.09	5.796	1	0.016	0.072	-	-

a. Variable(s) entered on step 1: zbmi, age, gender, cditot.

TABLE 39. *Exploratory Analyses - Logistic Regression 10% - Self-Monitoring Variable*

Step 1	Observed		Predicted zbmitenpctyes_no		% Correct
			0	1	
	zbmitenpctyes_no	0	130	0	100
		1	34	0	0
	Overall Percentage				79.3

a. The cut value is .500

Variables in the Equation

Step 1		B	S.E.	Wald	df	Sig.	Exp(B)	95% C.I.for EXP(B)	
								Lower	Upper
	zbmi	0.042	0.069	0.365	1	0.546	1.042	0.911	1.193
	age	0.104	0.081	1.663	1	0.197	1.11	0.947	1.3
	gender(1)	-0.631	0.494	1.635	1	0.201	0.532	0.202	1.4
	totwklyavgfd48wks	0.044	0.125	0.123	1	0.726	1.045	0.818	1.334
	Constant	-2.818	1.145	6.059	1	0.014	0.06	-	-

a. Variable(s) entered on step 1: zbmi, age, gender, totwklyavgfd48wks.

TABLE 40. *Exploratory Analyses – Kaplan-Meier Survival for 10% Weight Loss*

Means and Medians for Survival Time

gender	Mean				Median			
	Estimate	Std. Error	95% Confidence Interval		Estimate	Std. Error	95% Confidence Interval	
			Lower Bound	Upper Bound			Lower Bound	Upper Bound
male	10.66	0.445	9.788	11.532	.	.	.	.
female	10.306	0.31	9.698	10.915	.	.	.	.
Overall	10.421	0.255	9.921	10.92	.	.	.	.

** Estimation is limited to the largest survival time if it is censored.*

Overall Comparisons

	Chi-Square	df	Sig.
Log Rank (Mantel-Cox)	1.277	1	0.258

Test of equality of survival distributions for the different levels of gender

TABLE 41. *Exploratory Analyses – Kaplan-Meier Survival for 5% Weight Loss*

Means and Medians for Survival Time

gender	Mean				Median			
	Estimate	Std. Error	95% Confidence Interval		Estimate	Std. Error	95% Confidence Interval	
			Lower Bound	Upper Bound			Lower Bound	Upper Bound
male	9.226	0.596	8.057	10.395	.	.	.	.
female	8.396	0.429	7.556	9.236	.	.	.	.
Overall	8.665	0.35	7.979	9.35	.	.	.	.

** Estimation is limited to the largest survival time if it is censored.*

Overall Comparisons

	Chi-Square	df	Sig.
Log Rank (Mantel-Cox)	1.2	1	0.273

Test of equality of survival distributions for the different levels of gender