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Correlates of weight in adolescents: A path analysis

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Correlates of Weight in Adolescents: A Path Analysis

by

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Dedication

This project is dedicated to my son, Dylan.

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ABSTRACT

This study examined the interrelationships between adolescent weight and several other variables thought to impact weight and obesity: physical activity, depressive symptoms, binge-eating symptoms, dieting, socio-economic status, special education status, gender, and ethnicity. The sample consisted of 251 high school students in rural Florida who completed measures of depression, binge-eating, dieting, and physical activity. Measurement instruments included the Reynolds Adolescent Depression Scale-2nd Edition (RADS-2), the bulimia scale of the Eating Disorder Inventory-2 (EDI-2), the dieting scale of the Dutch Eating Behavior Questionnaire (DEBQ), and a physical activity questionnaire derived from the Youth Risk Behavior Scale for Students (YRBSS). The study utilized path analysis, a group correlational design, to determine whether the proposed path model fit the data. Obese and non-obese students also were compared with regard to a) the levels of binge-eating symptoms, and b) the relationship between binge-eating and depression. Path analysis results were not statistically or clinically significant, suggesting a poor fit of the model to the data. Results indicated 19% of participants were obese and 20% were overweight. More than three times as many obese students than non-obese students reported experiencing a binge-eating experience at some time in the past. However, on the bulimia scale of the EDI-2, obese and non-obese participants did not differ statistically in their responses. Statistically but

not clinically significant correlations were revealed between depression and binge-eating for the sample and also for non-obese students. For the sub-sample of 13 students who had both binged and dieted, 7 had binged first, 3 had dieted first, and 3 binged and dieted for the first time at the same age. Future research should continue to investigate the relationships of the variables related to obesity. Future directions might include a larger sample size and a modified sample selection process. Action research should continue in the areas of obesity prevention and intervention, and student services personnel should promote healthy lifestyle choices and a recognition of obesity as a socio-cultural problem.

Chapter I: Overview

According to recent estimates by the Center for Disease Control (CDC), at least 15% of all children and adolescents in the United States, and over 30% of adults, are obese (National Center for Health Statistics, 2002). The rapidly escalating problem of obesity receives almost daily attention in the news media, and many researchers continue to investigate its nature as well as the efficacy of prevention and intervention techniques. The traditional view of obesity as an individual problem is giving way to the perspective that obesity is a socio-cultural problem and that responsibility for treatment therefore lies with society as a whole and not only with the individual. In order to effectively prevent and treat obesity, we must appreciate the relationships among the constellation of variables that interact with obesity.

The Epidemic of Obesity

The CDC and most research studies define obesity using body mass index (BMI), with obese individuals having a BMI of at least 30 or at least at the 95^{th} percentile for their age and gender. BMI is an indirect measurement of adiposity or body fatness and is calculated from an individual's body weight in kilograms divided by height in meters squared (kg/m^2). Research indicates that, for the same BMI, the percentage of actual body fat tends to be higher for Asian-Americans and lower for African-Americans when compared to Caucasians (Prentice & Jebb, 2001).

Contrary to the common misperception that obesity is an eating disorder in the mental health realm of diseases, obesity is more accurately described as a medical

condition that affects progressively more and more of our country's adults and youth. Between 1980 and 2000, the prevalence of obesity doubled for children ages six to 11 and tripled for adolescents ages 12 -19 (National Center for Health Statistics, 2002). These rapidly escalating prevalence rates have led to the common perception today in the United States of obesity as an epidemic.

Obesity occurs at equal rates overall in adolescent boys and girls without respect to race, but prevalence varies between boys and girls of each race. Adolescent White males and females have similar rates of obesity (12.8% vs. 12.4%), Black females have a higher obesity rate than Black males (26.6% vs. 20.7%), and Mexican males have a higher rate than Mexican females (27.5% vs. 19.4%). Research also indicates that obesity prevalence is higher in low socio-economic status populations (Goodman, 1999; McMurray et al., 2000), and in children who receive special education services (Tershakovec, Weller, & Gallagher, 1994).

Obesity is associated with many negative and dangerous outcomes, both medical and psychosocial in nature. The CDC recently identified obesity as the second leading preventable cause of death in the United States, second only to smoking, and according to the same study obesity caused 400,000 deaths in 2000 (Mokdad, Marks, Stroup, & Gerberding, 2004). Obesity has been linked in adults to a host of medical problems including cardiovascular disease, cancer, coronary heart disease, hypertension, diabetes, gallstones, osteoarthritis, and prostate enlargement. Interestingly, up to one-half of adults with extreme obesity developed the condition in childhood or adolescence (Price, 2002; Steinbeck, 2001), and the more obese a child or adolescent is, the more likely he or she will be obese as an adult (Price, 2002). Obese youth suffer significant negative

psychosocial outcomes as well, including poor quality of life (Schwimmer, Burwinkle, & Varni, 2003), stigmatization (Latner & Stunkard, 2003), and stereotypes of obese children and adolescents as mean, ugly, lazy, stupid, selfish, unhappy, socially isolated, subject to teasing, and dishonest (Schwartz & Puhl, 2003). Thus, the propensity of negative outcomes associated with obesity clearly outlines the importance of understanding how to prevent and treat it effectively.

Risk Factors of Adolescent Obesity

Risk factors for obesity, or conditions that increase the likelihood of obesity, fall into one of two categories: fixed and variable. Fixed risk factors are not manipulable and therefore are not typically targeted for direct intervention. They may, however, point to particular demographically-defined groups of individuals that would benefit from prevention efforts. Research points to genetics, heritability, gender, race, age of the child, and socio-economic status as fixed risk factors. Alternatively, we can control and manipulate variable risk factors, and they present an excellent opportunity for intervention and prevention. Variable risk factors for obesity discussed in the literature include diet and physical activity, prenatal malnutrition, caloric intake during infancy, cultural trends, and psychological factors including infant temperament, self-esteem, anxiety, depression, and body image.

Genetics and heritability represent the most prominent fixed risk factors for obesity. Genetic research, leading to the discovery of specific genes and gene sequences related to obesity, has yielded evidence of some single-gene obesities. More progress is expected in the pursuit of this line of research (Price, 2002). In addition, heritability research over the last three decades, involving dozens of twin and adoption studies, has

firmly established the genetic role in obesity. Research consistently points to biological factors as accounting for up to two-thirds of the variance in obesity and weight (Grilo & Pogue-Guile, 1991; Price, 2002). It also appears that the influence of biological factors increases with the severity of obesity, and the risk of obesity if a family member is obese is much higher for extreme obesity (i.e., BMI over 40) than for moderate obesity (Price & Lee, 2001).

Other research-based, non-manipulable risk factors for obesity include gender and race. As stated in the earlier discussion on the prevalence of adolescent obesity, rates of obesity are similar for adolescent White males and females, adolescent obesity is higher for Black females than for Black males, and higher for Mexican males than for Mexican females. With regard to race, data also clearly indicate that obesity rates are higher for non-European populations such as African-American, Hispanic-American, and Native-Americans. Some researchers (Price, 2002) propose a genetic "thrifty" gene theory that applies to groups such as the Pima Indians in Arizona and Mexico, whose rates of obesity and diabetes are drastically lower in the non-Westernized lifestyle and environment. According to this theory, these groups adapted genetically in a manner that maximized the efficient use of energy, but in modern Westernized culture this metabolic tendency has become a liability. Genetic data do not yet exist to support or refute this theory, however genetic research related to obesity is largely still in its early stages and is expected to progress (Price, 2002).

Two final risk factors for adolescent obesity that could be considered fixed in nature include the age of the adolescent and his or her socioeconomic status. Research indicates that the risk of obesity is greatest at three points before adulthood: early

infancy, prepuberty, and adolescence (Stenbeck, 2001). Research is somewhat inconclusive on the role of socio-economic status in adolescent obesity, however much evidence suggests that for adult women low economic status is associated with obesity. In developing countries elsewhere in the world, however, high socio-economic status is correlated with obesity in children, women, and men (Berkowitz & Stunkard, 2002).

Perhaps more interesting than fixed risk factors for obesity are variable risk factors because of their potential for use in prevention and intervention. Cultural trends of the last several decades, including decreasing physical activity levels, increased dietary fat, more sedentary lifestyles, increased use of automobiles, television viewing, and more frequent dining out especially in fast food establishments, have been blamed by many researchers for the rise of obesity (Stunkard, 2002). It is likely that a genetic predisposition to obesity (i.e., a fixed risk factor) may interact with the recent cultural tendency towards increased dietary fat and sedentary lifestyle (i.e., a variable risk factor), resulting in the negative outcome of obesity or overweight for those individuals (Price, 2002). This negative interaction may serve to explain the drastic increase in obesity in America over the last several decades.

Support also can be found for other controllable risk factors including prenatal malnutrition, high caloric intake during infancy, and parental attempts to control food intake with their children, all of which reportedly increase the likelihood of obesity (Berkowitz & Stunkard, 2002). Prenatal malnutrition in the first half of the pregnancy has been found to predict obesity. Research also provides evidence that a vigorous feeding style during infancy predicts childhood obesity, as does caloric intake during infancy (Berkowitz & Stunkard, 2002). Interestingly, dietary intake in childhood has not

been found to predict later body fat levels. However, prompting of children by parents to clean their plates or eat more has been associated with weight in several studies (Berkowitz & Stunkard, 2002).

Other Variables Correlated with Adolescent Obesity

In addition to the risk factors for obesity of genetics and heritability, gender, race, age, socio-economic status, diet and physical activity, a review of the obesity literature reveals support for correlations between obesity and binge-eating behavior, depressive symptoms, and special education status. Evidence has accumulated in the past decade related to the relationship of binge-eating disorder with obesity. The American Psychiatric Association defines binge-eating disorder in the Diagnostic and Statistical Manual of Mental Disorders-Fourth Edition-TR (American Psychiatric Association, 2000) in an appendix for proposed diagnostic categories. Similar to bulimia nervosa, individuals with binge-eating disorder engage in binge-eating episodes but do not use purging or other inappropriate weight loss methods. Estimates of obese adults that have binge-eating disorder range from 3% to 30% (Stunkard, 2002), and European research has found that approximately 36% of obese children and adolescents also are binge-eaters (Decaluwe, Braet, & Fairburn, 2002). Research has also supported a relationship between binge-eating and dieting (Kinzl et al., 1999; Ross & Ivis, 1999), although it is not established which behavior precedes the other. Alternatively, research has firmly established dieting as a risk factor for anorexia nervosa and bulimia nervosa.

The amount of research on the relationship between depression and obesity over the past decades has ebbed and flowed, with a recent surge of interest. The urgency of understanding the nature of this relationship is emphasized by estimates that up to 8.5%

of adolescents in the United States suffer from depression (National Institute of Mental Health, 2000), and 7% of depressed adolescents may die by suicide as young adults (Weissman, Wolk, & Goldstein, 1999). Research from the 1960's and 1970's investigated depression and other psychological and behavioral constructs that were thought to underlie obesity. Later empirical studies in the 1980's and 1990's focused on obesity as a genetically mediated medical condition, and it was accepted that little or no relationship existed between obesity and depression (Stunkard, Faith, & Allison, 2003). Recently, however, research has focused once again on the possibility of a direct relationship between the two conditions (Goodman & Whitaker, 2002; Stunkard et al., 2003).

Little research has investigated the relationship between obesity and special education status. One study found that obese children were overrepresented in special education and remedial classes (Tershakovec, Weller, & Gallagher, 1994). However, this overrepresentation could be explained at least in part by the historical overrepresentation of minorities in special education (National Academy Press, 2002), along with the higher prevalence rates of obesity in minority children and adolescents (National Center for Health Statistics, 2002).

Research Objectives

In light of the serious and escalating nature of obesity as a public health concern, and the importance of identifying prevention and intervention opportunities, further research on the relationships between obesity and related variables is critical. Most obesity research conducted to date has focused on the relationships among one or two variables, and not on a larger view that considers the constellation of variables thought to

be associated with obesity. The present study will investigate the problem of obesity using a more comprehensive framework. The path diagram illustrated in Figure 1 portrays the relationships between obesity and correlated variables as currently supported in the literature. The model was constructed for the purpose of the present investigation, and draws upon research on the various interrelationships between adolescent obesity, binge-eating behavior, depressive symptoms, dieting, physical exercise, gender and ethnicity, socio-economic status, and special education status. The relationships depicted reflect those receiving the strongest empirical support, with the exception of genetics and heritability. The strong biological contribution to weight and obesity has received consistent support through two decades of research, and consequently the present model and study focus on other proposed relationships with obesity and weight.

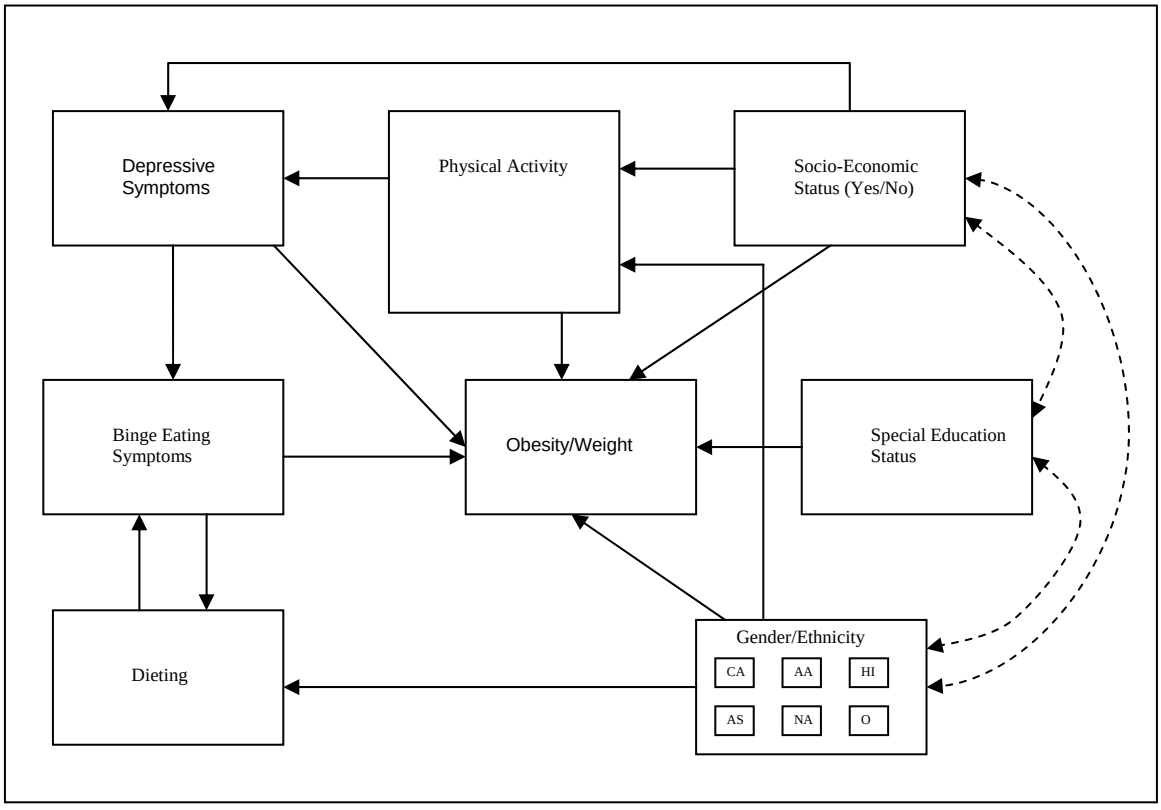

Figure 1. Proposed Path Diagram, Correlates of Weight in Adolescents

Further research into the relationship between obesity and depression is warranted given the lack of studies on this topic until very recently. If the rate of obesity continues to rise in adolescents, and depression is associated with obesity as a cause, effect, or correlate of obesity, it is critical for mental health and medical professionals to have an accurate understanding of the nature of this relationship. In particular, research will inform intervention efforts for that subgroup of obese individuals who also have symptoms of binge-eating, and are most at-risk for depression and other psychopathology compared to non-bingeing obese youth (Wilson, Heffernan, & Black, 1996).

Another shortcoming of existing obesity research is that much of the research has been conducted in Europe, limiting its generalizability to adolescents in America. Furthermore, much of the obesity literature neglects to differentiate between results for adolescents and adults. Given the known trajectory of obesity from childhood and adolescence into adulthood, it is necessary to identify the best potential areas for prevention and intervention, at the earliest age possible. In order to achieve this goal, more research specifically targeting children and adolescents is necessary. In addition, many of the existing studies on obesity and eating disorders included only females in their samples, further limiting the ability to generalize results to adolescents in America. Especially given the similar rates of obesity for males and females in the United States, more obesity research is needed that is inclusive of males.

The present study was designed to address some of the shortcomings in the existing obesity research by answering the following three research questions.

(1) To what degree do the data support the proposed model of the correlates of weight in adolescents?

(2) For the populations of obese and non-obese adolescents, what is the level of binge-eating symptoms, and what is the strength of the relationship between binge-eating symptoms and depressive symptoms?

(3) For the population of adolescents who engage in both binge-eating and dieting behavior, what is the order of precedence of binge-eating and dieting (i.e., which occurs first, binge-eating or dieting)?

It was expected that the data collected in this research study would support the proposed path model. Based on existing research, it was also expected that similar levels of binge eating symptoms would be identified for obese and non-obese adolescents, and that the level of binge-eating symptoms would positively correlate with the level of depressive symptoms. Finally, it was expected that, for adolescents who have a history of both bingeing and dieting behavior, a higher percentage of subjects would report that dieting preceded bingeing. Information gained from this study helps to inform critically needed efforts towards prevention and intervention for obesity by shedding more light on the nature of the relationships among obesity, depression, binge-eating, physical activity, dieting, socio-economic status, special education status, and gender and race.

Chapter II: Literature Review

Obesity in both children and adults currently poses a major public health threat in the United States. Recently identified by the Centers for Disease Control and Prevention as the number two preventable cause of death, obesity caused 400,000 deaths in 2000 and is solidly on track to overtake smoking as the number one preventable killer in the U.S. (Mokdad, Marks, Stroup, & Gerberding, 2004). The Centers for Medicare and Medicaid Services has revised its Medicare obesity coverage policy to eliminate language stating that obesity is not an illness, and intends to extend coverage for obesity treatments that are empirically based and shown to be effective in clinical trials (U.S. Department of Health and Human Services, 2004). The following review of the literature examines the available research on variables related to obesity. First, obesity research will be summarized with respect to its definition, prevalence in adolescents, outcomes, and risk factors including ethnicity, gender, socioeconomic status, and special education status. A discussion will follow of several constructs thought to be related to obesity, including binge-eating disorder, dieting, depression, and physical activity. These constructs will be summarized within the major sections of binge-eating disorder and depression. Together all of the research findings will be tied to a proposed path model for investigating and hypothesizing the relationships among the correlates of obesity.

Obesity in Adolescents

Obesity in both adults and adolescents represents an increasing problem in the United States and elsewhere in the world, and it is often referred to in the literature as an

epidemic (Ebbeling, Pawlak, & Ludwig, 2002). Obesity in children and adolescents poses a serious health risk. The fields of public health, medicine, and pediatric psychology have given increased attention to the problem of obesity in children and adolescents, as has the media. Given the current prominence of obesity as a health care concern throughout much of the developed and developing world, a firm understanding of the factors that correlate with and predict obesity carries obvious importance. The following discussion explores the definition and prevalence of obesity in adolescents, the medical outcomes associated with obesity, and the risk factors for obesity.

Definition of Obesity

In defining obesity, it is useful to begin with what obesity is not. Contrary to common belief, obesity is not an eating disorder, but rather a medical condition. In the Diagnostic and Statistical Manual of Mental Disorders, Fourth Edition (DSM-IV-TR), the American Psychiatric Association defines diagnostic criteria for anorexia nervosa and bulimia nervosa, but not for obesity, because there is insufficient evidence that obesity is associated with a psychological or behavioral syndrome (American Psychiatric Association, 2000). The DSM-IV-TR does, however, define research criteria for binge-eating disorder, a diagnosis proposed for further study that is quite prevalent in obese individuals.

Obesity is usually defined in terms of body mass index (BMI), an index of weight and height equal to body weight in kilograms divided by height in meters squared (BMI = kg/m^2). BMI is an indirect measurement of adiposity or body fatness, and is intended as a screening tool for underweight or overweight. Epidemiological studies conducted around the world have used various definitions of obesity, including BMI at or above 95^{th}

percentile, weight-for-height ratio greater than two standard deviations from the median, and an age-adjusted BMI cutoff of 30 or greater (Ebbeling, Pawlak, & Ludwig, 2002). Average BMI varies by gender and age throughout childhood, and the Center for Disease Control (CDC) recommends the use of its BMI-for-age growth charts for children and adolescents age two and older (Center for Disease Control, n.d.). Figure 2 shows the

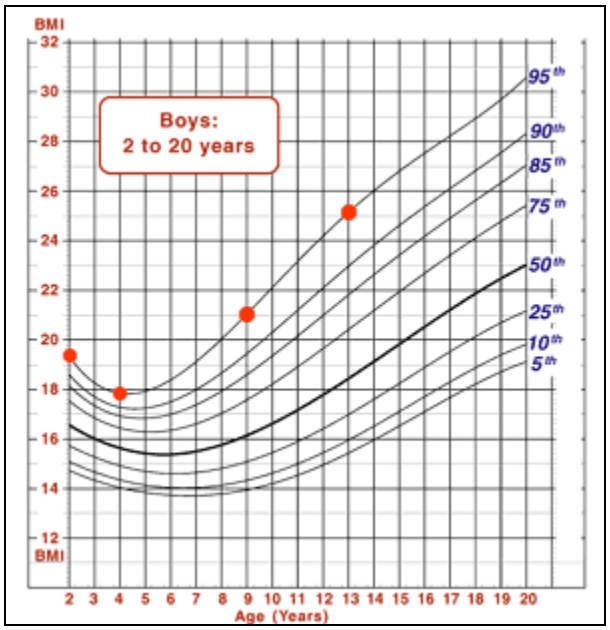

Figure 2. BMI growth chart for boys aged 2-20. From Using the BMI-for-age Growth Charts, Center for Disease Control (n.d.). Retrieved April 19, 2003, from http://www.cdc.gov/nccdphp/dnpa/growthcharts/training/modules/module1/text/intro.htm

BMI-for-age growth chart for boys, with BMI decreasing until age 4 and then increasing each year thereafter. Table 1 depicts the cutoff points recommended by the CDC to identify children and adolescents who are "at risk of overweight" and "overweight." The 85th and 95th percentile cutoff points correspond to CDC criteria classifying adults as "overweight" (i.e., adult BMI of 25) and "obese" (i.e., adult BMI of 30), respectively (Center for Disease Control, n.d.). Similarly, the 5th percentile cutoff point is the

criterion for identifying children and adolescents who are underweight, and it corresponds to an adult BMI of 18 or lower (Field, Barnoya, & Colditz, 2002).

Table 1

BMI Cutoff Points for Child, Adolescent, Adult Weight Classifications

Child/Adolescent			Adult
Classification	Percentile	BMI	Classification
Overweight	>= 95th	>=30	Obese
At Risk of Overweight	>= 85th	>= 25	Overweight
Normal	6th – 84th	19 - 24	Normal Weight
Underweight	<= 5th	<= 18	Underweight

Note. Cutoff points recommended by Centers for Disease Control. Adapted from Using the BMI-for-age Growth Charts, Center for Disease Control (n.d.). Retrieved April 19, 2003, from http://www.cdc.gov/nccdphp/dnpa/growthcharts/training/modules/module1/text/intro.htm

According to the CDC, obesity cutoff criteria for children and adolescents are based upon the 2000 CDC BMI-for-age-growth charts which are sex-specific. The adult BMI cutoff of 30 for obesity emerged in 1997 as an international standard when the World Health Organization (WHO) published terminology and classifications for overweight and obesity. These criteria have since been embraced by the CDC and the National Heart, Lung, and Blood Institute (NHLBI), among other federal agencies.

The CDC cites several empirically-based advantages to using its BMI-for-age charts as a screening tool for obesity, overweight, and underweight in children and adolescents (Center for Disease Control, n.d.). First, the charts represent a previously

unavailable reference source for adolescents. The measure can be used for an individual continuously into adulthood because it is consistent with the BMI index used for adults. BMI-for-age also correlates well with health risks for cardiovascular disease including high blood pressure and elevated insulin. Finally, web-based CDC training materials state that BMI correlates significantly with total and subcutaneous body fat.

While BMI serves as the commonly accepted measurement tool for obesity and other weight classifications in the United States and internationally, some researchers argue that more direct measures should be employed to determine levels of fat in the body (Prentice & Jebb, 2001). Direct methods for measuring body fat include waist-hip ratio, waist circumference, skinfold thickness measurements (e.g., tricep skinfold thickness), underwater weighing, dual energy x-ray absorptiometry (DMX), and other complex laboratory-based methods. These measures are necessarily more accurate and valid than the proxy method of BMI because they directly measure the amount of body fat. In some cases, BMI can even be inaccurate in its representation of body fat percentage. For example, the aging process for adults entails a continuous increase in the ratio between fat and lean body mass, even for individuals whose BMI remains the same throughout their lifespan. In addition, racial differences have been documented relating to the accuracy of BMI. Asian-American persons have a higher actual body fat percentage than Caucasians, and African-Americans have a lower percentage than Caucasians, for the same BMI. Similarly, the same level of body fat for Caucasians translates to a higher BMI for African-Americans and a lower BMI for Chinese, Thai, and Indonesians. Prentice and Jebb (2001) also point out the systematic error that occurs when utilizing BMI for athletes and others who pursue rigorous physical training and

buildup of muscle mass, which weighs more than body fat. Consequently, the high level of muscle mass leads to an overestimation of body fat in these individuals. Other researchers counter, however, that highly fit individuals rarely have enough muscle mass to be misclassified as obese (Field, Barnoya, & Colditz, 2002). Thus, BMI provides an established, well-accepted, and simple but not always accurate measurement tool for body fat percentage.

Prevalence of Adolescent Obesity

At least 15% of children and adolescents in the U.S. are obese, according to data from the most recent National Health and Nutrition Examination Survey (NHANES), which reports data for 1999-2000 (National Center for Health Statistics, 2002). The 15% prevalence rate applies to children ranging from six to 11 years, and to adolescents ranging from 12 – 19 years. These data represent the latest of several NHANES studies conducted since 1963, and were gathered by the CDC through household interviews and physical examinations of each participant. Participant data for all NHANES studies were stratified by sex, age, race, and Hispanic origin. The NHANES 1999-2000 study included 3,601 participants, a smaller sample size than either of the two previous studies from 1994 and 1980, which had 14,468 and 11,207 participants respectively. Consequently, standard errors of estimates cited for the latest study were somewhat higher than for the 1994 study, ranging from 1.2 to 3.3 percentage points for the 2000 data vs. standard errors of 0.9 to 2.5 percentage points for the 1994 data. Children and adolescents were classified as obese if their BMI was greater than or equal to the 95th percentile for their age and gender. Table 2 shows the alarming increase in obesity prevalence between 1980 and 2000, during which obesity rates doubled for children age

six to 11 years, and tripled for adolescents. These prevalence rates for children and adolescents trail those for adult obesity. The adult obesity rate according to the same 1999-2000 NHANES study was 31%, up from 23% in 1994 and 15% in 1980. These escalating rates of obesity cast light on the common reference to the condition as an "epidemic" in U.S. society.

Table 2

Prevalence of Obesity in U.S. Children, Adolescents, and Adults

Age in Years	NHANES II 1976-1980 n = 11,207	NHANES III 1988-1994 n = 14,468	NHANES 1999-2000 n = 3,601
6 – 11[a]	7	11	15
12 – 19[a]	5	11	15
>20[b]	15	23	31

Note. Adapted from National Center for Health Statistics, 2002. Retrieved April 19, 2003 from http://www.cdc.gov/nchs/products/pubs/pubd/hestats/overwght99.htm and http://www.cdc.gov/nchs/products/pubs/pubd/hestats/obese/obse99t2.htm

a. Obesity defined for children and adolescents: BMI >= 95th percentile for gender, age

b. Obesity defined for adults: BMI >= 30

The overall obesity prevalence for adolescents closely represents the rates for boys and girls in the NHANES 1999-2000 survey (National Center for Health Statistics, 2002). Interestingly, boys and girls 12 – 19 years of age both have a 15.5 % prevalence rate. However, the prevalence of adolescent obesity varies considerably among racial groups including non-Hispanic Whites, non-Hispanic Blacks, and Mexicans. As

illustrated in Table 3, White non-Hispanic boys and girls have similar prevalence rates of 12.8 and 12.4 %, respectively. However, obesity is more common in non-Hispanic Black females (26.6%) than males (20.7 %), and it is more prevalent in Mexican males (27.5%) than females (19.4 %). Adolescent obesity is again defined as having a BMI at or above the 95th percentile for age and gender, according to the CDC's 2000 BMI-for-age growth charts.

Table 3

Prevalence of Adolescent Obesity by Gender and Race, 1999-2000

	Total	Non-Hispanic White	Non-Hispanic Black	Mexican
Males 12 – 19 years	15.5%	12.8 %	20.7 %	27.5 %
Females 12 – 19 years	15.5%	12.4 %	26.6 %	19.4 %

Note. Adapted from National Center for Health Statistics, 2002. Retrieved April 19, 2003 from http://www.cdc.gov/nchs/products/pubs/pubd/hestats/overwght99.htm

Recent studies on the relationship between socioeconomic status (SES) and weight status for adolescents indicate that overweight and obesity are inversely related to SES. A North Carolina school-based study with a sample of 2,389 adolescents classified students as low, moderate, and high SES based on parental education level (McMurray et al., 2000). Using a criterion of BMI over the 85th percentile, the low SES group had the highest rate of overweight or obesity (41%), as compared to the moderate SES rate (35%), and the high SES rate (24%). Similar findings were reported by a large study

based on a sample of 15,483 adolescents from the National Longitudinal Study of Adolescent Health (Goodman, 1999). This study defined SES in terms of parental income, parental education, and resident parent occupation, and defined obesity as a BMI greater than the 95th percentile for age and sex. Obesity was linearly related to parental occupation and was also significantly related to household income and parent education.

Little research exists on the relationship between obesity and special education status, but there is some evidence that obese children are overrepresented in special education settings. The historical overrepresentation of minorities in special education (National Academy Press, 2002), combined with the higher rates of obesity in minority children and adolescents, would seem to predict an overrepresentation of obese children and adolescents in special education. In a study of black, inner-city elementary school students, obese students were twice as likely as non-obese students to be placed in special education or remedial settings (Tershakovec, Weller, & Gallagher, 1994). Given the homogeneous sample, this study inherently controlled for race and socio-economic status, so that educational setting could not be related to those factors. The results of this study should be interpreted with caution due to the facts that these data are 10 years old and data were only collected in elementary schools. In addition, the authors did not draw conclusions related to causality, and the study was cross-sectional rather than longitudinal.

Interestingly, another study has suggested a predictive link between learning problems and obesity. Lissau and Sorensen (1993) conducted a prospective longitudinal study whose participants consisted of a randomly selected sample of 1258 third graders in Copenhagen, Denmark, or 25% of the 1974 third grade population in the Copenhagen

municipality. The authors concluded that special education and learning difficulties in the third grade predicted obesity at age 20-21. The study controlled for socio-economic status, BMI in childhood, and gender in their data analysis. Baseline occurred in 1974 when subjects were in the 3rd grade, and follow-up was at age 20 or 21. Unfortunately these results are also somewhat dated, and may or may not generalize to Americans. In addition, the authors included no information regarding race or ethnicity of participants, preventing any analysis or control of race as a factor.

Outcomes of Adolescent Obesity

Obesity poses a serious health risk to the U.S. and other societies. Obesity in adults is associated with increased risk of mortality from all causes, especially cardiovascular disease. Obese adults are more likely than normal weight adults to develop a myriad of health problems including coronary heart disease, hypertension, Type 2 diabetes mellitus, numerous types of cancer (e.g., breast, endometrial, gastric, colon, esophageal, stomach), gallstones, osteoarthritis of the hip and knee, and prostate enlargement (Field, Barnoya, & Colditz, 2002). Given the rising prevalence of obesity in our country, it is also reasonable to expect the rates of all of these medical conditions to rise in future decades. Since obesity is in most cases a preventable disease, health professionals should place a significant focus on successful efforts towards obesity prevention.

Furthermore, research suggests that between one-third and one-half of extremely obese adults first develop obesity in childhood or adolescence (Price, 2002; Steinbeck, 2001), and the more extremely obese an individual is in childhood or adolescence, the more likely he or she will be obese as an adult (Price, 2002). Even if a child or

adolescent does not become an obese adult, he or she risks negative medical outcomes as a child or adolescent. For example, rising obesity rates are likely related to the recent increase in Type 2 diabetes mellitus in early adolescence (Steinbeck, 2001).

Poor quality of life can also be viewed as a negative outcome of severe obesity for children and adolescents. Although research exists on the health-related quality of life for adults with obesity, very little research has been conducted on the quality of life for children and adolescents with obesity. A recent hospital-based study of children and adolescents ages 5 to 18 years compared the health-related quality of life of 106 severely obese subjects with that of a group diagnosed with cancer and another group of healthy controls (Schwimmer, Burwinkle, & Varni, 2003). Using the PedsQL 4.0 (Varni, Seid, & Kurtin, 2001), a 23-item pediatric quality of life inventory, the researchers reported that the scores of obese subjects indicated an impaired health-related quality of life in all the domains measured: physical, psychosocial, emotional, social, and school functioning. The authors defined impairment as a score falling at least one standard deviation below the mean score for healthy participants. Surprisingly, the degree of impairment in all areas was similar to that reported by children and adolescents diagnosed with cancer and receiving chemotherapy.

Obese children and adolescents face frequent stigmatization by both peers and adults in society. There is evidence that this bias against obese children has increased significantly since the 1960's. Latner and Stunkard (2003) recently replicated a study conducted in the 1960's in which 458 5th- and 6th- graders ranked six drawings of one healthy child, one obese child, and four children with disabilities such as facial disfigurement, a missing hand, and a child in a wheelchair. Participants indicated how

much they liked each child by ranking the drawings from one to six with one representing the child they liked the most. Results indicated that the obese child was liked the least, as in the 1960's study. The healthy child had the highest mean rank of 1.97, the obese child had a mean rank of 4.97, and the children with disabilities had mean ranks ranging from 3.09 to 3.86. In addition, the obese child was liked 40.8% less in the current study than in the original study, and the authors concluded that stigmatization of obese children may have increased significantly since that time. According to attitudinal research, obese children and adolescents are associated with negative characteristics such as mean, ugly, lazy, stupid, selfish, unhappy, socially isolated, subject to teasing, and dishonest, and biased attitudes against obese peers apparently increases with age (Schwartz & Puhl, 2003). In summary, given the wide array of medical and psychosocial outcomes, prevention of adolescent obesity is critical.

Risk Factors of Adolescent Obesity

The success of prevention programs depends to a great extent on the understanding of factors that increase the likelihood of obesity, and also those that reduce its likelihood. Factors that can be influenced or manipulated provide the best opportunities for prevention and intervention, such as physical exercise and prenatal nutrition. On the other hand, some factors such as genetics and heritability are more fixed in nature and do not offer much in the way of intervention opportunities. Fixed factors thought to influence the likelihood of adolescent obesity include genetics, gender, race, and the age of the child. Fixed risk factors will be discussed first, followed by controllable factors.

An increasing amount of research has been conducted in the last two decades on the genetic contribution to obesity. Research surrounding the Human Genome Project has generated a prolific amount of data on DNA sequence and gene function as well as innovations in research technology. Scientists have provided evidence of several "single-gene obesities," or obesities which have been linked to a single gene. While these data and technology are impressive, little is still known regarding the specific genetic patterns that contribute to most common obesities. However, the field of genetics continues to operate in a period of rapid growth. Thus, the state of its knowledge base is expected to climb to new levels in the next several years, and will most likely illuminate the link between genetics and obesity (Price, 2002).

Genetic research can be distinguished from studies on heritability of obesity. Some of the most interesting studies that appear in the obesity literature have strived to distinguish the influence of inherited biological traits from that of environmental factors shared by family members. Many twin and adoption studies over the last two decades have demonstrated a strong influence of biological factors on obesity. One commonly cited seminal review (Grilo & Pogue-Geile, 1991) provides a good introduction to the importance of heritability in the occurrence of obesity. The authors analyzed approximately 45 studies conducted between 1970 and 1990, with sample sizes totaling approximately 28,000 pairs, or 56,000 subjects. The studies compared various pairs of related individuals, including biological siblings reared together, adoptive siblings reared together, and twins reared together versus reared apart. Twin studies produced data for both dizygotic twins (i.e., fraternal twins originating from two different eggs), and the more rare monozygotic twins (i.e., identical twins originating from the same egg). Also

included in their review were studies on correlations of weight for spouses, and adoptive parents compared to their adoptees.

Grilo and Pogue-Guile (1991) reported strong evidence for biological influence on obesity based on the collective results of the studies reviewed. The authors reported that non-shared experiences among family members were a much stronger influence on weight, fatness, and obesity than were shared experiences among family members. According to Grilo and Pogue-Guile, examples of non-shared experiences include perinatal insult, peer relationships, or differences in parenting. Examples of shared experiences might include similar child-rearing practices and living conditions. For example, average correlations for weight in monozygotic twins reared apart, .72, were similar to that of monozygotic twins reared together, .80. The average correlations for BMI were also similar, .62 for twins reared apart and .74 for twins reared together. The authors also reported that adoptive siblings were uncorrelated in weight or fatness, and adopted children did not resemble their adoptive parents in weight or fatness. Also, their review found that adopted children and biological children resembled their biological parents in weight to the same degree.

Grilo and Pogue-Guile reported several methodological weaknesses in their meta-analysis, the most significant being the wide variation in subject age within many of the studies analyzed ranging from pairs of infants to elderly adults. Some of the studies combined results for subjects of different ages, making it difficult to identify patterns in weight occurring at different ages. In addition, the study makes no mention of race or ethnicity. These limitations notwithstanding, the authors found evidence of only minor influence of shared environmental experiences on weight. They concluded that unshared

experiences account for approximately 20% of the variability in weight, and that genetics accounts for most of the variability in weight and obesity.

A more recent review of the genetics and heritability research on obesity draws conclusions quite similar to those outlined in the seminal analysis by Grilo and Pogue-Guile (1991), one decade later. According to Price (2002), results from multiple twin and adoption studies suggest that up to two-thirds of the variance in obesity and weight in adults can be attributed to genes, and little influence can be attributed to family environment. While the author's summary of the heritability literature concurs with earlier findings, it should be noted that the methods used to conduct this review of the literature were not well documented, and the number of studies and their sample sizes were not clear.

Price and Lee (2001) also conducted research on odds ratios that represent the risk of obesity if a family member is obese, as determined by the ratio between prevalence in the family and prevalence in the population. The odds ratio associated with extreme obesity, or BMI greater than 40, is much higher than that for moderate obesity. Thus, it appears that the influence of genetics may increase with severity of the obesity. In sum, according to the literature the contribution of genes to overweight and obesity is clearly larger than any other factor, and while it obviously cannot be manipulated, at least with today's technology, it also cannot be ignored as an ongoing topic of research if one considers such possibilities as genetic manipulation or gene therapy.

Other non-manipulable risk factors for obesity possibly related to genetics are those of gender and race. Prevalence rates in adolescents do not show a clear difference between girls and boys, as shown in Table 3, but a much greater disparity exists between

adult women and men. In addition, the data in Table 3 support existing research on the higher obesity prevalence rates in non-European races including African-American, Hispanic-American, and Native-Americans. One theory advanced by Price (2002) and others is that these groups developed genetically in a way to allow them to survive hardship conditions by maximizing the efficient use of energy, however in modern Westernized culture it has become a cause of obesity. Groups such as the Pima Indians in Arizona are thought to perhaps have developed a "thrifty" genotype that mediates the storage and expenditure of energy. No genetic evidence exists to support this theory as yet.

Another unchangeable risk factor for obesity that is highly relevant to adolescent obesity is the age of the child. Research has shown that the risk of obesity increases at three points in the life cycle prior to adulthood: early infancy, prepuberty, and adolescence (Steinbeck, 2001). While the age of the child obviously cannot be altered, this knowledge provides good insight for intervention and prevention in terms of the best times to implement a program.

Socioeconomic status (SES), another potential fixed risk factor for obesity, has been found in some studies to correlate with obesity in children and adolescents, but the research is not consistent in this area. Berkowitz and Stunkard (2002) reported that 40% of the studies they reviewed found an inverse relationship between obesity and SES for children and adolescents, 40% showed no relationship, and 25% showed a positive relationship. The relationship between obesity and SES for adult women is consistently inverse, however. Interestingly, studies in developing countries showed a strong positive correlation between obesity and SES for children, as well as for women and men. Little

research exists on the eating behaviors of children of low SES in the U.S., but some researchers have theorized that diet may play an important role in weight status (McMurray et al., 2000).

In a study on the influence of physical activity, socioeconomic status, and ethnicity on the weight status of 2389 adolescents in North Carolina (McMurray et al., 2000), subjects in the low SES status group had a higher rate of overweight and obesity (41%) than participants in the moderate (35%) and high (24%) SES groups. The authors theorized that this higher rate of obesity was not related to physical activity, because individuals in the low SES group reported higher levels of physical activity as well as high-intensity activities. However, members of this group reported twice as much time spent on television viewing and video game play, suggesting that sedentary behavior may play a key role in weight status.

Despite the body of research pointing to genetics as accounting for most of the variability in weight, it is clear that some factor or combination of factors in the environment must be responsible for the rapid increase in obesity rates in recent decades. One common theory is that western culture has brought changes in lifestyle, especially relating to diet and exercise. As some researchers propose (Price, 2002), it is likely that the increase in dietary fat and the trend towards more sedentary lifestyles interact in a negative way for individuals who have a genetic predisposition towards overweight and obesity. Several researchers have found that lower levels of physical activity are associated with obesity in children (Berkowitz & Stunkard, 2002; Steinbeck, 2001). Physical activity also tends to decrease during adolescence in all children, and is influenced by parental physical activity levels. However, more research is called for

regarding physical activity as a risk factor for obesity, because no studies exist to verify that physical activity levels have decreased in the same time frame in which obesity has dramatically increased (Steinbeck, 2001).

Other controllable risk factors for adolescent obesity appear in the literature as well. Prenatal malnutrition has been found to increase the likelihood of obesity, if it occurs in the first trimester or first half of the pregnancy (Berkowitz & Stunkard, 2002). While breast versus bottle feeding does not appear to influence development of obesity, caloric intake during infancy does predict early childhood adiposity. However, childhood dietary intake does not reliably predict later levels of body fat. Evidence also suggests that a vigorous feeding style during infancy (i.e., rapid, long sucks with shorter intervals between sucks) predicts obesity. Several studies have also suggested a relationship between weight and parental attempts to control food intake with their children, such as prompting of children to eat more or clean their plates (Berkowitz & Stunkard, 2002).

In their analysis of risk factors for the development of early obesity, Berkowitz and Stunkard (2002) also refer to cultural trends in recent decades such as the increasing tendency of Americans to eat meals outside the home, dine in fast food establishments, a decline in physical education for students in the U.S., and an increased use of automobiles. Television viewing represents another controllable risk factor for obesity, due to its effects of reducing physical activity and increasing consumption of calories while watching TV or afterwards resulting from food advertising (Birch & Fisher, 1998).

One final area of research on risk factors for obesity is that of psychological factors, which could arguably be viewed as either fixed or variable factors. Research is not conclusive on whether infant temperament is related to later body fatness, or whether

a relationship exists between obesity and self-esteem, depression, or anxiety (Berkowitz & Stunkard, 2002). However, some evidence suggests that obese adolescents and children have greater body image dissatisfaction than non-obese peers.

Binge Eating Disorder in Adolescents

Eating disorders can be defined as "severe disturbances in eating behavior, maladaptive and unhealthy efforts to control body weight, and abnormal attitudes about body weight and shape" (Wilson, Heffernan, & Black, 1996, p. 541). In earlier decades, researchers often viewed obesity as an eating disorder caused by the same eating behaviors in all obese individuals (Stunkard, 2002). However, more recent research has shed light on the wide variation in eating behaviors in the obese population. Increasingly, binge-eating disorder has emerged as a correlate and contributor to obesity. Binge-eating disorder will now be discussed with regard to its definition and prevalence in adolescents, risk factors, and the relationship of binge-eating with dieting and obesity.

Definition of Binge-Eating Disorder

The DSM-IV-TR (American Psychiatric Association, 2000) defines diagnostic criteria for three eating disorders: anorexia nervosa, bulimia nervosa, and eating disorder not otherwise specified. The latter criteria are intended for disorders that do not satisfy the requirements for anorexia or bulimia, and the manual includes binge-eating disorder as an example. In 1994, the American Psychiatric Association formally defined binge-eating disorder in Appendix B of the DSM-IV as a proposal for a new diagnostic category (American Psychiatric Association, 1994). Those criteria remain in the DSM-IV-TR, as shown in Table 4.

Table 4

DSM-IV-TR Research Criteria for Binge-Eating Disorder

A. Recurrent episodes of binge eating. An episode of binge eating is characterized by both of the following:

   (1) eating, in a discrete period of time (e.g., within any 2-hour period), an amount of food that is definitely larger than most people would eat in a similar period of time under similar circumstances
   (2) a sense of lack of control over eating during the episode (e.g., a feeling that one cannot stop eating or control what or how much one is eating)

B. The binge-eating episodes are associated with three (or more) of the following:

   (1) eating much more rapidly than normal
   (2) eating until feeling uncomfortably full
   (3) eating large amounts of food when not feeling physically hungry
   (4) eating alone because of being embarrassed by how much one is eating
   (5) feeling disgusted with oneself, depressed, or very guilty after overeating

C. Marked distress regarding binge eating is present.

D. The binge eating occurs, on average, at least 2 days a week for 6 months.

E. The binge eating is not associated with the regular use of inappropriate compensatory behaviors (e.g., purging, fasting, excessive exercise) and does not occur exclusively during the course of Anorexia Nervosa or Bulimia Nervosa.

Note: From American Psychiatric Association (2000, p. 787)

Binge-eating disorder and bulimia nervosa share some of the same diagnostic criteria. Episodes of binge eating occur in both binge-eating disorder and bulimia nervosa. However, binge eating frequency is defined differently for the two disorders. Bulimia nervosa requires a frequency of two or more binge eating episodes a week for three months, while binge-eating disorder requires episodes to occur on two or more days per week for six months. In addition, bulimia requires the regular use (i.e., at least two times a week) of compensatory behaviors such as self-induced vomiting, misuse of

laxatives, diuretics, or enemas. The nonpurging type of bulimia specifies the regular use of fasting or excessive exercise, but not purging behaviors such as vomiting, laxatives, diuretics, or enemas. In order to meet the criteria for binge-eating disorder, a person could not regularly engage in any of these compensatory behaviors, although they may sometimes engage in them. Researchers on binge-eating disorder have varied in their criteria for "regular" compensatory behaviors, ranging from episodes occurring twice a week to not at all.

Prevalence of Binge-Eating Disorder

Many of the prevalence figures found in the research on binge-eating disorder do not provide separate rates for adolescents. Some research suggests that onset for binge-eating disorder occurs in late adolescence or in the early 20's, that females are 1.5 times more likely to develop the disorder than males, and the overall prevalence for adults in the general population ranges from 0.7%-4% (American Psychiatric Association, 2000). A limited number of studies have yielded prevalence estimates among adolescents. One of the most recent, a school-based epidemiological study, found a 1% prevalence of binge-eating disorder in Norwegian 15-year olds (Rosenvinge, Borgen, & Borresen, 1999). It is also evident that research on binge eating in adolescents often fails to use a strict definition of binge-eating disorder, so that prevalence rates reflect subclinical levels of binge eating. For example, in a Canadian school-based study of 1031 girls and 888 boys, 18.9% of the girls and 17.5% of the boys reported engaging in binge eating at least once in the last year without compensatory weight loss behaviors (Ross & Ivis, 1999).

Risk Factors for Binge-Eating Disorder

Similar to the findings of research on obesity, evidence exists for the contribution of both fixed and variable factors in binge-eating disorder. Literature supports the influence of the fixed risk factor of genetics, as well as a myriad of variable risk factors. In the area of heritability research, a recent large twin study involving 2163 female twins (Bulik, Sullivan, & Kendler, 2002) examined genetic factors, common environmental factors, and unique environmental factors. Using bivariate twin analysis, the researchers found a moderate heritability factor for binge eating, a strong heritability link for obesity, and a moderate overlap in the genetic contribution to obesity and binge-eating disorder. The study used a broad definition of binge-eating, however, and it included only white women.

The first controlled study to examine risk factors of binge-eating disorder, often cited in the recent eating disorder literature, was conducted in England in 1998 (Fairburn et al.). This community-based case control study differentiated personal and environmental factors that were reported to occur prior to the onset of disordered eating in the lives of 52 females age 16-35 with binge-eating disorder, 104 healthy control subjects, 102 with other psychiatric disorders, and 102 with bulimia nervosa. Compared to the healthy controls, those with binge-eating disorder were more likely to have had a pregnancy prior to the age of onset, and they reported significantly higher levels of negative self-evaluation, parental depression, major depression, conduct problems, deliberate self-harm, critical comments by family about shape, weight, or eating, and teasing about shape, weight, eating or appearance. Significantly higher exposure to several parent-related risk factors were reported including parental criticism, high

expectations, minimal affection, under involvement, maternal low care, and maternal overprotection. These subjects were also significantly more likely to report a history of sexual abuse, severe repeated physical abuse, and bullying.

The authors also reported that subjects with binge-eating disorder were differentiated from their matched subjects with other psychiatric disorders by increased exposure to low parental contact, critical comments by family about shape weight or eating, and the incidence of childhood obesity. Interestingly, there was no significant difference in individual risk factors between the group with binge-eating disorder and the group with bulimia nervosa, although levels of exposure to some factors were higher in subjects with bulimia nervosa than those with binge-eating disorder. The most significant drawback to this study is that adolescent subjects were not differentiated from adults, with generic results provided for the entire sample. However, since the mean age of onset of disordered eating was 16.8 years, it can reasonably be assumed that the risk factors occurred before or during adolescence for many or most of the subjects. Other limitations of this study include the fact that adolescent boys and men were not included in the sample, as well as the retrospective self-report nature of the study which prevents conclusions about the predictive ability of risk factors.

Interestingly, a recent prospective longitudinal study on the association of psychiatric disorders with the onset of binge-eating disorder concurred with the finding of Fairburn et al. (1998) that depression predicted binge-eating disorder for adolescents (Zaider, Johnson, & Cockell, 2002). When controlling for age, sex, ethnicity, SES, and comorbid psychiatric disorders, dysthymic disorder was the only psychiatric condition found to predict onset of binge-eating disorder or bulimia nervosa in the sample of 201

adolescents. There was no evidence that substance use was a risk factor for eating disorder symptoms in the sample. The authors concluded that individuals who experience chronic depressive symptoms in early adolescence are at significant risk for development of binge-eating disorder or bulimia nervosa during adolescence.

Relationship of Binge-Eating Disorder to Obesity

There is a general consensus in the literature that a sizeable proportion of obese adults and adolescents have binge-eating disorder. In adult patients seeking treatment for obesity, prevalence estimates for binge-eating disorder have varied widely from 3.4% to 30%, with interview-based methods of identification yielding lower prevalence rates (Stunkard, 2002). It should be noted that, while most individuals with binge-eating disorder identified from community samples are overweight, some have never been overweight. One community-based study found that of adult women with binge-eating disorder, 45% were of normal weight, 39% were overweight, and 12% were obese (Kinzl et al., 1997). Studies have reported that, compared with obese persons who do not binge, obese bingers have more severe obesity, earlier onset of overweight, earlier onset and more frequent dieting, and higher levels of psychopathology including depression, substance use, and emotional disorders. In his recent analysis of the literature, Stunkard (2002) found continued support for these findings.

One recent European study of 126 children and adolescents ages 10-16 seeking inpatient treatment for obesity found that 36.5% had engaged in binge-eating episodes over the previous month (Decaluwe, Braet, & Fairburn, 2002). However, of the 126 patients, 6.1% reported binge-eating at least two times per week as required to meet the DSM-IV criteria of binge-eating disorder. Females and males had similar rates of binge-

eating, or 37.3% and 35.3% respectively. Obese bingers were younger than obese non-bingers, with mean ages of 12.24 years and 13.23 years respectively. Unlike obese adults, obese bingers and obese non-bingers in this population did not differ significantly in degree of overweight.

Relationship of Binge-Eating Disorder to Dieting

From a theoretical standpoint, some health experts argue that for those on a restrictive diet that excludes fat, bingeing is sometimes nature's way of fighting back in order to allow the body to obtain the nutrients it needs and is being denied (Hartley, 1998). Research over the last two decades has documented many relationships between nutrients, levels of neurotransmitters such as serotonin, dopamine, and norepinephrine, and related effects on mood and behavior. Severe restrictions in diet can lead to deficiencies in various nutrients such as Vitamin B12, B6, Thiamin, Riboflavin, Folic Acid, and Vitamin C, then resulting in abnormal symptoms and behavior ranging from depression to aggression. Conversely, bingeing episodes, often triggered by a stressful event and involving high fat, high calorie foods, may parallel drug or alcohol abuse in their immediate ability to provide comfort and gratification (Hartley, 1998).

Research on both adolescents and adults has found a correlation between dieting behavior and binge eating status. One study found that women with binge eating behaviors dieted significantly more frequently than those who did not binge eat; and that those who went on one or more diets in the last year or engaged in chronic restrained eating behavior also engaged in more frequent binge eating episodes (Kinzl et al., 1999). In another study of 1919 high school adolescents, compared to the non-bingeing control group, both girls and boys who had engaged in binge eating in the last year were more

likely to report skipping meals and trying to lose weight (Ross & Ivis, 1999). The authors described a gradient in the prevalence of dieting behaviors that increased with the severity of bingeing/purging behavior, with the most dieting occurring in students with symptoms of bulimia nervosa. This study, however, used loose criteria for identifying bingeing, resulting in a sample group with subclinical levels of binge eating, limiting generalizability to adolescents with binge-eating disorder.

Though it seems clear that a relationship between dieting and binge eating exists, the literature shows some disagreement regarding whether or not dieting should be considered a risk factor for binge-eating. Because research has suggested dieting is a risk factor for bulimia nervosa and anorexia nervosa, and binge-eating disorder is similar to bulimia, early research on binge-eating disorder centered around a similar theoretical relationship between dieting and binge-eating disorder. In one outpatient study of 88 women and 2 men with binge-eating disorder or nonpurging bulimia nervosa, 60% of those with binge-eating disorder reported that dieting preceded binge eating (Santonastaso, Ferrara, & Favaro, 1999). These results were more variable than those of subjects with nonpurging bulimia nervosa, 88.9% of whom reported that they began bingeing after dieting. This study was limited for the purposes of research on adolescents because it included only adults in the sample and relied on retrospective self-report of the order of occurrence of dieting and bingeing for each patient. According to Stunkard's comprehensive review (2002), recent research indicates that more often than not, bingeing behavior precedes dieting rather than the other way around. It seems likely that the relationship between binge-eating disorder may be a bidirectional one, with dieting preceding binge eating for some adolescents but bingeing leading to dieting for others.

Depression in Adolescents

Interestingly, as recently as the 1970's, researchers generally held that depression in children and adolescents was a transitory state and developmentally normal, or did not exist at all (Hammen and Rudolph, 1996). Since that time, the research literature on child and adolescent depression has grown enormously and continues to receive attention. Depression also has received varying amounts of attention in the literature on obesity over the past several decades. In the 1960's and 1970's, many studies focused on psychopathology such as depression that was assumed to accompany or cause obesity. In the 1980's and 1990's obesity research moved towards a focus on the contribution of genetics and heritability to variations in weight, along with non-shared environmental causes of obesity. More recently, some studies have again examined the relationship between obesity and depression, as well as between binge-eating disorder and depression. In the interest of preventing depression, binge-eating, and obesity, a need exists to understand the nature of these relationships. The need to understand the depression component of the model is further highlighted by the finding that up to 7% of adolescents who develop depression may die by suicide as young adults (Weissman, Wolk, and Goldstein, 1999). The following sections outline the definition of depression, its prevalence in adolescence, risk factors, and the relationship of depression with binge-eating disorder, physical activity, and obesity.

Definition of Depression

The Diagnostic and Statistical Manual of Mental Disorders, Fourth Edition (American Psychiatric Association, 2000) places depression within the Mood Disorders section in Axis I of the DSM-IV-TR manual. It lists criteria for three discrete depressive

disorders: major depressive disorder, dysthymic disorder, and depressive disorder not otherwise specified. The depressive disorders may present differently in children and adolescents than adults, with an irritable or cranky mood rather than a depressed mood. Table 5 lists the criteria for a major depressive episode, one or more of which is required for a diagnosis of major depressive disorder. In addition to a consistently depressed or irritable mood or loss of interest in pleasurable activities over a period of two weeks, an adolescent experiencing a major depressive episode also may have trouble sleeping, eating too much or too little, trouble with concentration, motor agitation, fatigue, feelings of worthlessness, or suicidal thoughts. The average length of a depressive episode ranges between 16 and 36 weeks (Hammen & Rudolph, 1996).

Dysthymic disorder constitutes a somewhat less severe but very persistent form of depression, as compared to major depressive disorder. Rather than symptoms occurring most of the day every day for two weeks as in major depressive disorder, dysthymic disorder in an adolescent is characterized by a depressed or irritable mood for more days than not for at least one year, or two years for adults. The individual is not symptom-free for more than two months and has never had a major depressive episode or a manic episode. Depressive symptoms for dysthymic disorder are similar to those of major depressive disorder and may include eating too much or too little, sleeping too much (i.e., hypersomnia) or too little (i.e., insomnia), fatigue, low self-esteem, poor concentration, and feelings of hopelessness.

The third variety of depression as outlined in the DSM-IV-TR, depressive disorder not otherwise specified, serves as a category for depressive symptoms that donot fully meet the criteria for major depressive disorder or dysthymic disorder. This

Table 5

DSM-IV-TR Criteria for Major Depressive Episode

A. Five (or more) of the following symptoms have been present during the same 2-week period and represent a change from previous functioning; at least one of the symptoms is either (1) depressed mood or (2) loss of interest or pleasure.

    (1) depressed mood most of the day, nearly every day, as indicated by either subjective report or observation made by others. **Note**: in children and adolescents, can be irritable mood.
    (2) markedly diminished interest or pleasure in all, or almost all, activities most of the day, nearly every day (as indicated by either subjective account or observation made by others)
    (3) significant weight loss when not dieting or weight gain (e.g., a change of more than 5% of body weight in a month), or decrease or increase in appetite nearly every day. **Note**: in children, consider failure to make expected weight gains.
    (4) insomnia or hypersomnia nearly every day
    (5) psychomotor agitation or retardation nearly every day
    (6) fatigue or loss of energy nearly every day
    (7) feelings of worthlessness or excessive or inappropriate guilt (which may be delusional) nearly every day (not merely self-reproach or guilt about being sick)
    (8) diminished ability to think or concentrate, or indecisiveness, nearly every day (either by subjective account or as observed by others)
    (9) recurrent thoughts of death (not just fear of dying), recurrent suicidal ideation without a specific plan, or a suicide attempt or a specific plan for committing suicide

B. The symptoms do not meet criteria for a Mixed Episode.

C. The symptoms cause clinically significant distress or impairment in social, occupational, or other important areas of functioning.

D. The symptoms are not due to the direct physiological effects of a substance (e.g., a drug of abuse, a medication) or a general medical condition (e.g., hypothyroidism).

E. The symptoms are not better accounted for by Bereavement, i.e., after the loss of a loved one, the symptoms persist for longer than 2 months or are characterized by marked functional impairment, morbid preoccupation with worthlessness, suicidal ideation, psychotic symptoms, or psychomotor retardation.

Note: From American Psychiatric Association (2000, p. 356)

category covers several provisional disorders for which research criteria are provided, including premenstrual dysphoric disorder, minor depressive disorder, recurrent brief depressive disorder, and postpsychotic depressive disorder of schizophrenia. By definition, symptoms present for depressive disorder not otherwise specified are similar to those discussed for major depressive disorder and dysthymic disorder, but are present to a lesser degree.

Prevalence of Depression

According to the National Institute of Mental Health (NIMH), epidemiological studies indicate that up to 8.5% of adolescents in the United States suffer from depression (National Institute of Mental Health, 2000). Research by the NIMH also indicates that in recent decades, onset of depression has become earlier, and that earlier onset is related to more severe depressive symptoms in adulthood. The rate of depression in adolescents is higher than the 2-3% prevalence rate estimated for children aged six to eleven (Hammen and Rudolph, 1996). During childhood, boys and girls have similar rates of depression, but the rate of depression for adolescent girls is much higher than for adolescent boys, with the biggest gender difference occurring at about age 14. This trajectory continues into adulthood, when depressed females outnumber males approximately 2:1 to 3:1 (Hammen & Rudolph, 1996).

Risk Factors for Depression

Risk factors represent conditions or events which have been found to co-occur or correlate with the construct in question. Although the direction of causality is not always clear, the more risk factors and the fewer protective factors that are present for the adolescent, the more likely depressive symptoms will also be present. Risk factors

identified in the literature for adolescent depression include stress, cigarette smoking, loss of a parent or loved one, breakup of a romantic relationship, attention, conduct, or learning difficulties, abuse or neglect, and other trauma including natural disasters (National Institute of Mental Health, 2000). Cognitive factors that have been found to predict depressive symptoms include pessimism, low self-concept, and a "depressive attributional style," or the tendency to attribute positive outcomes to external, specific, and unstable factors and negative outcomes to internal, global, and stable causes (Hammen & Rudolph, 1996). Although boys and girls are equally at-risk for depression during childhood, by adolescence girls are twice as likely to develop depression as boys (National Institute of Mental Health, 2000). This increased vulnerability for girls emerges in early to middle adolescence, at around the age of 14 (Hammen & Rudolph, 1996).

Some evidence exists that socio-economic status (SES) acts as a risk factor for depression in adolescents. The National Longitudinal Study of Adolescent Health looked at the effects of SES on the self-rated health of a nationally representative sample of 15,483 subjects (Goodman, 1999). SES was defined in terms of parental education, occupation, and household income; dependent variables included depression, obesity, asthma, suicide attempt in the last year, and prior sexually transmitted disease. Depression was measured using the Center for Epidemiological Studies Depression Scale (CES-D) with separate cutoff scores for male and female subjects. According to the results, 9.3% of the sample scored above the cutoff level for depression. Using multiple logistic regression analysis to analyze the data, the researcher reported that the indicators of SES were consistently linearly associated with measures of depression, as well as with

obesity, but not for asthma. The author hypothesized that factors contributing to the SES effect on health included health-related behaviors, psychological characteristics, residential characteristics, social support, and access to health care. One limitation of this study was a reliance on the CES-D as a measure of depression, rather than a diagnostic interview. Also, because the study was school-based, the sample included only enrolled students and not adolescents who did not attend school, preventing generalizability to all adolescents.

In addition, research suggests that genetics and heritability play an important role in early-onset depression, defined as two or more episodes of depression before age 25. According to preliminary results from one study on early-onset depression, in which the mean age of onset was 15.6 years, 87% of the sample reported either a first- or second-degree relative with affective disorder (Smith, Muir, and Blackwood, 2003). However, the preliminary results of this ongoing study do not indicate use of a non-depressed control group. Until a specific genetic marker can be located, or twin studies are conducted to rule out environmental influences, it will remain unclear whether the familial transmission of depression is due to biological or psychosocial causes, or both. Based on available research, it seems reasonable to hypothesize that depression results from some combination of the two, with adolescents biologically predisposed to depression and then exposed to a triggering event.

Relationship of Depression with Binge-Eating Disorder

Recent research points to a probable bidirectional relationship between depressive symptoms and binge-eating disorder. A prospective longitudinal study (Zaider, Johnson, & Cockell, 2002) examined the association of psychiatric disorders with the onset of

binge-eating disorder and bulimia nervosa during adolescence. The researchers reported that, in the sample of 201 adolescents referred from school nurse offices and clinics, dysthymic disorder predicted the onset of binge-eating disorder or bulimia nervosa. Conversely, adolescents with eating disorder symptoms at baseline were significantly more likely to have dysthymic disorder at follow-up, suggesting a cyclical relationship between the two disorders. Regarding age of onset of binge eating and depressive symptoms, 40% of the sample reported that depression or anxiety had preceded their eating problems, 20% reported that eating problems preceded their depression or anxiety, and 40% reported that their difficulties with eating and depression or anxiety had started within the same year. While they stopped short of claiming evidence of causality, the authors concluded that a bidirectional association most likely exists between depression and binge-eating disorder.

Another recent study examined the correlation between binge eating and depressive symptoms (Ackard, Neumark-Sztainer, Story, & Perry, 2003). A sample of 4746 middle and high school girls and boys from Minnesota completed a depressive mood scale and a 221-item questionnaire on nutrition and eating habits. The authors declined to draw conclusions about causality because the study was cross-sectional, but they reported that students who met the criteria for binge eating disorder scored significantly higher on a depressive mood scale than those who indicated subclinical or no binge eating. One limitation of this study was the questions used to assess binge eating. The questions were adapted from an adult measurement scale for binge-eating disorder and therefore reliability and validity data were not available.

Relationship of Depression with Physical Activity

It seems reasonable that because an adolescent with depression is highly likely to experience the depressive symptom of fatigue (Hammen and Rudolph, 1996), that it would follow that his or her level of physical activity would be low. Unfortunately, little research exists on the relationship between depression and physical activity in adolescents in regard to exercise as a treatment for depression or as a preventative measure for depression (Paluska, 2000). However, according to a recent review of the literature on treatment of clinically depressed adults with physical exercise (Brosse, Sheets, Lett, and Blumenthal, 2002), intervention studies have provided evidence that exercise treatment is more effective than no treatment and it is as effective as psychotherapy and medication treatment. In addition, both cross-sectional and prospective studies found significant relationships between regular physical activity and lower scores on depression questionnaires. This correlation of exercise with fewer depressive symptoms has been suggested in samples of clinically depressed adults, as well as in healthy, non-depressed adults, cardiac patients, patients with chronic obstructive pulmonary disorder (COPD), and patients with neuromuscular disorders, rheumatoid arthritis, and osteoarthritis. The authors caution that the existing research base on adults is characterized by poor design and many methodological flaws. In addition, it is uncertain whether these correlations and effects of physical activity will generalize to adolescents.

German research on adolescents does support the idea that physical exercise is associated with lower levels of anxiety and depression, as measured by the anxiety/depression scale of the German version of the Achenbach Child Behavior

Checklist (Kirkcaldy, Shephard, & Siefen, 2002). A group of 1000 adolescents were given a questionnaire about physical activity level and asked to rank their regular continuous involvement in endurance sports such as cycling, swimming, and running. The authors reported that the adolescents who engaged in regular physical activity also had significantly lower anxiety-depression scores as compared to less active groups in the sample. However, the generalizability of this study to American youth is questionable since the study was conducted in Europe.

Relationship of Depression with Obesity

Theory and research on obesity in the 1960's and 1970's often focused on psychological and behavioral aspects that were thought to underlie or co-occur with obesity. In the following decades, obesity came to be viewed as predominantly a genetically mediated, biologically-based, physical disorder, and research on psychological correlates of obesity declined (Wilson et al., 1996). For many years, researchers of obesity and depression have had little communication and it was taken for granted that little or no relationship existed between the two disorders (Stunkard et al, 2003). Recently, however, several well-regarded medical researchers in the field of obesity and eating disorders have turned their attention towards understanding the relationship between depression and obesity (Goodman & Whitaker, 2002; Stunkard et al., 2003). Thus, in a sense, the focus of research on depression and obesity has come full circle with recent renewed attention on a possible direct relationship between the two disorders.

Stunkard et al. (2003) proposes a "moderator/mediator" framework for investigating the link between depression and obesity in adults, characterized by

moderator variables which define who is affected by the independent variable, and mediator variables which define to what extent the dependent variable is affected. According to this research-based theory, moderating variables include severity of depression, severity of obesity, gender, socioeconomic status, gene-environment interactions, and adverse childhood experiences. Mediating variables acting between the constructs of depression and obesity include eating and physical activity, teasing, disordered eating, and stress. This theoretical framework is valuable and especially useful for guiding future research, however it is based on research with adults. A similar model based on research in adolescent depression and obesity would be helpful in understanding these relationships for adolescents.

Stunkard et al. (2003) view severity of depression as a moderator variable for the association between adolescent depression and obesity because, while studies have not found a relationship between subclinical levels of depression and obesity in adults, one longitudinal study found a predictive relationship between major depression in children six to 17 years and their adult BMI as measured 10-15 years later (Pine, Goldstein, Wolk, and Weissman, 2001). This study used an experimental group of 90 children with major depression and a control group of 87 children with no psychiatric disorder. Children and adolescents with depression at baseline had a mean adult BMI of 26.1, compared to 24.2 for the control group. Although these results make a valuable contribution to the research, the study failed to differentiate children from adolescents at baseline, and they did not examine shorter-term effects of depression on BMI that may have been evident before the participants reached adulthood. It should also be noted that childhood depression did not specifically predict adult obesity, but rather a higher BMI. Also,

depression was not isolated as the sole predictor of BMI, because adult poverty was also found to predict adult BMI. The study controlled for socioeconomic status, age, gender, cigarette and alcohol use, and pregnancy and medication use, but it did not control for parental BMI.

Some recent evidence does point to a possible causal role of depression in the occurrence and persistence of obesity within adolescence. Researchers used data from the 1995 and 1996 "waves" of the National Longitudinal Study of Adolescent Health (Goodman and Whitaker, 2002). The school-based sample consisted of 9374 adolescents and was nationally representative of students in grades seven through 12. There was no significant correlation between baseline depression and baseline obesity. However, when controlling for socioeconomic status, physical activity, BMI at baseline, age, race, gender, parental obesity, number of parents in the home, self-esteem, delinquent behavior, and smoking, researchers reported that depressive symptoms predicted obesity at follow-up. Conversely, no evidence was found that obesity at baseline predicted depression at follow-up. Some limitations of this study were the use of self-reported rather than measured height and weight at baseline, the use of a self-report measure of depressive symptoms rather than a diagnostic interview, and the non-inclusion in the sample of youth un-enrolled in school.

In summary, little research exists on the correlational or the causal relationship between depression and obesity in adolescents. Some longitudinal evidence supports a causal relationship between childhood depression and adult BMI, and between adolescent depression and adolescent obesity. Theoretical explanations of a causal relationship from obesity to depression in adolescents have not yet received empirical support. Thus, the

available research suggests that the causal relationship between depression and obesity is unidirectional, with depression as a predictor of weight and obesity.

Conclusion

In conclusion, there are many risk factors and critical medical and psychosocial outcomes associated with obesity in adolescence. As emphasized by Steinbeck (2001), obesity has traditionally been viewed as a medical problem in the individual, caused by the individual, and which should be solved by the individual, rather than as a social issue with negative medical and psychosocial outcomes. Rather than taking this limiting view, we should strive to treat obesity as a preventable problem, placing responsibility for prevention not on one person or institution, but on society as a whole through community-based programs. Additional research is necessary to confirm the proposed risk factors discussed in the literature, and prevention programs should focus on those risk factors that can be influenced or manipulated. It is only through effective, wide-reaching prevention programs that the epidemic of childhood and adolescent obesity and its debilitating comorbid conditions can be slowed and ultimately stopped.

Research indicates that adolescents who develop binge-eating disorder represent the subpopulation of obese adolescents who are perhaps the most in need of intervention, due to their higher rate of depression and other psychopathology. Some treatments for binge-eating disorder have shown promise, particularly cognitive-behavioral therapy and interpersonal psychotherapy, but research shows they are not effective for approximately 40% of those who seek help (Stice, 1999). Additional knowledge must be gained in the mental health and health care fields about this disorder and its relationship to depression and obesity in order to improve treatment success rates.

Much of the literature on obesity, eating disorders, and depression fails to differentiate between results for adolescents and adults. In order to effectively plan for assessment and intervention for adolescents with disordered eating behaviors, it is necessary to be aware of the differences between adolescents and adults. For example, one study found that, compared to adults treated for an eating disorder, adolescents more often presented with a diagnosis of eating disorder not otherwise specified, had a lower global severity score, greater level of denial, less desire for help, and a history of fasting (Fisher et al., 2001). A need exists for additional research that focuses on adolescents. Furthermore, many of the studies on eating disorders included only females in their sample, and much of the literature on binge-eating disorder has been conducted in Europe. Additional research should be conducted that looks at both girls and boys, and more investigations are needed in the United States. More varied samples in terms of age, gender, nationality and culture will improve the external validity of that research for those demographic variables.

The research described in this review indicates the presence of several relationships among obesity, physical activity, depressive symptoms, binge eating, and dieting. If it can be determined that binge-eating disorder truly has a causal relationship with both depression and obesity, then targeting binge eating for intervention and prevention research would potentially also benefit adolescents with depression and obesity. Similarly, if there is evidence that physical activity has a causal relationship with both depression and obesity, then support will be given to targeting physical activity for intervention and prevention research. In addition, evidence that special education status has a high correlation with weight and obesity would support a focus on this

population for intervention and prevention. The same concept applies to correlations of weight with SES and gender/race, the other non-manipulable risk factors for obesity.

Research Questions

The objective of the present study was to answer three research questions:

(1) To what degree do the data support the proposed model of the correlates of weight in adolescents?

(2) For the populations of obese and non-obese adolescents, what is the level of binge-eating symptoms, and what is the strength of the relationship between binge-eating symptoms and depressive symptoms?

(3) For the population of adolescents who engage in both binge-eating and dieting behavior, what is the order of precedence of binge-eating and dieting (i.e., which occurs first, binge-eating or dieting)?

Path Model

Figure 1, appearing in Chapter 1, illustrates the proposed path diagram that depicts the relationships between the correlates of obesity as currently supported by the literature. Straight lines with arrows represent relationships with exogenous outcome variables to be analyzed in the study. The direction of the arrow symbolizes the direction of causality most clearly supported in the literature. Curved dotted lines with bidirectional arrows represent assumed correlations between exogenous variables. Additional details related to the path model and the data analysis to be performed will be addressed in the data analysis section of Chapter 3.

Chapter III: Research Methods

Design

The study used a path analysis design, which is a group correlational design in which the independent variables are continuous and are not manipulated. The path model represents hypothetical relationships between pairs of variables in the model. The model included exogenous variables, which are defined as those that are caused by forces outside the model, and endogenous variables, or those that are influenced by other variables within the model. Some relationships were hypothesized to be unidirectional, with the flow of causality from one variable to another in only one direction, and other relationships were thought to be bidirectional, with the flow of causality in both directions. The path model in its entirety represents the hypothetical relationships of the constellation of variables most significantly related to weight and obesity, as supported by theory and research.

The path diagram in Figure 3 includes four exogenous variables (i.e., not predicted by other variables on the diagram): socio-economic status, special education status, gender/ethnicity, and physical activity. The model also includes four endogenous variables (i.e., predicted by variables on the diagram): weight, dieting, binge-eating, and depression. All variable data types are continuous except for three dichotomous variables: socio-economic status, special education status, and gender/ethnicity.

Figure 3 contains "MODEL1," the initial model for the correlates of weight in adolescents. The model was constructed by the primary investigator for the purpose of

the present study. The model shows the initially proposed relationships between adolescent obesity, binge-eating behavior, depressive symptoms, dieting, physical exercise, gender and ethnicity, socio-economic status, and special education status. The relationships depicted in the model represent those that receive the most empirical and theoretical support in the literature.

Path analysis, a type of structural equation modeling, was used to analyze the model and to evaluate the model's goodness of fit with the data. Each of the rectangular boxes in the path diagram represents a measured variable which is an indicator of a latent variable (Hatcher, 1996). For example, depressive symptoms represent the total scaled score on the RADS-2, which in turn is an indicator of the latent variable depression. Straight lines on the diagram symbolize prediction, leading from predictor variable to outcome variable (Hatcher, 1996). For example, the model proposed that physical activity, binge-eating symptoms, depressive symptoms, socio-economic status, special education status, and gender/ethnicity act as predictors of weight. Curved, dotted lines on the path diagram are bidirectional with two arrows and represented an expected correlation between two exogenous variables (Hatcher, 1996). For example, a correlation was expected between socio-economic status and special education status (Tershakovec, Weller, & Gallagher, 1994), and between socio-economic status and gender/ethnicity (National Research Council, 2002). In the statistical analysis, the mathematical model assumed a correlation of zero between any two unrelated variables on the model.

Following data collection, it became necessary to modify the path model to be used in data analysis to the one shown in Figure 4. Initially, gender and ethnicity were to be combined as a single variable to allow analysis of gender-ethnic specific groups such

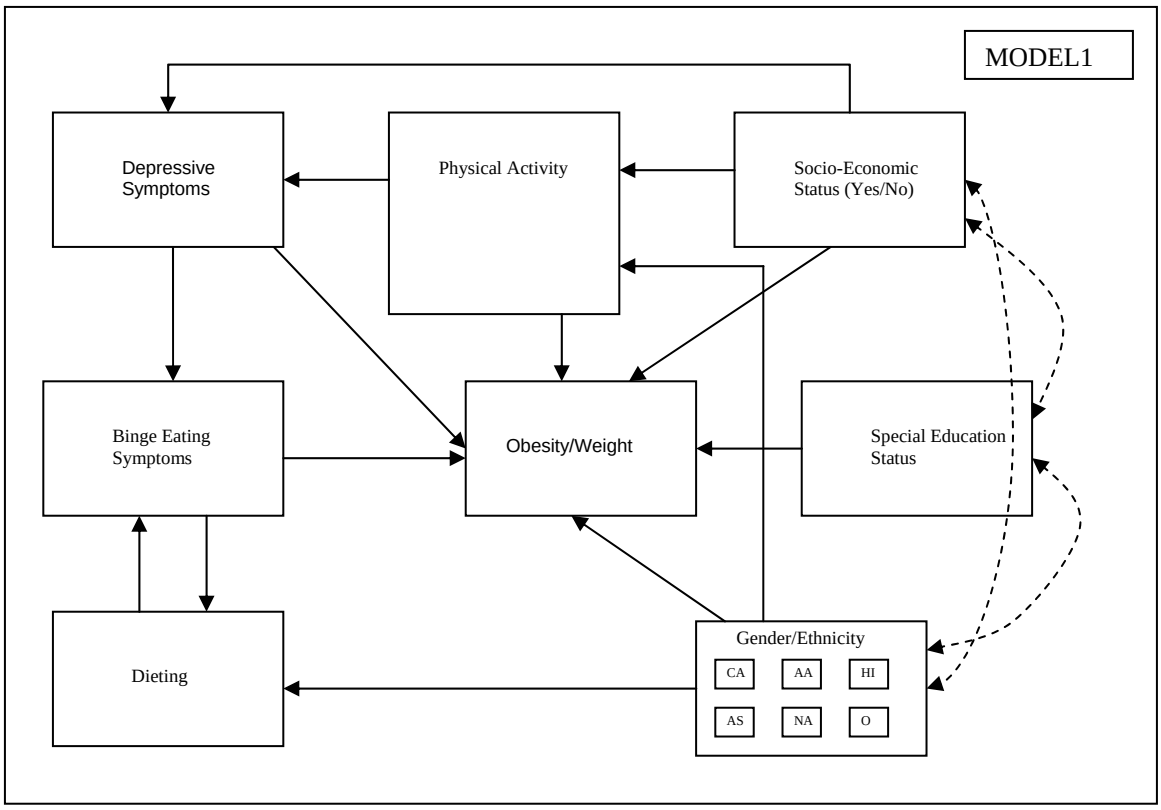

Figure 3. MODEL1: Initial Path Diagram, Correlates of Weight in Adolescents.

as Caucasian females, African-American females, Latino-Hispanic males, etc. However, the small sample sizes of many of these gender-ethnicity groups precluded using them in the path analysis. As discussed in the following section, only three sub-groups had more than 10 participants: White Females, White Males, and Latino/Hispanic Females. It was decided not to exclude gender entirely from the model because to do so would cause the path estimate results to be biased if gender did influence other variables in the model. Consequently, the gender and ethnicity variables became separate variables within the model. Both gender and ethnicity were treated as exogenous variables in the model, with ethnicity but not gender depicted as influencing weight directly, as suggested in the literature (National Center for Health Statistics, 2002). The MODEL2 diagram also illustrates the coding of dummy variables used in the data set for the variables socio-

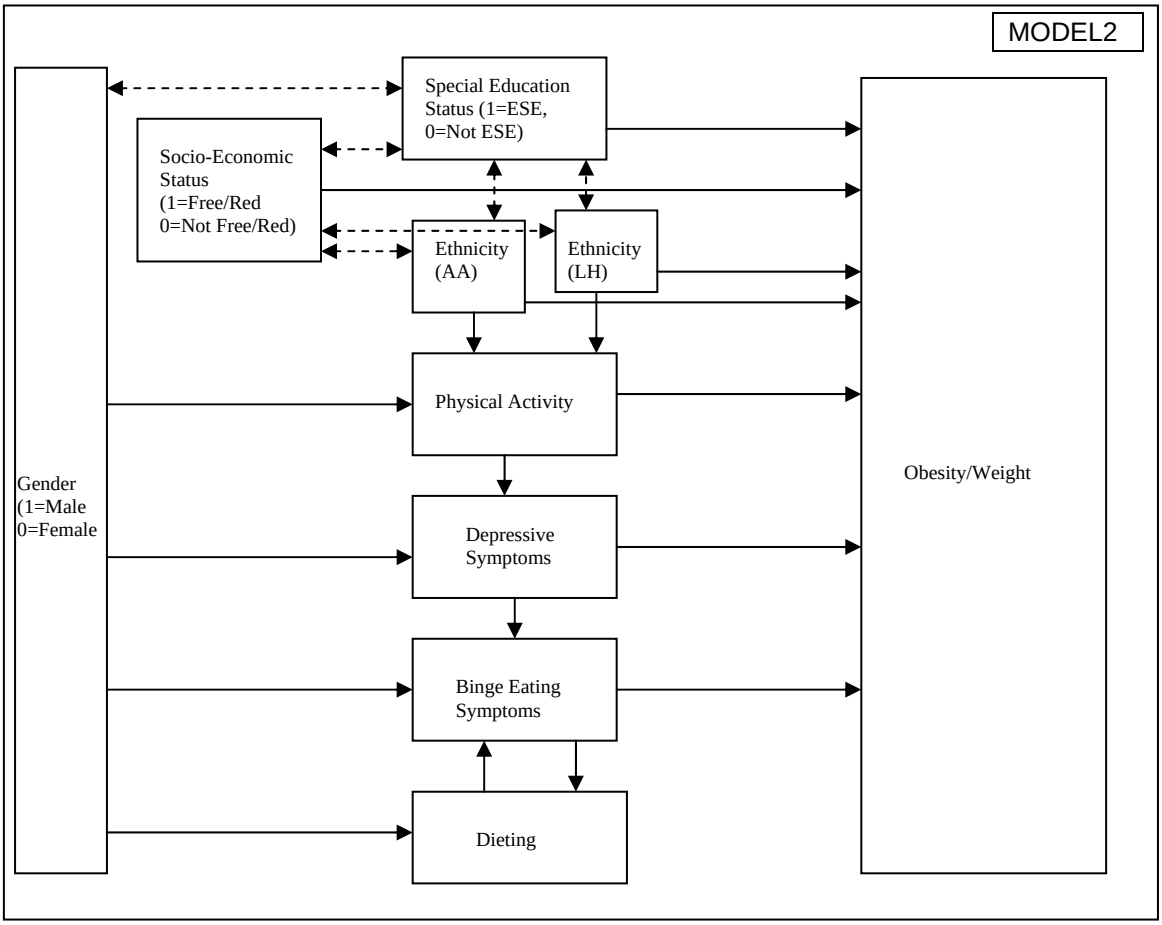

Figure 4. MODEL2: Revised Path Diagram, Correlates of Weight in Adolescents.

economic status, special education status, gender, and ethnicity. Because they are categorical in nature, these variables had to be treated as dummy-coded variables, in order to include them in the path analysis, and ethnicity had several possible values while the other categorical variables each had two possible values. Consequently, a "reference" group had to be chosen for ethnicity, to which all other ethnicities would be statistically compared during the data analysis. Caucasian, which was chosen by the greatest number of participants, was chosen as the reference group. The selection of Caucasian as a reference group often occurs in research, as in the National Research Council's use of

risk indices when calculating odds ratios for various ethnic groups when examining special education placement rates (National Research Council, 2002). The path model reflects that the Caucasian ethnicity served as the reference group, so that the path analysis essentially compared the relationships of the African-American group and the Latino/Hispanic group to those of the Caucasian group. Thus, the model contains boxes for the African-American and Latino/Hispanic groups but not for the Caucasian group.

Participants and Setting

The study was conducted during Spring, 2005 of the 2004-2005 school year and Fall, 2005 of the 2005-2006 school year in two central Florida high schools, hereafter referred to as High School 1 and High School 2. Both schools were within the same county and school district. High School 1 had an approximate enrollment of 1,638 9^{th} – 12^{th} graders in 2004-2005, and 1,700 in 2005-2006. The school was located in a growing, suburban/rural area of west-central Florida. As illustrated in Table 6, High School 1 was less demographically diverse than the district or state, with a 2005-2006 student population that was 92.2% White, 0.9% Black, 4.5% Hispanic, 0.9% Asian, 0.5% American Indian, and 1.1% Multiracial. Approximately 43.1% of High School 1's enrollment was reported to be economically disadvantaged, a rate similar to the 43.5% rate for the district and 45.9% rate for the state of Florida (Florida Department of Education, 2006b). High School 1 received a grade of D for the 2004-2005 school year and a C for 2005-2006 from the state of Florida as part of the statewide school grading accountability system. According to the Federal Government's No Child Left Behind accountability system, High School 1 failed to make Adequate Yearly Progress in either the 2004-2005 or 2005-2006 school year, meeting 73% and 77% of the criteria

respectively (Florida Department of Education, 2006a). According to the most recent data available from the Florida School Indicators Report, for the 2004-2005 school year, 0.5% of students were designated as Limited English Proficient. The report indicated a stability rate of 90.7% of students that remained enrolled throughout the year, and a graduation rate of 73.7%. The school also reported that 23.5% of students had disabilities, and 5.3% of students took the SAT college admission test (Florida Department of Education, 2005).

Table 6

Demographic Diversity of High Schools, District, and State

Ethnicity	High School 1	High School 2	District	State
White	92.2%	64.1%	78.9%	47.7%
Black	0.9%	14.3%	4.5%	23.4%
Hispanic	4.5%	19.7%	11.3%	23.4%
Asian	0.9%	0.5%	1.7%	2.2%
American Indian	0.5%	0.2%	0.3%	0.3%
Multiracial	1.1%	1.3%	3.3%	3.0%

Note. Adapted from Florida Department of Education, 2006b. Retrieved September 24, 2006 from http://doeweb-prd.doe.state.fl.us/eds/nclbspar/main0506.cfm.

High School 2 had an approximate enrollment of 1,294 students in 2004-2005 and 1,352 in 2005-2006. The school was located in a rural area of west-central Florida. Demographically, High School 2 was more diverse than High School 1 and the school population in 2005-2006 was reported to be 64.1% White, 14.3% Black, 19.7% Hispanic,

.5% Asian, .2% American Indian, and 1.3% Multiracial. Table 6 contrasts the ethnic makeup of High School 1 with that of High School 2, along with the district and state of Florida. Approximately 50.7% of students were reported to be economically disadvantaged in that same school year (Florida Department of Education, 2006b). High School 2 also received a D grade for both the 2004-2005 and 2005-2006 school year from the state of Florida as part of the statewide school grading accountability system. According to the Federal Government's No Child Left Behind accountability system, High School 2 also failed to make Adequate Yearly Progress in either the 2004-2005 or 2005-2006 school year, meeting 60% and 64% of the criteria respectively (Florida Department of Education, 2006a). The most recent data available from the Florida School Indicators Report show that for the 2004-2005 school year, 2.4% of High School 2 students were designated as Limited English Proficient. The report indicated a stability rate of 91.2% of students that remained enrolled throughout the year, and a graduation rate of 72.6%. The school also reported that 26.4% of students had disabilities, and 4.2% of students took the SAT college admission test (Florida Department of Education, 2005).

The participants of this study consisted of 252 students in grades 9 through 12. One participant's data were excluded from the data set because she was pregnant, resulting in an N of 251. This student's data were excluded due to the weight gain associated with pregnancy, because her weight was not representative of her normal weight. As shown in Table 7, participants were 56% female and 44% male. Fifty-three percent attended High School 1 and 47% High School 2. Table 8 shows the distribution of participants across ethnicity and gender. The participants were 79% Caucasian, with Latino/Hispanics identifying themselves as the largest minority. Seven students

identified themselves as a combination of two or three ethnicities, such as Caucasian and Latino/Hispanic. Table 9 depicts the ethnic distribution of the participants within each school, and this distribution resembled school enrollment. As shown in Table 10,

Table 7

Distribution of Participants by Gender and School

Gender	N	Percentage
N=Total Participants	251	100%
Female	141	56%
Male	110	44%
High School 1	133	53%
High School 2	118	47%
High School 1		
Female	66	50%
Male	67	50%
High School 2		
Female	75	64%
Male	43	36%

Table 8

Distribution of Participants by Ethnicity and Gender

Ethnicity	N	Percentage
Total Participants	251	100%
AA= African American	12	4.8%
Female (50%)	6	
Male (50%)	6	
AS= Asian	6	2.4%
Female (100%)	6	
Male	0	
CA= Caucasian	200	79.7%
Female (52%)	104	
Male (48%)	96	
LH= Latino/Hispanic	21	8.4%
Female (81%)	17	
Male (19%)	4	
NA= Native American	3	1.2%
Female (33.3%)	1	
Male (66.7%)	2	
OT= Other	2	0.8%
Female (100%)	2	
Male	0	
AA+CA+LH	1	0.4%
AA+LH	1	0.4%
AA+OT	1	0.4%
CA+LH	3	1.2%
CA+NA	1	0.4%

Table 9

Distribution of Participants by Ethnicity, Gender, and High School*

Ethnicity	High School 1		High School 2	
	N	Percentage	N	Percentage
AA= African American	2	1.5%	10	8.5%
Female	1		5	
Male	1		5	
AS= Asian	4	3%	2	1.7%
Female	4		2	
Male	0		0	
CA= Caucasian	116	87.2%	84	71.2%
Female	55		49	
Male	61		35	
LH= Latino/Hispanic	7	5.3%	14	11.9%
Female	5		12	
Male	2		2	
NA= Native American	1	0.8%	2	1.7%
Female	0		1	
Male	1		1	
OT= Other	0	0%	2	1.7%
Female (100%)	0		2	
Male	0		0	

* Note: Percentages do not sum to 100%. Three participants from High School 1 and four from High School 2 provided more than one ethnicity and are excluded from this table.

Table 10

Distribution of Participants by Grade and School

	Overall		High School 1		High School 2	
Grade	N	Percentage	N	Percentage	N	Percentage
Total Participants	251	100%	133	53%	118	47%
9	89	35%	54	40.6%	35	29.7%
10	63	25%	31	23.3%	32	27.1%
11	42	17%	18	13.5%	24	20.3%
12	57	23%	30	22.6%	27	22.9%

participants came from all grades but overall twice as many 9th graders participated than 11th graders. The participants were also similar to the schools' enrollment in terms of the percentage of special education students. Approximately 45% of participants overall were eligible for free or reduced lunch, with 50% eligible at High School 1 and 38% at High School 2. Table 11 illustrates free and reduced lunch eligibility by school. As shown in Table 12, 26% of High School 1's participants and 19% of High School 2's participants were enrolled in one or more special education programs at the time of data collection. The initial goal for participant recruitment was 200 students from each school. Following the 1st round of data collection at both schools in Spring 2005, a 2nd round of data collection was conducted at High School 2 in Fall 2005 in an effort to increase the numbers of minority participants in the study.

Table 11

Distribution of Participants by Free/Reduced Lunch and School

	Overall		High School 1		High School 2	
Free/Reduced Status	N	Percentage	N	Percentage	N	Percentage
Total Participants	251	100%	133		118	
Elig. Free or Reduced	112	45%	67	50%	45	38%
Not Elig. Free or Reduced	139	55%	66	50%	73	62%

Measures

Four self-report measures were utilized to gather data for this study: the Demographic and Physical Activity Questionnaire (DPAQ), the Reynolds Adolescent Depression Scale-2nd Edition (Reynolds, 2002), the Eating Disorders Inventory-2 (Garner, 1991), and the Dutch Eating Behavior Questionnaire (DEBQ). In addition, the school nurse, health assistant, and the examiner measured height and weight for each participant, which they used in turn to calculate body mass index (BMI). Each measurement tool is reviewed in this section with regard to the test components, test administration, and technical adequacy. The data elements provided by the instrument for the present study are defined.

Demographic and Physical Activity Questionnaire (DPAQ)

School nurses and clinic staff administered the Demographic and Physical Activity Questionnaire (DPAQ, Appendix B) that contains 10 demographic and personal information items, six physical activity items, and requires approximately 5 minutes to complete. The present study derived seven data elements from this questionnaire, including BMI, free or reduced lunch status, special education services received, gender,

Table 12

Distribution of Participants by ESE (Exceptional Student Education) Status

Special Education Status	N	Percentage
Total Participants (N)	251	100%
Not Special Education	195	78%
Special Education	56	22%
*A=EMH (Educable Mentally Handicapped)	8	14%
*B=TMH (Trainable Mentally Handicapped)	2	4%
*C=OI (Orthopedically Impaired)	4	7%
*F=Speech Impaired	13	23%
*G=Language Impaired	12	21%
*I=VI (Visually Impaired)	2	4%
*J=EH (Emotionally Handicapped)	6	11%
*K=SLD (Specific Learning Disability)	29	52%
*L=Gifted	4	7%
*V=OHI (Other Health Impaired)	2	4%
High School 1	133	100%
Not Special Ed	99	74%
Special Ed	34	26%
High School 2	118	100%
Not Special Ed	96	81%
Special Ed	22	19%

*Note: Some students classified with >1 special education exceptionality; A-V percentages calculated as percent of students classified as ESE.

ethnicity, and two types of physical activity engaged in within the last seven days. Following is a discussion of each data element gathered via the questionnaire.

Body Mass Index (BMI)

Weight was measured using a beam balance scale, and height was measured with the participant standing straight against a vertical scale attached to the beam balance scale. Both measurements were taken with participants wearing no shoes or heavy jackets. BMI was determined after measuring each student's height in inches and weight in pounds. BMI was calculated using an instrument that used the generally accepted formula (kg / m^2). At High School 1, the nurse utilized a standard BMI wheel on which the height and weight were lined up to yield the BMI. At High School 2, the nurse used a BMI calculator which produced the BMI after the height and weight were keyed in. All measurements and calculations were performed by the school nurse, health assistant, or the examiner. The BMI, stated as a decimal number with a resolution of 0.1 (e.g., 18.5), was then recorded on the survey for each participant in the appropriate box.

Socio-Economic Status (SES)

Free or reduced lunch status, which served as the socio-economic status for the study, was determined for each student from reports generated by the data entry employee at High School 2. The status was represented by a 'Y' to indicate that the student was eligible for free or reduced lunch, or 'N' to indicate that the student was not.

Exceptional Student Education (ESE) Status

The special education codes for each student were obtained from reports generated by the data entry employee at High School 2. ESE Status had possible labels of A-Educable Mentally Handicapped, B-Trainable Mentally Handicapped, C-

Orthopedically Impaired, F-Speech Impaired, G-Language Impaired, I-Visually Impaired, J-Emotionally Handicapped, K-Specific Learning Disability, L-Gifted, and V-Other Health Impaired.

Sex/Grade/Age/Ethnicity

Students provided demographic information on the survey about themselves. Each participant marked the checkbox for Male or Female, a checkbox for 9th, 10th, 11th, or 12th grade, and a checkbox for ages ranging from 13 to 20 years. Ethnicity was provided by marking the appropriate checkbox for the question, "Do you think of yourself as…?" Choices consisted of African American, Asian, Caucasian/White, Latino/Hispanic, Native American/Alaskan, and Other, with a space for the student to write an ethnicity not listed. For the purposes of this study, sex and ethnicity were initially to be combined into one variable to allow analysis of data for each combination of sex and ethnicity (e.g., African American girls, African American boys, Caucasian girls, and Caucasian boys). However, due to the small sizes of some of the groups it became necessary to treat ethnicity and gender as two separate variables during data analysis. The path model was modified accordingly and given the name MODEL2.

Physical Activity

The DPAQ questionnaire measured physical activity via seven questions, with the first two questions each providing the number of days during which the student engaged in exercise that either a) made the student sweat and breathe hard, or b) did not make the student sweat and breathe hard. The replies for those two items were summed and used as the physical activity variable in the study. These two items were selected to represent

physical activity because together they make up all types of exercise, assuming that all exercise either does or does not make one sweat and breathe hard.

The physical activity questions on the revised survey were derived from the Youth Risk Behavior Survey Surveillance (YRBSS), developed in 1990. The YRBSS was used to monitor behaviors linked to death, disability, and social problems in the United States. Test-retest reliability for the physical activity items on the YRBSS was found to be somewhat low at approximately .55 (Brener et al., 1999). However, it is unclear what time interval was used in this analysis.

Reynolds Adolescent Depression Scale-2nd Edition (RADS-2)

The Reynolds Adolescent Depression Scale-2nd Edition, or RADS-2 (Reynolds, 2002), was used to screen participants for depressive symptoms. The RADS-2 was published in 2002 and is the most recent version of the Reynolds Adolescent Depression Scale, originally published in 1987 by William Reynolds. The RADS-2 is a self-report measure designed for adolescents 11-20 years and can be administered in 5-10 minutes, either individually or in a group. It consists of 30 items written at the 3rd- grade level, and it provides standard T-scores and percentile ranks for total depression and each of four subscales: Dysphoric Mood, Anhedonia/Negative Affect, Negative Self-Evaluation, and Somatic Complaints. The T-Score for total depression provided the data element for depressive symptoms in the current study. The item content is designed to reflect DSM-IV criteria for depression. Empirically supported cut-off scores indicate the clinical severity of depressive symptoms (e.g., normal, mild, moderate, or severe) and assist in identification of adolescents who may have Major Depressive Disorder. In addition, six critical items are included to inform the clinician that the adolescent may need immediate

intervention. The materials include a hand-scorable test booklet and an optional summary/profile form that can be used to chart results of the four subscales. The present study used the hand-scorable test booklets but not the summary/profile forms, which are helpful for analyzing and communicating results but are not necessary for scoring.

The RADS-2 was standardized on a sample of 3,300 students. The sample was stratified by gender, age, and ethnicity to reflect the 2000 U.S. Census, and normative data are provided in the professional manual for each subgroup. The normative sample included an equal number of males and females, and each age group consisted of 1,100 adolescents. Considerable evidence of reliability and validity for the RADS-2 is also provided in the manual, and includes a school-based study with a sample of 9,000 adolescents and a clinic-based study with a sample of 297 adolescents. Reliability data exist for the total scale as well as each of the four subscales, and include internal consistency, test-retest, and standard errors of measurement. Internal consistency estimates for the RADS-2, based on a school sample of over 9,000 students, was high on the Depression Total scale ($r = .93$), moderately high ($r = .86$) for the subscales, and moderately high ($r = .86$) for males and females. Test-retest reliability, measured for 1750 students at a two week time interval, was determined to be high for the Depression Total scale ($r = .85$), and moderately high for the subscales ($r = .82$). Standard errors of measurement on the total school sample of over 9000 students ranged from 2.71 for the Depression Total to 4.5 for the Somatic Complaints subscale, based on T Scores which have a mean of 50.

Validity data on the RADS-2 include results of content, criterion-related, convergent, discriminant, and clinical validity analyses. Content validity was assessed

for the standardization sample of 3,300 adolescents through item-with-total Depression Total scale coefficients, and was considered to be adequate (median r = .53). The median item-with-total correlation coefficients for the four subscales also were considered to be high and ranged from .53 to .66. The RADS-2 manual also reports high criterion validity as measured by correlations between the RADS-2 and the Hamilton Depression Rating Scale, a clinical interview for depression (r = .82). Criterion validity also is supported by high correlations between the RADS-2 and other self-report measures of depression such as the Adolescent Psychopathology Scale (r = .74 to .76), the Minnesota Multiphasic Personality Inventory (r = .78), and the Beck Depression Inventory (r = .80). Convergent validity of the RADS-2 has been demonstrated with measures of related constructs including self-esteem, anxiety, and suicidal behaviors. RADS-2 discriminant validity was assessed using scales of social desirability, IQ, conduct disorder, substance abuse, and mania and determined to be low and therefore acceptable (r = .11 to .37). Finally, the RADS-2 manual reported support for contrasted clinical groups validity with a difference of over two standard deviations (sd = 2.19) in standard scores between a clinical sample of adolescents with depression and a school-based control group.

Eating Disorder Inventory -2 (EDI-2)

The Eating Disorder Inventory-2 (EDI-2) was published in 1991 and is the expanded form of the Eating Disorder Inventory (EDI), originally published in 1983. The EDI and EDI-2 were designed to assess symptoms of anorexia nervosa and bulimia nervosa in adolescents and adults 12 years and older. It is intended as a screening instrument and not as an exclusive method for diagnosing eating disorders. The EDI-2 requires about 20 minutes to complete and consists of 91 self-report items on 11

subscales. Items were written on a 5th grade level (Netemeyer & Williamson, 2001). Eight subscales originate from the EDI and remain unchanged in the EDI-2: 1) drive for thinness, 2) bulimia, 3) body dissatisfaction, 4) ineffectiveness, 5) perfectionism, 6) interpersonal distrust, 7) interoceptive awareness, and 8) maturity fears. Three subscales were added to the 64-item EDI to create the EDI-2: 1) asceticism, 2) impulse regulation, and 3) social insecurity.

The EDI-2 manual contains references to over 250 studies, reflecting the large amount of research involving the EDI and eating disorders. Reliability and validity data on the EDI-2 are strong (Ash, 1994; Shinke, 1994). Internal consistency was estimated at .8 and higher for the eight original subscales, for both eating disorder samples and nonpatient comparison groups (Ash, 1994). Test-retest reliability estimates for one- and three-week intervals between the tests are in the .8 and higher range. Validity research has found that the original EDI differentiates between patients with eating disorders and non-patient samples. Other research has shown evidence of the EDI's convergent validity with correlations between the EDI and other eating disorder scales such as the Eating Attitudes Test (Ash, 1994).

Most students participating in the study were observed to fill out the one bulimia scale on the EDI-2 in less than five minutes. The current study utilized only the EDI-2 bulimia scale for the binge-eating symptoms variable. The study did not make use of the other ten subscales. The bulimia scale is made up of six bingeing items and one purging item. Each item is on a six-point Likert scale with responses ranging from "always" to "never." All subscale scores on the EDI-2 are calculated by adding the raw item scores together for that subscale. The bingeing subscale score for the present study was

calculated in this manner. Because the study examined binge eating symptoms rather than purging symptoms, the one purging item was not included in the subscale raw score used for data analysis. Raw scores are converted to percentile ranks for each subscale of the EDI-2, and the manual does not report standard scores, T-scores, or scaled scores. The manual does list percentile ranks for normative groups of bulimia nervosa patients, anorexia nervosa patients, high-school boys and girls, and female and male college students. However, the high-school percentile ranks were inappropriate for data analysis in the current study because of the exclusion of the purging item from the subscale score. Similarly, Perez and Joiner (2003) measured disordered eating using only the bingeing items from the bulimia scale and reported that the raw bulimia subscale without the purging item had a kappa reliability coefficient of 0.82.

In order to address the research question related to the order of precedence of dieting and binge-eating, one question was added on the EDI-2. On the bulimia scale of the EDI-2, the question was added: "Have you ever experienced a binge eating episode -- a time when you ate an amount of food in less than 2 hours that was definitely larger than most people would eat in the same situation, and you felt like you could not control yourself or stop yourself? _____ (Yes/No) If "Yes", how old were you when you first experienced an episode: _____ years old (ex: 14 years old)." The wording for this question was based on the definition of a "binge" in the Diagnostic and Statistical Manual of Mental Disorders (American Psychiatric Association, 2000) and was intended to clearly and simply define "binge eating" for participants.

Dutch Eating Behavior Questionnaire (DEBQ)

The Dutch Eating Behavior Questionnaire (Van Strien, 2002) provided the dieting data for the study. This questionnaire was designed for adults, adolescents, and children nine years old and up. The DEBQ was created in 1986 and consists of 33 items on 3 scales that measure eating behavior: 1) emotional eating, 2) external eating, and 3) restrained eating. Questions are presented on a five point Likert scale and are estimated to be on a reading level between 5^{th} and 8^{th} grades. It is available in Dutch or English. Each scale in the DEBQ correlates to a theoretical cause of overeating, and the author further recommends guidelines for intervention and treatment of disordered eating based on the assessment results for each scale. According to the theory underlying the restraint scale, individuals overeat after a period of dietary restriction and the decision to eat less than wanted is no longer governing behavior. The emotional eating scale, which corresponds to psychosomatic theory, assumes that one overeats in response to negative emotions. The external eating scale is related to externality theory which proposes that individuals overeat in response to external cues such as the smell and sight of food.

Only the DEBQ's restraint scale, which has 10 items, was utilized in the present study. The restraint scale was selected to measure dieting behavior because it is regarded as a preferred measure of dieting behavior and it has been used in several studies with children and adolescents to examine dieting behavior (Netemeyer & Williamson, 2001). The other two scales of the DEBQ were not used in the present study because they do not provide information about the subject's dieting behavior. The DEBQ yields raw scores and norm-referenced scaled scores for each of the 3 scales. The scale score was calculated by dividing the raw score by the number of items completed; the score is not

valid if more than one item is omitted. Descriptive categories for test score ranges include very high, high, above mean, mean, below the mean, low, and very low. The entire DEBQ requires 10 minutes to complete, and participants in this study required no more than five minutes to complete the restraint scale.

Research supports the reliability and validity of the scores of the DEBQ restraint scale (Netemeyer & Williamson, 2001). The DEBQ is described as having excellent factorial validity, satisfactory to good reliability, and satisfactory concurrent and discriminant validity (Thames Valley Test Company, 2004). Reliability for the DEBQ restraint scale ranged from .92 to .95 (Cronbach's alpha) in a sample of 1170 subjects. The DEBQ manual also reports strong factorial validity for the restraint scale, with 68% of the variance among the items explained by a single factor when using exploratory and confirmatory factor analysis.

The DEBQ was normed in the Netherlands on samples consisting of over 2689 Dutch adults and adolescents including high school females, female college students, female eating disorder patients, obese men, obese women, non-obese men, and non-obese women. Additional normative statistics are provided for smaller subgroup samples, including 53 female obesity clinic patients, 154 female subscribers of a ladies weekly magazine, 77 female athletes, 54 female dance students, 104 high school boys, and 68 non-patient obese females. The fact that the DEBQ was not normed on Americans presents a limitation for the study and effectively calls into question the validity of the measurement tool for an American sample of adolescents, despite the evidence of validity for the original Dutch samples. However, it should be emphasized that, normative data issues notwithstanding, the DEBQ is regarded by researchers as a preferred measure of

dieting behavior in the United States and it has been used in several studies with children and adolescents (Netemeyer & Williamson, 2001).

In order to address the research question related to the order of precedence of dieting and binge-eating, prior to distributing the questionnaires one question was added on the DEBQ. On the DEBQ restraint scale, the question was added: "At what age did you first go on a diet (i.e., deliberately eat less in order to lose weight)? ____ years. Note: Leave this question blank if you have never been on a diet." The wording for this question resembled the wording for eating restraint items on the DEBQ and was intended to clearly and simply define "diet" for participants.

Procedure

The investigator for the present study obtained approvals from the school district's research office and from the University of South Florida Institutional Review Board (IRB) in January 2005.

Recruitment

After approvals were granted, the process to recruit participants began in both high schools in February and continued through May, 2005. The initial objective was to recruit approximately 200 students per school for a total of 400 participants. A larger sample of 1000-5000 would provide more reliability but would limit the practicality of the study, while a smaller sample of less than 200 would increase the likelihood of nonnormality and reduce the ability to accurately calculate goodness of fit (West, Finch, & Curran, 1995). All participants for the study who provided parent consent were accepted into the study, therefore constituting a convenience sample. The first round of data collection yielded 134 participants from High School 1 and 60 participants from

High School 2, for a total of 194 participants. According to the proposed study, if recruitment goals were not met then the study might be expanded to a 3rd school. However, by the time it became evident that the total number of participants would not meet the recruitment goal, it was too late in the school year to begin the recruitment and data collection process at an additional school. Rather than expand the study to a third high school in the fall, recruitment began again at High School 2 in Fall, 2005. Because High School 2 represented the most demographically diverse school in the district, and High School 1 one of the least diverse, it was expected that the number of minority participants could be maximized by recruiting at High School 2. According to the Florida Department of Education (2006b), the third high school under consideration was less diverse than High School 2 and the school population in 2005-2006 was reported to be 84.6% White, 3.6% Black, 9% Hispanic, 0.7% Asian, 0.6% American Indian, and 1.5% Multiracial. This contrasted with demographics at High School 2 of 64.1% White, 14.3% Black, 19.7% Hispanic, 0.5% Asian, 0.2% American Indian, and 1.3% Multiracial. Students signed up for the study at High School 2 from October through December 2005, and this second round of data collection yielded 58 new participants for an overall total of 252 students. None of the new participants for the second round had participated in the first round of data collection.

Several recruitment strategies were employed in each school. Due to different restrictions specified by the school principals, recruitment was school-wide at High School 1 but not at High School 2. Within this framework, recruitment procedures were followed as similarly as possible between the two schools. Recruitment procedures used

exclusively for High School 1 will be discussed first, followed by procedures used at High School 2, and lastly the procedures identical to both schools will be outlined.

In one method of recruitment at High School 1, the morning news show ran a daily announcement for approximately four weeks to request volunteers for the study. In addition, flyers were posted around High School 1 and on the clinic door advertising the study to students. Several teachers at this school agreed to make announcements about the study in their classes, including the ROTC teacher and some of the special education teachers. The examiner also made announcements in other classrooms such as the performance-based diploma program and a special education classroom for mentally handicapped students. Students were invited to come to the clinic between classes at High School 1 to sign up for the study. The clinic assistant maintained the sign up sheets in a secure location and once a week gave them to the primary investigator.

At High School 2, recruiting of participants was initially limited to Physical Education (P.E.) and Life Skills Management classes. The school nurse at High School 2, who served as the primary school contact for the primary investigator, made announcements in these classes to recruit participants. In addition, teachers made announcements in the E.S.O.L. (English for Speakers of Other Languages) class and the EMH (Educable Mentally Handicapped) class. Sign-up sheets were distributed in each class by the school nurse or teacher, and students were invited to sign up for the study at that time. The school nurse collected the sign up sheets and then forwarded them to the primary investigator.

Several recruitment procedures were identical at both schools. In both schools, P.E. and Life Skills Management teachers provided extra class credit towards their grade

to students for participating in the study. In addition, participants from both schools were informed that, if they participated in the study, they would be eligible for a drawing to win one of many prizes donated by local businesses. Businesses donated a total of 64 prizes for this study, which included movie passes, bowling passes, and gift certificates for restaurants, clothing stores, haircuts, and manicures. Letters used to solicit business donations were typed on school letterhead and approved by both school principals.

Obtaining Consent

At both schools, each student interested in participating in the study was instructed to write their student ID, name, address, and whether their parents spoke Spanish on the sign-up sheet. Teachers, clinic staff, and nurses forwarded the completed sign-up sheets to the principal investigator on at least a weekly basis. Data entry staff supplied printed address labels for the student population, and the principal investigator mailed a consent form home to each parent with a self-addressed stamped return envelope. A short note was included explaining that their son or daughter had signed up for the study. Parents signed and returned the consent form in the mail to the investigator. The consent form also included a child assent form to be signed by the student at the school prior to data collection. If the student indicated his or her parents spoke Spanish, the consent form was sent to the parent in both Spanish and English. Students who were 18 years or older did not require parent consent but were required to sign the informed consent themselves. Please refer to Appendix A for a copy of the English and Spanish parent consent forms.

At both schools, many students signed up for the study but provided no parent consent. Teachers followed up on students missing consent by hand-distributing consent

forms to return directly to the school. The number of consent forms that were returned by hand was not tracked, but most forms were returned in response to the mailing. In addition, a small number of students returned their mailed consent forms directly to the school, rather than mailing them. In those cases, students handed the forms to their teacher or to clinic staff who in turn gave them to the primary investigator. At High School 1, a total of 219 students signed up and 134 (61%) provided parent or adult consent. At High School 2, a total of 348 students signed up and 118 (34%) provided consent. High School 1 had a higher return rate, possibly due to the fact that the primary investigator provided school psychological services to this school, and thus had more regular and frequent contact with teachers, staff, and students.

Survey Packet Organization

The packets were pre-numbered consecutively beginning with 1, with the packet numbers written on each questionnaire in the packet. To counterbalance any potential order effects, the four questionnaires appeared in a pre-selected, counterbalanced order for each package. The four questionnaires had a total of 24 possible orders, and packets one through 24 were each in a unique order. That unique order of questionnaires was then repeated for packets 25 through 48, and so on for the rest of the packets. In this manner, an unintentional effect of the order of the questionnaires, such as unreliable results on the last questionnaire due to fatigue of the participant, was minimized.

Data Collection

During data collection, participating students were called to the school clinic at High School 1, where one office and the exam room were reserved for the study. Participants came first to the office which contained a round table and chairs. Similarly,

participants were called to a conference room at High School 2, occupied by a large conference room with chairs and the nurse's measuring station at the far end of the room for privacy. In both schools, students arrived in groups of five to ten at the beginning of a class period. As the students arrived, they were first directed to fill out the packet of questionnaires: the Nutrition/Physical Activity Questionnaire, the Reynold's Adolescent Depression Scale – 2nd Edition (RADS-2), the Eating Disorders Inventory – 2, (EDI-2), and the Dutch Eating Behavior Questionnaire (DEBQ). The principal investigator remained in the room with the students as they filled out the questionnaires. A standard list of instructions was posted and explained to assist the students in filling them out.

As each student completed the packet of questionnaires, he or she was instructed to proceed to the measuring station to have his or her height and weight measured and BMI calculated. Weighing, measuring, and BMI were typically conducted by the nurse at both schools. If the nurse was not available at High School 1, the health assistant conducted the weighing and measuring and BMI calculation, and in a few cases at High School 1 when neither the nurse nor health assistant was available, the principal investigator completed the height, weight, and BMI calculation. If the nurse was not available at High School 2, the principal investigator performed this role. When weighing and measuring were complete, the student's BMI (kg/m^2) was calculated using a BMI wheel (at High School 1) or a special BMI calculator (at High School 2). The calculator takes pounds and inches as input, converts to kilograms and meters, and then displays the BMI. The BMI was then written on the student's packet.

Following the weighing and measuring, each participant turned in his or her completed packet to the primary investigator, at which time the student received a ticket

for the drawing. Tickets for the drawings consisted of two identical parts with the student's name and ID written on each half; one half was given to the student and the other half placed in a box to be used during the drawing at the completion of that round of data collection for that school. At this point the student was instructed to return to class. Overall, each student missed a portion of one class period, and it took each student approximately 15-30 minutes to complete the forms and have their BMI calculated. This time requirement was consistent to expectations, based upon an early field test of the packet in which a student completed the packet in 9 minutes.

Post Data Collection Procedures

After students returned to class, the primary investigator hand scored the depression protocols the same day students completed them. The school psychologist, social worker, and/or guidance counselors were notified immediately, usually the same day, of any students whose scores fell in the at-risk range for depression. Standard procedures for students reported to have depressive symptoms were then followed whereby a school mental health professional talked with the student in confidence, and the student's parents were notified if deemed appropriate by the professional. Generally, according to school procedures, if a counselor, social worker, or psychologist speaks with the student and the student presents as depressed and in need of mental health care, parents are notified and a referral for outside counseling is offered by the school social worker. If the offer is declined by the family, the student is monitored on a weekly basis by staff, and with an understanding that the student can request to see the counselor or other mental health professional at school. It should be noted that the primary investigator was the school psychologist for High School 1, but not for High School 2.

Approximately 10% of participants scored above the cutoff for depression, and these data are further discussed in Chapter 4.

The prizes involved in the study attracted attention from students and staff, and the final step regarding participant prizes was the drawing for those prizes. Drawings were held in each school after each round of data collection was complete: once at High School 1 and twice at High School 2. Prizes were awarded by randomly selecting tickets from the box until all prizes had been awarded. Clinic staff in each school notified student winners and gave the prizes to the students. High School 1 participants received 38 prizes after the first round of data collection, and High School 2 participants won a total of 26 prizes including both rounds of data collection. In addition, participating clinic staff at both schools were provided with gift certificates.

Tracking of Participants

Several procedures were used in the tracking of participant packets. With regard to packet numbers, the packet number and the student's name were recorded in a separate "packet log" during data collection at both schools. Packets and packet numbers were distributed to students in numerical order as students arrived in the clinic, so that the first student participant received packet number one, the second received packet number two, and so on. The students' names did not appear on the packet or questionnaires, but the packet log identified what student completed a particular packet of questionnaires. The primary investigator kept all packet logs at a home office.

The primary investigator used the packet number on the questionnaires to link the questionnaires to a student name and ID on the packet log, so that the remaining data could be accessed in student records for each participant. A shaded box entitled "for

school use" appeared at the top of the Nutrition/Physical Activity questionnaire with the labels 'BMI,' 'SES,' and 'ESE.' The socio-economic status was coded as 'Y' or 'N' at the top of the questionnaire in the box labeled 'SES', depending on the free or reduced lunch status of the student. Likewise, if the student was served in any special education program, the codes of the programs were written at the top of the page in the box labeled 'ESE'. Data were recorded for each participant in an Excel file format which was then exported and used for analysis. Each student was identified in the data file by their packet number (1-252). Neither the student's name or school ID was entered in the data file.

Inter-rater Agreement

Inter-rater agreement was assessed for 36 participants in a randomly selected, 14% subset of the sample. Inter-rater agreement was calculated for the measurement of BMI, including height, weight, and calculation of BMI, and for the determination of socio-economic status and ESE status, the RADS-2 depression survey, and both eating behavior questionnaires, the EDI-2 and DEBQ. Packets were marked to identify those to be used for the inter-rater agreement calculation. Inter-rater agreement for BMI was calculated at High School 1 between the school nurse and health assistant if both were available, or between one of the two and the principal investigator. At High School 2, inter-rater agreement for BMI was calculated between the school nurse and the primary investigator. BMI's were considered in disagreement if the height, weight, or BMI index were not the same for both individuals. Inter-rater agreement on socio-economic status, special education status, and protocols for the RADS-2, EDI-2, and DEBQ were calculated between the primary investigator and a school psychology

practicum student, trained in psychoeducational assessment. The student looked up the free and reduced lunch status and special education status on printouts provided by High School 2, and compared them to those recorded by the principal investigator. Similarly, the student scored the protocols for the RADS-2, EDI-2, and DEBQ and compared the scores to those of the principal investigator. Table 13 shows inter-rater agreement rates for each variable. For BMI, height and weight were measured and BMI was then

Table 13

Inter-rater Agreement

Variable Measured	Percent
BMI	94%
Depression (RADS-2)	100%
Dieting (DEBQ)	100%
Bingeing (EDI-2)	97%
Socio-economic Status	100%
Exceptional Student Education Status	100%

calculated, and the two BMI's were compared. For agreement on socio-economic status, the free/reduced lunch eligibility was compared. For ESE status, the ESE categories were compared. Finally, agreement on the RADS-2, EDI-2, and DEBQ was based upon the standard scores calculated on the protocols. Inter-rater agreement ranged from 94% - 100% across the variables. In the few cases of disagreement, the two individuals resolved the disagreement and determined which rater was correct. In order to eliminate the disagreement, in the case of BMI, the student's height and weight were checked again

by both individuals; in the case of the Eating Disorders Inventory-2, the individuals reviewed the scoring protocol.

Chapter IV: Results

Data Analysis

Data analysis techniques employed to answer the study's three research questions included descriptive statistics, structural equation modeling, and correlation analysis. The Statistical Analysis Software (SAS) software package was utilized to perform all analyses. The following discussion outlines data analysis results for each research question.

Question 1: To what degree do the data support the proposed model of the correlates of weight in adolescents?

A descriptive analysis was done first on the data in order to assess normality, possible outliers, and the appropriateness of doing the path analysis. Descriptive statistics on the variables include a summary of the data and a correlation matrix for each variable in the model overall and for both schools. The reader may also refer to the description of participants in Chapter 3 for summaries of data on gender and school, ethnicity and gender, grade, socio-economic status, and special education status. The distributions of the following continuous variables are now described: BMI, age, physical activity, depressive symptoms, dieting, binge eating symptoms, age at first diet and first binge, have you ever been on a diet, and have you ever binged.

As shown in Table 14, the mean BMI overall for participants was 24.2, which falls at the high end of the normal range. According to the descriptive statistics in Table 14, the mean BMI was slightly higher at High School 2 (M=24.5) than High School 1

Table 14

Distribution of BMI

Variable Name	Mean	Median	Mode	Min	Max	Std. Dev.	Skew-ness	Kurtosis
BMI (N = 250*)	24.2	23.0	23.0	16.0	50.4	5.5	1.7**	4.4**
Age 14	23.0	22.0	20.0	17.0	38.1	5.0	1.5	2.4
Age 15	23.2	22.0	22.0	16.0	44.0	5.0	1.7	4.8
Age 16	24.2	23.0	21.0	17.0	37.0	4.4	0.9	0.1
Age 17	26.0	24.5	21.0	18.0	50.4	7.5	1.9	3.8
Age 18	24.6	23.0	23.0	18.0	47.0	5.4	2.0	6.0
Age 19	26.6	26.9	-----	20.5	32.0	5.0	-0.3	-1.6
High School 1	24.0	23.0	21.0	16.0	50.4	4.9	1.7	5.8
Age 14	22.9	21.8	20.0	13.0	38.1	5.0	1.8	4.2
Age 15	23.1	23.0	22.0	16.0	32.0	3.5	0.3	0.5
Age 16	25.3	24.3	22.0	18.5	37.0	4.9	0.7	-0.1
Age 17	25.0	23.3	21.0	18.0	50.4	7.5	2.3	6.5
Age 18	23.1	21.5	20.0	19.0	31.0	3.8	1.0	-0.4
Age 19	27.2	29.0	--	20.5	32.0	6.0	-1.3	--
High School 2	24.5	23.0	23.0	17.0	48.0	6.1	1.7	3.3
Age 14	23.0	22.0	18.0	17.0	37.0	5.2	1.4	2.0
Age 15	23.2	21.0	19.0	17.0	44.0	6.5	1.9	3.5
Age 16	23.5	22.8	21.0	17.0	33.0	4.0	0.9	0.2
Age 17	27.3	25.0	23.0	20.0	48.0	7.5	1.7	3.0
Age 18	26.4	25.0	23.0	18.0	47.0	6.5	1.8	4.7
Age 19	24.7	24.7	24.7	24.7	24.7	--	--	--

*note: missing BMI data for 1 participant
** note: distribution has moderate to extreme positive skew, leptokurtic distribution

(M=24.0). The mean BMI was highest for 19-year olds (M=26.6) and lowest for 14-year olds (M=23.0). The BMI data distribution showed a positive skew. Table 15 illustrates the prevalence of obesity, overweight, normal weight, and underweight in the data. The weight categories are based upon those used for the 1999-2000 NHANES 1999-2000 study: the body mass index-for-age percentiles developed by the National Center for Health Statistics, published in 2000 and currently recommended for use with children and adolescents by the Centers for Disease Control. As described in Chapter 2, these criteria define obesity as a BMI above the 95th percentile for age and sex, however, it is important to understand that the cutoffs for each weight category have remained the same since the 2000 publishing date. The present data show a higher prevalence of obesity in adolescents than reported in the 2000 NHANES study, at a rate of 19% compared to the earlier NHANES rate of 15%. Table 15 also displays weight categories for the three ethnicities with the largest sample sizes: Caucasian (N=198), Latino-Hispanic (N=21), and African-American (N=11).

Table 16 shows the data distribution for age, with a mean participant age of 16 years overall and for both schools and participants ranging from 14 to 21 years. Table 17 defines the ages of the participants in the study. Table 18 lists the distribution of physical activity, or the sum of the first two physical activity answers on the DPAQ (how many of the last 7 days did you participate in exercise that either did or did not make you sweat and breathe hard?). The mean total for the data set was 6.3 days, with High School 2 showing a slightly lower mean than High School 1, 6.1 days vs. 6.5 days. It should be noted that the responses for the two questions could have referred to the same days of the week, or to different days of the week. Consequently the total number of days for a

Table 15

Weight Category Distribution

	N	**Obese (%)	**Overweight (%)	**Normal (%)	**Underweight (%)
*Total Participants	247	46 (19%)	50 (20%)	149 (60%)	3 (1%)
Ages 14-19					
Males	108	22 (20%)	21 (19%)	64 (59%)	2 (2%)
Females	139	24 (17%)	29 (21%)	85 (61%)	1 (1%)
High School 1	130	24 (18%)	29 (22%)	75 (58%)	3 (2%)
High School 2	117	22 (19%)	21 (18%)	74 (63%)	0
Age 14	35	6 (17%)	8 (23%)	21 (60%)	0
High School 1	18	3	4	11	0
High School 2	17	3	4	10	0
Age 15	71	11 (15%)	16 (23%)	42 (59%	2 (3%)
High School 1	41	6	12	21	2
High School 2	30	5	4	21	0
Age 16	58	12 (21%)	11 (19%)	35 (60%)	0
High School 1	24	7	5	12	0
High School 2	34	5	6	23	0
Age 17	36	9 (24%)	6 (16%)	20 (57%)	1 (3%)
High School 1	21	4	4	12	1
High School 2	15	5	2	8	0
Age 18	42	6 (16%)	8 (19%)	28 (65%)	0
High School 1	24	3	3	18	0
High School 2	18	3	5	10	0
Age 19	4	1 (25%)	1 (25%)	2 (50%)	0
High School 1	3	1	1	1	0
High School 2	1	0	0	1	0

* note: BMI data missing for 1 participant, Age data missing for 1 participant

** note: Weight category definitions based on body mass index-for-age percentiles developed by National Center for Health Statistics in collaboration with the National Center for Chronic Disease Prevention and Health Promotion (2000).

http://www.cdc.gov/growthcharts

Table 15 (Continued)

Weight Category Distribution

	N	Obese (%)	Overweight (%)	Normal (%)	Underweight (%)
AA=African-American***	11	4 (36%)	0	7 (64%)	0
Female	6	4 (67%)	0	2 (33%)	0
Male	5	0	0	5 (100%)	0
CA=Caucasian***	198	36 (18%)	39 (20%)	121 (61%)	2 (1%)
Female	102	15 (15%)	21 (21%)	66 (65%)	0
Male	96	21 (22%)	18 (19%)	55 (57%)	2 (2%)
LH=Latino-Hispanic***	21	3 (14%)	9 (43%)	9 (43%)	0
Female	17	3 (18%)	6 (35%)	8 (47%)	0
Male	4	0	3 (75%)	1 (25%)	0

*** note: Weight category data not shown for the following ethnicities: AS=Asian, NA=Native American, OT=Other, AA+CA+LH, AA+LH, AA+OT, CA+LH, CA+NA

Table 16

Distribution of Age by School

Variable Name	Mean	Median	Mode	Min	Max	Std. Dev.	Skew-ness	Kurtosis
Age (N=250)*	16.0	16.0	15.0	14	21	1.4	0.4	-0.4**
High School 1	16.0	16.0	15.0	14	21	1.5	0.5	-0.4
High School 2	16.0	16.0	16.0	14	20	1.4	0.4	-0.5

*note: missing age data for 1 participant
**note: distribution considered to be fairly normal

Table 17

Distribution of Age

Age	N	%
Total Participants *	250	100%
Age 14	35	14%
Age 15	71	28%
Age 16	58	23%
Age 17	37	15%
Age 18	43	17%
Age 19	4	2%
Age 20	1	.4%
Age 21	1	.4%

* note: missing age data for 1 participant

participant did not necessarily correspond with actual days of the week on which exercise took place, but rather an arithmetic sum of the two responses. Fourteen-year olds had the highest mean of 7 days, and 19-year olds had the lowest mean of 3.5 days. The distribution of the physical activity variable was fairly normal. Table 18 also contains the distribution of physical activity by school for each age level.

Table 19 illustrates the distribution of the depression variable, as represented by T-Scores obtained on the RADS-2 screener. The depression data had a slight positive skew, with a mean T-Score of 47.4 which fell within the normal range and below the mean of 50. The mean depression T-Scores for the two schools were almost identical. Girls (M=47.8) had a slightly higher mean T-Score than boys (M=46.8). As shown in Table 20, 10% of all participants scored above the cutoff on the RADS-2 and were referred to a counselor, social worker, or psychologist. High Schools 1 and 2 had very similar referral rates, with 9.8% of High School 1 participants and 10.2% of High School

Table 18

Distribution of Physical Activity

Variable Name	Mean	Median	Mode	Min	Max	Std. Dev.	Skew-ness	Kurtosis
Total Physical Activity (N=251)	6.3	6	7	0	14	3.7	0.3	-0.4*
High School 1	6.5	7.0	7.0	0	14	3.8	0.2	-0.5
High School 2	6.1	6.0	7.0	0	14	3.7	0.4	-0.2
Age 14	7.0	7.0	7.0	1	14	3.5	0.2	-0.5
Age 15	6.4	6.0	7.0	0	14	3.3	0.4	0.2
Age 16	6.9	7.0	7.0	0	14	4.2	0.3	-0.8
Age 17	6.1	7.0	7.0	0	14	3.8	-0.2	-0.6
Age 18	5.1	5.0	2.0	0	14	3.8	0.8	0.2
Age 19	3.5	2.5	2.0	2	7	2.4	1.8	3.1
Age 20	10.0	10.0	10.0	10	10	-----	-----	-----
Age 21	8.0	8.0	8.0	8.0	8.0	-----	-----	-----
High School 1**	6.5	7.0	7.0	0	14	3.8	0.2	-0.5
Age 14	7.1	7.5	4.0	1	13	3.6	-0.1	-0.7
Age 15	6.5	7.0	7.0	0	14	3.0	0.2	0.3
Age 16	7.1	6.5	14	0	14	4.8	0.2	-1.1
Age 17	6.6	7.0	7.0	0	14	4.0	-0.2	-0.5
Age 18	5.6	5.0	5.0	0	14	3.9	0.7	-0.02
High School 2**	6.1	6.0	7.0	0	14	3.7	0.4	-0.2
Age 14	6.8	7.0	7.0	1	14	3.5	0.4	0.1
Age 15	6.3	6.0	4.0	0	14	3.7	0.6	0.2
Age 16	6.7	7.0	7.0	1	14	3.9	0.5	-0.4
Age 17	5.6	6.5	7.0	0	11	3.6	-0.5	-0.8
Age 18	4.5	4.0	2.0	0	14	3.6	0.9	1.0

*note: Distribution considered to be fairly normal

**note: Age data not broken down by school for ages 19-21 due to small number of participants ages 19-21

Table 18 (continued)

Distribution of Physical Activity

Variable Name	Mean	Median	Mode	Min	Max	Std. Dev.	Skew-ness	Kurtosis
High School 1	6.5	7.0	7.0	0	14	3.8	0.2	-0.5
Grade 9	6.8	7	7	0	14	3.2	0.2	-0.1
Grade 10	7.3	7	7	0	14	4.3	0.9	-0.8
Grade 11	6.3	7	4	0	14	4.1	-0.1	-0.8
Grade 12	5.4	5	2	0	14	3.8	0.6	0.1
High School 2	6.1	6.0	7.0	0	14	3.7	0.4	-0.2
Grade 9	6.7	6	7	0	14	3.3	0.8	0.3
Grade 10	6.3	6.5	7	0	14	4.3	0.4	-0.6
Grade 11	5.7	6.5	7	0	14	3.8	0.3	-0.3
Grade 12	5.4	6	2	0	14	3.4	0.4	-0.1

2 participants referred to school mental health service providers. Girls were somewhat more likely to score above the cutoff than boys, as indicated by the 11% referral rate for girls compared to 9% for boys. Interestingly, the rate of students scoring above the cutoff (86^{th} percentile) was lower than that in the standardization sample of the RADS-2.

The distribution of dieting responses appears in Table 21, and these data were positively skewed. The overall mean scaled score of 2.2 fell within the "Mean" range on the Restrained Eating norms for Dutch high school females. The DEBQ manual lists no norms for high school males. Participants at the two high schools had similar mean scaled scores on the DEBQ for dieting behavior, and both fell within the "Mean" range.

Table 22 shows the distribution of binge eating data on the EDI-2 bulimia scale, which

Table 19

Distribution of Depression: RADS-2 T-Score

Variable Name	Mean	Median	Mode	Min	Max	Std. Dev.	Skew-ness	Kurtosis
RADS2 T-Score (N=251)	47.4	46	38	31	74	9.4	0.5*	-0.3*
High School 1	47.3	46	45	31	74	9.4	0.6	-0.2
High School 2	47.4	47	38	31	74	9.4	0.4	-0.4
Female	47.8	47	51	31	74	9.6	0.3	-0.4
Male	46.8	45	45	31	74	9.1	0.7	0.1
Age 14	46.4	45	36	32	64	9.1	0.5	-0.6
Age 15	46.4	45	36	31	74	10.0	0.6	-0.2
Age 16	47.7	47.5	38	31	67	8.7	0	-0.9
Age 17	50.3	50	43	33	73	9.8	0.4	-0.3
Age 18	45.7	44	44	31	74	8.7	1.2	1.8
Age 19	51.8	50.5	49	49	57	3.8	1.3	0.8
Age 20	46.0	46.0	46.0	46	46	-----	-----	-----
Age 21	53.0	53.0	53.0	53.0	53.0	-----	-----	-----
High School 1	47.3	46	45	31	74	9.4	0.6	-0.2
Grade 9	46.6	46	46	31	68	9.3	0.3	-0.7
Grade 10	48.1	45	45	33	69	10	0.5	-0.6
Grade 11	48.1	45.5	38	36	73	9.8	1.1	1.0
Grade 12	47	46.5	39	34	74	9	.9	1.3
High School 2	47.4	47	38	31	74	9.4	0.4	-0.4
Grade 9	45.7	44	36	32	74	9.5	1.1	1
Grade 10	47.8	49	51	31	66	10.1	0	-1
Grade 11	49.8	50	50	37	67	7.6	0.3	-0.1
Grade 12	47.3	45	41	31	69	10	0.6	-0.2

*note: distribution has Slight positive skew

Table 20
Depression Scores Above Cutoff

	Total Participants (N)	Above Cutoff (N)	Above Cutoff (%)
Total Participants	251	25	10%
High School 1	133	13	9.8%
High School 2	118	12	10.2%
Female	141	16	11%
Male	110	10	9%

also was positively skewed. As discussed in Chapter 2, in the present study EDI-2 data are reported using raw scores due to the exclusion of the one purging item in the subscale, so the percentiles in the manual for high school students are inappropriate. Table 23 shows the distribution of responses for Age at First Diet and Age at First Binge, completed by participants who replied "yes" to the questions, "Have you ever experienced a binge eating episode?" and, "Have you ever been on a diet?" Participants responded how many years old they were at those times. Students indicated they first dieted as young as 9 years old, and first binged as young as 10 years old, and the median ages for dieting and bingeing were 14.5 and 14.0, respectively. The mean age for first binge was 9 months earlier at High School 2 (M=13 years, 5 months) than High School 1 (M=14 years, 2 months), and the mean age for first diet was 7 months earlier at High School 1 (M=14 years, 1 month) than High School 2 (M=14 years, 8 months). According to Table 24, 33% of participants responded that they had been on a diet, and Table 25 illustrates that 12% responded they had experienced a bingeing episode. Table 26 further illustrates that 6% reported they had both binged and dieted at some time in the

Table 21

Distribution of Dieting: DEBQ Scaled Score

Variable Name	Mean	Median	Mode	Min	Max	Std. Dev.	Skew-ness	Kurtosis
DEBQ Scaled Score (N=250)*	2.2	1.95	1.0	1.0	14.0	1.3	4.0**	29.6**
High School 1	2.3	2.2	1.0	1.0	14.0	1.4	4.5	35.3
High School 2	2.1	1.9	1.0	1.0	10.0	1.2	2.9	15.4
Age 14	2.5	2.2	2.9	1.0	10.0	1.6	3.1	13.9
Age 15	2.1	1.8	1.0	1.0	14.0	1.6	5.6	40.0
Age 16	2.3	2.2	1.0	1.0	5.0	1.1	0.7	-0.4
Age 17	2.3	2.1	1.0	1.0	5.0	1.0	0.8	-0.1
Age 18	1.9	1.7	1.0	1.0	4.1	0.9	0.7	-0.5
Age 19	1.9	1.6	----	1.0	3.4	1.1	1.0	-0.4
Age 20	3.3	3.3	3.3	3.3	3.3	-----	-----	-----
Age 21	3.1	3.1	3.1	3.1	3.1	-----	-----	-----
High School 1	2.3	2.2	1	1	14	1.4	4.5	35.3
Grade 9	2.4	2.2	1	1	14	1.8	4.9	30.7
Grade 10	2.4	2.6	3	1	4.6	1.1	0.5	-0.5
Grade 11	2.2	2	1	1	4.6	1.1	0.7	-0.6
Grade 12	2.1	2.2	1	1	4.1	0.9	0.4	-1.1
High School 2	2.1	1.9	1	1	10	1.2	2.9	15.4
Grade 9	2.2	1.8	1	1	10	1.6	3.9	19.1
Grade 10	2.2	1.9	1.2	1	5	1.1	0.8	-0.1
Grade 11	2.0	1.9	1.2	1	5	1	1.6	3.3
Grade 12	2.1	1.9	1	1	4.1	1	0.7	-0.4

*note: missing data for one DEBQ score
**note: distribution was leptokurtic with extreme positive skew

Table 22

Distribution of Binge Eating: EDI2 Raw Score

Variable Name	Mean	Median	Mode	Min	Max	Std. Dev.	Skew-ness	Kurtosis
EDI2 Raw Score (N=251)	1.0	0.0	0.0	0	18	2.2	3.6*	18.6*
High School 1	1.1	0	0	1	10	1.9	2.1	4.5
High School 2	1.0	0	0	0	18	2.4	4.4	24.4
Age 14	1.6	1	0	0	6	2.1	1.0	-0.7
Age 15	0.8	0	0	0	10	1.9	3.0	10.0
Age 16	0.6	0	0	0	3	1.0	1.7	1.2
Age 17	1.5	0	0	0	18	3.5	3.6	14.3
Age 18	1.0	0	0	0	11	2.2	3.1	11.3
Age 19	2.0	2	----	0	4	1.8	0	-3.3
Age 20	0	0	0	0	0	-----	-----	-----
Age 21	6.0	6	6	6	6	-----	-----	-----
High School 1	1.1	0	0	1	10	1.9	2.1	4.5
Grade 9	1.5	0	0	0	10	2.3	1.7	2.8
Grade 10	0.5	0	0	0	3	1.0	1.6	1.3
Grade 11	1	0	0	0	6	1.7	2	4.1
Grade 12	1.1	0	0	0	7	1.9	1.9	2.9
High School 2	1.0	0	0	0	18	2.4	4.4	24.4
Grade 9	0.8	0	0	0	6	1.7	2.1	3.3
Grade 10	0.8	0	0	0	11	2.1	4	18.5
Grade 11	1.3	0	0	0	18	3.7	4.3	19.8
Grade 12	1	0	0	0	11	2.3	3.5	14.1

*Note: distribution is leptokurtic with extreme positive skew

Table 23

Distribution of Age at 1st Diet and Age at 1st Binge

Variable Name	Mean	Median	Mode	Min	Max	Std. Dev.	Skew-ness	Kurtosis
AGE at 1st Diet* (N=84)	14.4	14.5	15.0	9	18	1.6	-0.5	0.8
High School 1	14.1	14.0	13.0	9	18	1.8	-0.3	0.8
High School 2	14.7	15.0	15.0	12	17	1.3	-0.3	-0.3
AGE at 1st Binge** (N=26)	13.9	14.0	14.0	10	18	1.9	-0.2	0.1
High School 1	14.2	15.0	15.0	10	18	2.2	-0.7	0.3
High School 2	13.4	13.0	13.0	11.5	6	1.4	0.5	-0.2

* note: Age at 1st Diet distribution showed slight negative skew,

** note: Age at 1st Binge distribution was fairly normal

Table 24

Distribution of Have You Ever Been on a Diet Variable

Reply	N	Percentage
*N	250	100%
No	167	67%
Yes	83	33%
*High School 1:	132	
No (65%)	86	
Yes (35%)	46	
High School 2:	118	
No (69%)	81	
Yes (31%)	37	

*Note: missing data for 1 participant

Table 25

Distribution of Have You Ever Binged Variable

Reply	N	Percentage
*N	250	100%
No	221	88%
Yes	29	12%
High School 1:	133	
No (86%)	115	
Yes (14%)	18	
*High School 2:	117	
No (91%)	106	
Yes (9%)	11	

*Note: missing data for 1 participant

past. The bingeing and dieting data will be further discussed with the results of research question number 3.

Correlation matrices were created to determine the correlation of each continuous variable with other continuous variables: BMI, physical activity, depressive symptoms, dieting behavior, and binge eating behavior. Table 27 shows the correlation matrix of variables across the entire data set, and Tables 28 and 29 show the correlation matrix for Schools 1 and 2, respectively. For the aggregated data, the analysis revealed statistically significant correlations between depression and BMI ($r = .25$, $p = .0001$), between dieting and BMI ($r = .21$, $p = .0007$), between depression and dieting ($r = .20$, $p = .0017$), and between depression and binge eating ($r = .14$, $p = .0228$). The clinical significance of these correlations could be viewed as minimal, however. For example, the correlation of .25 between depression and BMI would explain about 6% ($R^2 = .0625$) of the variance of

Table 26

Distribution of Ever Dieted and Ever Binged

Reply	N	Percentage
N*	249	100%
Have Neither Binged nor Dieted	152	61%
Have Dieted but not Binged	68	27%
Have Binged but not Dieted	14	6%
Have Dieted and Binged	15	6%

*note: missing Ever-Dieted data for one participant, Ever-Binged data for one participant

the two variables. The correlation matrices for the two schools revealed statistically similar relationships between the variables at the two locations, providing some justification for continuing with the path analysis of the aggregated data set. A Box's M

Table 27

Correlation Matrix for Entire Data Set

	BMI	Physical Activity	Depressive Symptoms	Dieting Behavior	Binge Eating Behavior
BMI (N=250)	1.0	.02	.25 p = .0001	.21 p = .0007	-.01
Physical Activity (N=251)		1.0	-.08	.06	-.11
Depressive Symptoms (N=251)			1.0	.20 p = .0017	.14 p = .0228
Dieting Behavior (N=250)				1.0	.07
Binge Eating Behavior (N=251)					1.0

analysis of the two samples, using an alpha level of .05, did show that the populations of the two schools were not identical, chi square(15) = 29.7, p=.0132. However, in a test of homogeneity of within covariance matrices, the natural logs of the determinants of the sample covariance matrices suggested that the differences between the two schools was small (High School 1 natural log = 11.95687; High School 2 natural log = 12.66374). Appropriateness of doing the path analysis was determined by examining whether the assumptions required for path analysis were met by the data. Assumptions for a path analysis include a) normality of variable distributions, as measured by kurtosis (g_1), skewness (g_2), and outliers in the distribution; b) independence, as determined by comparing demographic characteristics of the sample to the schools (e.g., race, gender, age); and c) minimal measurement error. With regard to independence, the sample closely resembled the school populations along demographic variables. The small amount of missing data also supported the independence assumption. Reliability estimates addressed the measurement error assumption and will be discussed in more detail at the end of this chapter. Satisfaction of the first assumption of normal distribution was more difficult since the dieting and bingeing variable distributions were extremely positively skewed. Non-normal variable distributions are known to produce inflated chi-squared values and an increased possibility of a rejection of a true model, and this tendency increases with the extent of the non-normality. Only the dieting and bingeing variables had clearly non-normal distributions, however the chi-square results should be viewed with some level of caution.

Once it was deemed appropriate to proceed with the path analysis, maximum likelihood estimation was used to estimate the model parameters including path

Table 28

Correlation Matrix for High School 1

	BMI	Physical Activity	Depressive Symptoms	Dieting Behavior	Binge Eating Behavior
BMI (N=132)	1.0	.05	.39	.00	.13
Physical Activity (N=133)		1.0	-.06	-.02	-.34
Depressive Symptoms (N=133)			1.0	-.03	.04
Dieting Behavior (N=132)				1.0	.22
Binge Eating Behavior (N=133)					1.0

Table 29

Correlation Matrix for High School 2

	BMI	Physical Activity	Depressive Symptoms	Dieting Behavior	Binge Eating Behavior
BMI	1.0	-.03	.15	.28	-.05
Physical Activity		1.0	.00	.06	-.10
Depressive Symptoms			1.0	.25 p = .0074	.10
Dieting Behavior				1.0	.03
Binge Eating Behavior					1.0

Note: N= 118 for all variables

coefficients and variance estimates. The chi-square (X^2) statistic is a test of the null hypothesis that the model fits the data. The likelihood ratio effectively tests the null hypothesis for goodness of fit of the model, and statistical significance was determined at the p=.05 level. Three alternative fit indices, the RMSEA Estimate, Bentler's Comparative Fit Index (CFI), and McDonald's Centrality Index, also were considered in the path analysis. Figure 5 shows the results of the path analysis, including path coefficients or parameter estimates for each pair of variables in the equation. Table 30 shows the unstandardized parameter estimates and standard error for each path coefficient, as well as the variance and standard error of each exogenous variable, and the covariances among each pair of exogenous variables that had a relationship in MODEL2.

Upon the initial path analysis in SAS, it became necessary to modify the model to remove one of the relationships between the binge eating and dieting variables. The complexity of a two-directional relationship between the variables created impossible negative R squared values in the output. Consequently, as reflected in Figure 5, the weaker of the two relationships was removed to remedy this problem in SAS.

The chi-square results in Figure 5 (chi-square(19) =34.1123, p=.0178, alpha = .05) indicate that the null hypothesis should be rejected and that the model does not fit the data. Because the RMSEA Estimate should be < .05 to be considered indicative of a good fit of the data, the RMSEA Estimate of .0588 also failed to provide support for the model. Bentler's CFI should be larger than .9 in order to indicate a good fit of the model, and since Bentler's CFI was .7533 for the present model it offered no support. McDonald's Centrality (.9678) provides the only support for goodness of fit, with a value

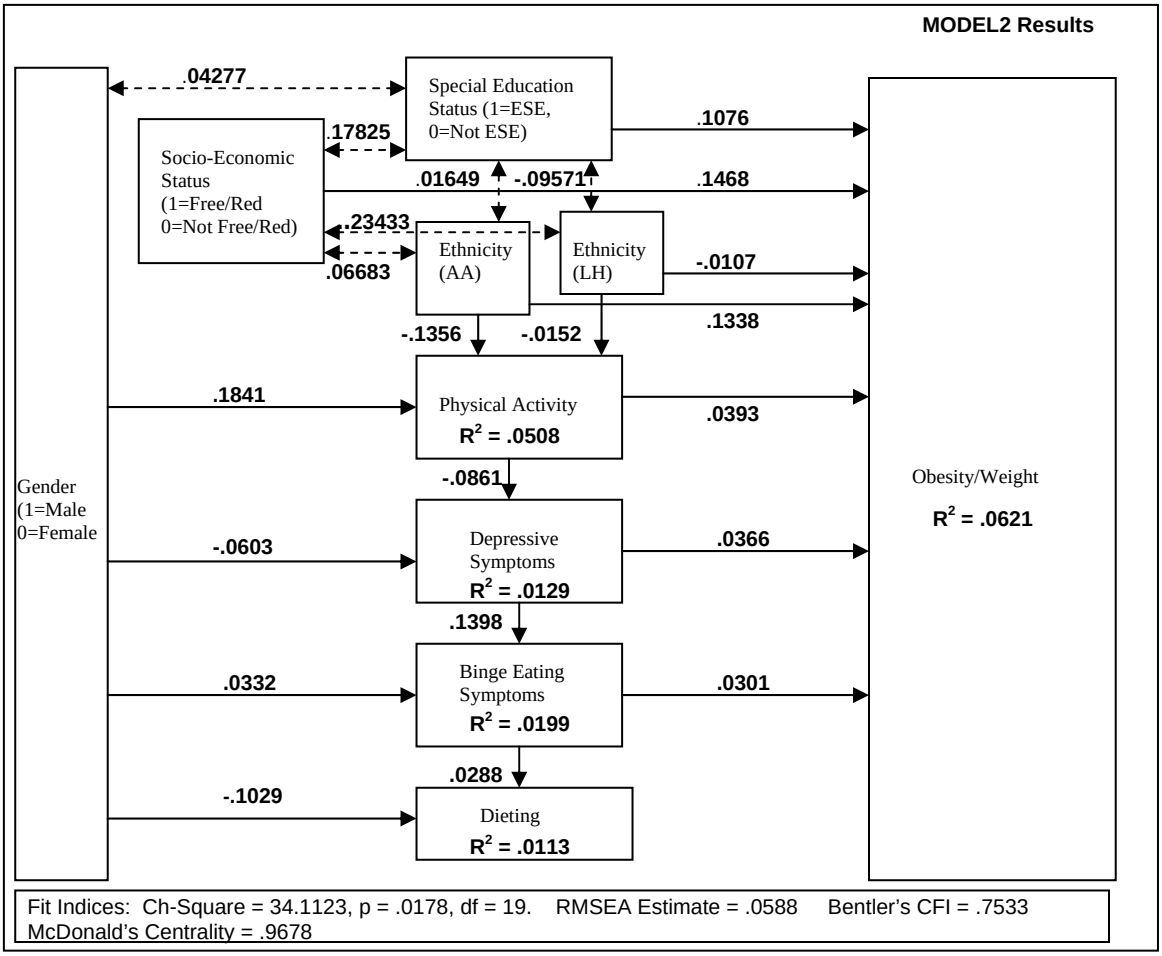

Figure 5. Path Analysis Results: MODEL2.

greater than .9. In summary, the path analysis suggests a poor fit of the model to the data, with only one of the four indices providing support for the null hypothesis.

Figure 5 also shows the R^2 values for each endogenous variable, which indicate the percentage of variance in that variable accounted for by the variables that directly affect it. The R^2 values suggest 6% of the variance in BMI is accounted for by the variables affecting it in the model. Similarly, the R^2 values for physical activity (R^2=.0508), depressive symptoms (R^2=.0129), binge eating symptoms (R^2=.0199), and dieting (R^2=.0113) indicate the percentage of variance of those variables explained by the variables in the model.

Table 30

MODEL2 Unstandardized Parameter Estimates and Standard Errors

Parameter Description	Unstandardized Parameter Estimate	Standard Error
Path Coefficients		
BMI from Physical Activity	.0583	.0961
BMI from Depression	.0216	.0383
BMI from Bingeing	.0775	.1663
BMI from Low SES	1.6255	.7461
BMI from In ESE	1.4257	.8691
BMI from African American	3.3150	1.6065
BMI from Latino-Hispanic	-.2045	1.2749
Physical Activity from Male	1.3712	.4851
Physical Activity from African American	-2.2644	1.0754
Physical Activity from Latino Hispanic	.1957	.8415
Depression from Physical Activity	-.2160	.1670
Depression from Male	-1.1263	1.2432
Dieting from Male	-.2483	.1593
Bingeing from Depression	.0320	.0150
Bingeing from Dieting	.0035	.1157
Variances of Exogenous Variables		
Low SES	.24766	.02309
In ESE	.17278	.01611
Male	.24860	.02248
African American	.04946	.00461
Latino Hispanic	.08300	.00753

Table 30 (continued)

MODEL2 Unstandardized Parameter Estimates and Standard Errors

Parameter Description	Unstandardized Parameter Estimate	Standard Error
Covariances Among Exogenous Variables		
Low SES with In ESE	.03687	.01384
In ESE with Male	.00887	.01331
Low SES with African American	.0074	.00731
In ESE with African American	.00152	.00609
Low SES with Latino Hispanic	.03360	.00959
In ESE with Latino Hispanic	-.01146	.00792
African American with Latino Hispanic	-.00474	.00418

The path analysis also produced standardized path coefficients for each direct effect of a variable on another variable. Path coefficients quantify the amount of change in a dependent variable that corresponds with a one-unit change in the independent variable. The standardized path coefficients in Figure 5 appear above or next to the arrows between variables. The path coefficients range in magnitude from .0113 to .23433. However, given the poor fit of the model to the data, the path coefficients have limited meaning. Overall, the results of the path analysis suggested that the model in Figure 5 was a poor fit for the data.

When it was determined that the model fit the data poorly, an additional path analysis was conducted to determine whether a modified path model might have a better fit with the data. Rather than utilizing statistical methods to identify potential causal paths to add or drop, two relationships were added to the model based on theory and research. Figure

6 shows the results of the final path analysis, with two relationships added to reflect socio-economic status directly influencing physical activity and depressive symptoms. Table 31 shows the unstandardized parameter estimates and standard error for each path coefficient, as well as the variance and standard error of each exogenous variable, and the covariances among each pair of exogenous variables that had a relationship in MODEL3. Once again, the chi-square statistic (chi-square(17) =32.0018, p=.015, alpha = .05) indicated that the null hypothesis should be rejected and that the model did not fit the data. The RMSEA Estimate (.0619), Bentler's CFI (.7551), and McDonald's Centrality (.9681) also provided little support for goodness of fit, leading to a similar conclusion that the model in Figure 6 was a poor fit for the data.

Question 2: For the populations of obese and non-obese adolescents, a) what is the rate of binge-eating symptoms, and b) what is the strength of the relationship between binge-eating and depression?

Table 32 provides one perspective on the first component of this research question. In order to show a definitive rate of binge eating for obese and non-obese students, the Yes/No responses to the question, "Have you ever experienced a binge eating episode?" were compiled using the SAS software. The bulimia subscale of the EDI-2 was not used to determine the rate of binge-eating because no cut-off score defines whether the respondent has binged. As shown in Table 32, 27% of obese participants replied that they had experienced at least one binge-eating episode, compared to 8% of non-obese participants. A chi-square analysis indicated that this difference was statistically significant (chi-square(1)=13.1, p=.0003).

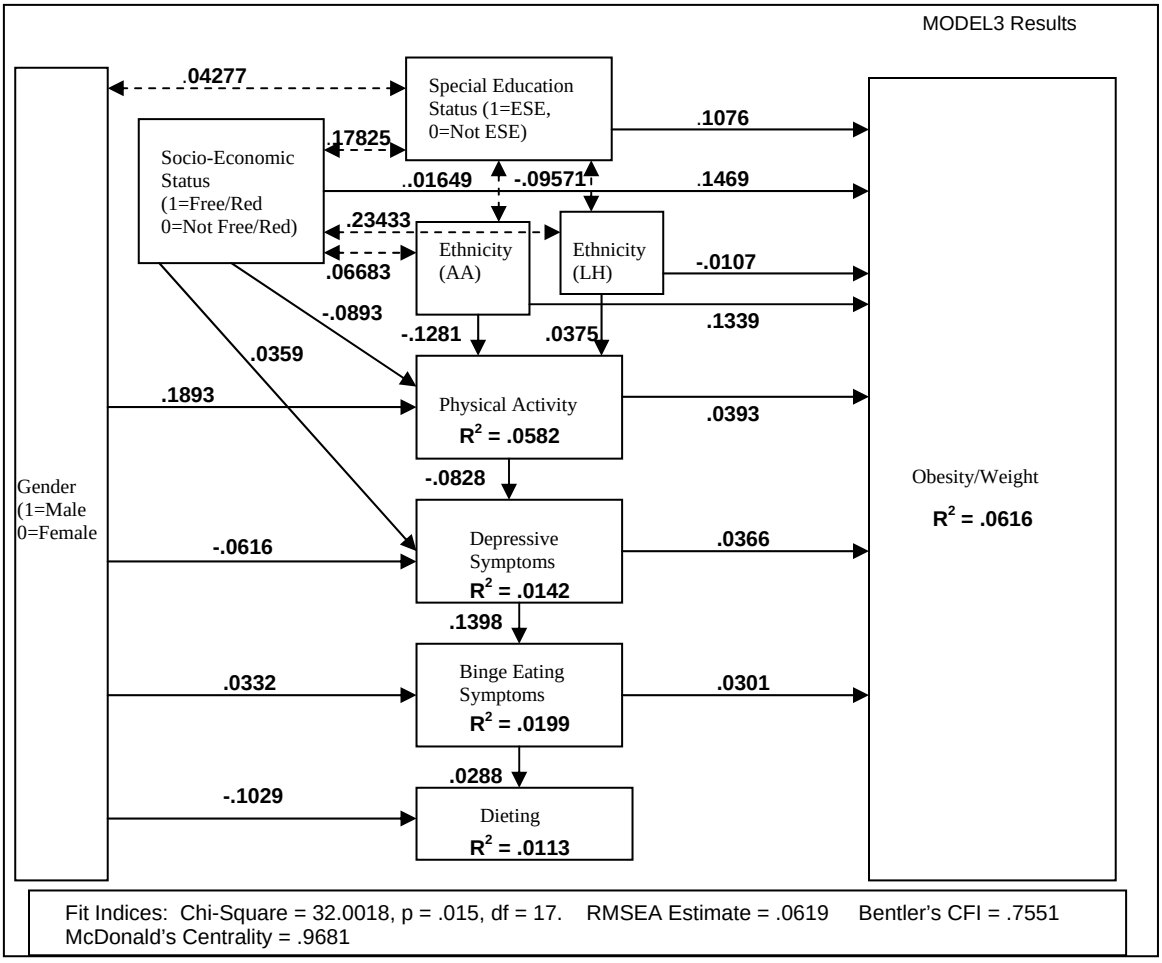

Figure 6. Path Analysis Results: MODEL3.

In addition, as another comparison of binge-eating behavior in obese and non-obese participants, an independent T-test was conducted to determine if there was a statistically significant difference in EDI-2 binge-eating scores between the two groups. A pooled t-test was used since the variances of the binge-eating variable were equal for the two groups (F=1.0, p=1.0). According to results of the pooled t-test [t(248)= -0.03, p=.97], the obese students' level of bingeing on the EDI-2 bingeing scale (M=1.0612, SD=2.1789) was not statistically different from that of non-obese students (M=1.0498, SD=2.1789).

Table 31

MODEL3 Unstandardized Parameter Estimates and Standard Errors

Parameter Description	Unstandardized Parameter Estimate	Standard Error
Path Coefficients		
BMI from Physical Activity	.0583	.0963
BMI from Depression	.0216	.0383
BMI from Bingeing	.0775	.1663
BMI from Low SES	1.6255	.7485
BMI from In ESE	1.4257	.8691
BMI from African American	3.3150	1.6050
BMI from Latino-Hispanic	-.2045	1.2747
Physical Activity from Low SES	-.6661	.4938
Physical Activity from Male	1.4099	.4840
Physical Activity from African American	-2.1392	1.0752
Physical Activity from Latino Hispanic	.4835	.8649
Depression from Physical Activity	-.2077	.1675
Depression from Low SES	.6718	1.2300
Depression from Male	-1.1500	1.2432
Dieting from Bingeing	.0163	.0372
Dieting from Male	-.2499	.1593
Bingeing from Depression	.0321	.0150
Bingeing from Male	.1424	.2804
Variances of Exogenous Variables		
Low SES	.24766	.02309
In ESE	.17278	.01611
Male	.2486	.02248
African American	.04946	.00461
Latino Hispanic	.08300	.00753

Table 31 (continued)

MODEL3 Unstandardized Parameter Estimates and Standard Errors

Parameter Description	Unstandardized Parameter Estimate	Standard Error
Covariances Among Exogenous Variables		
Low SES with In ESE	.03687	.01384
In ESE with Male	.00887	.01331
Low SES with African American	.00740	.00731
In ESE with African American	.00152	.00609
Low SES with Latino Hispanic	.03360	.00959
In ESE with Latino Hispanic	-.01146	.00792
African American with Latino Hispanic	-.00474	.00418

Table 32

Binge-Eating History of Obese and Non-Obese students

	Obese Students (N=49)	Non-Obese Students (N=200)
Answered Y to Ever Binged Question (N=29)	13 (27%)	16 (8%)
Answered N to Ever Binged Question (N=220)	36 (73%)	184 (92%)

The second component of the question was addressed using simple correlation coefficients (Pearson's r) between binge-eating and depressive symptoms for four populations within the sample: obese binge-eaters, obese non-bingers, non-obese bingers, and non-obese non-bingers. Obese groups were defined using the cutoff scores for boys and girls for obesity from the 2000 CDC BMI-for-age growth charts. The RADS-2 Depression Total standard score was used to represent depressive symptoms for analysis. As seen in Table 33, confidence intervals were calculated for all correlation coefficients. With alpha = .05, the data analysis shows a statistically significant relationship between depressive symptoms and binge-eating behavior for all participants (N=251, r=.14, p=.02) and also for non-obese participants (N=201, r=.15, p=.04). Clinical significance of these correlations must be considered, however, with depressive symptoms explaining approximately 2% of the variance in binge-eating for all students (R^2=.0196) and for non-obese students (R^2=.0225). These results are consistent with those obtained on the path analysis for the relationship between depression and binge-eating.

Question 3: For the population of adolescents who engage in both binge-eating and dieting behavior, what is the order of precedence of binge-eating and dieting (i.e., which occurs first, binge-eating or dieting)?

The answer to the third research question required examining the data for participants that report a history of both binge-eating and dieting behavior. Within this population, the age of onset of binge-eating was compared to the age of onset of dieting. These ages were provided on the EDI-2 and DEBQ answer sheets, respectively. As illustrated in Table 26, 6% of all participants, or 15 students, responded that they had

Table 33

Correlation of Binge-Eating and Depressive Symptoms for Obese and Non-Obese Students

	N	Pearson Correlation	p-value	95% Confidence Intervals
All Students *	251	.14	p = .0228	.02 to .26
Obese Students	49	.17	p = 0.2413	-.12 to .43
Non-Obese Students *	201	.15	p = 0.0391	.01 to .28
Obese Bingers	13	-.13	p = 0.6618	-0.63 to 0.45
Obese Non-Bingers	36	.29	p = 0.0859	-0.05 to .56
Non-Obese Bingers	16	.30	p = 0.2643	-.24 to .69
Non-Obese Non-Bingers	184	.10	p = 0.1682	-.04 to .24

* statistically significant, alpha = .05

both dieted and binged. Two of those participants failed to answer the age when bingeing first occurred, leaving an N of 13 for analysis. Table 34 shows that 23% of this group dieted before they binged, 54% binged before they dieted, and 23% dieted and binged for the first time at the same age. Exact 95% confidence intervals indicate that, for example, the true percentage of bingers and dieters in the population who dieted first lies between 19% and 75%, quite a wide range of possibility.

Reliability Estimates on Measurement Instruments

Since reliability of questionnaires may change from one sample to another, the data analysis also included internal consistency reliability estimates, in the form of Cronbach's alpha parameters, for the measurement instruments that have multiple items: the RADS-2 depression screener, the EDI-2 eating disorders inventory, and the DEBQ dieting scale. As shown in Table 35, the depression and dieting scales had the strongest

Table 34

Precedence of Dieting and Bingeing

	N	Percentage	95% Confidence Intervals
Dieted First	3	23%	19% - 75%
Binged First	7	54%	46% - 95%
Dieted and Binged at Same Age	3	23%	19% - 75%
Total Bingers and Dieters	13	100%	100%

Table 35

Reliability Estimates for Measurement Instruments

Measurement Instrument	Standardized Reliability Estimate: Cronbach Coefficient Alpha
*EDI-2 Bingeing	0.69
DEBQ Dieting	0.93
RADS-2 Depression	0.92

*note: Purging item 6 excluded from data analysis

reliability with standardized cronbach's alpha coefficients of .92 and .93. The EDI-2 bulimia scale had a coefficient alpha of .69, considered somewhat low. This scale had fewer items than the DEBQ or RADS-2, and only six out of the seven items were included in the calculation because the purging item was excluded from data analysis. The DEBQ dieting measure had 10 items and the RADS-2 depression screener had 30 items, and their larger number of items most likely influenced their higher reliability.

Chapter V: Discussion

The purpose of this study was to investigate the relationships between obesity and other variables found to be associated with obesity in the literature. This chapter includes a discussion of each research question and the results of the data analysis. The reader also will find the implications of the data and the contribution of this study to the obesity literature. Following the implications discussion, assumptions and limitations of the study are reviewed. Finally this chapter provides suggestions related to future research.

Question 1: To what degree do the data support the proposed model of the correlates of weight in adolescents?

Results of the path analysis indicated a poor fit of the data to the proposed path model. Results for the first research question began with descriptive data for the variables in the path model including BMI, age, physical activity, depressive symptoms, dieting, binge-eating symptoms, age at first diet and first binge, and the questions "Have you ever been on a diet…," and, "Have you ever experienced a binge-eating episode...." These descriptive data will be discussed first, followed by the results of the path analysis.

The BMI data reflected an overall obesity rate of 19% for all participants, higher than the 15% obesity rate for adolescents published in the 1999-2000 NHANES data. If this rate can be generalized to the U.S. population, it would suggest that the obesity rate has continued to rise for adolescents. Males in the study had a higher rate of obesity than females, at 20% vs. 17%. The schools had similar rates of obesity to one another, 18% for High School 1 vs. 19% for High School 2, despite their different demographic

makeup: approximately 25.5% of High School 2 participants were non-white, vs. 10.6% of High School 1 participants. Given the typically higher rate of obesity in African-American and Hispanic/Latino adolescents, it might have been expected that High School 2 would have a higher rate of obesity than High School 1.

Data on the ages of participants indicated a fairly normal distribution, characterized by fewer students at the oldest and youngest ages. Because binge-eating disorder typically has onset in the late teens (American Psychiatric Association, 2000), and there were fewer participants in their late teens, the prevalence of binge-eating symptoms may have been underestimated in this study. Data for the next variable, physical activity, tended to show falling levels of physical activity as the age of the participant increased. Data analysis showed a statistically significant but weak negative correlation between age and physical activity ($r=-0.13$, $p = .04$). This finding could imply that students exercise less as they get older, and is consistent with research that indicates physical activity declines during adolescence (Steinbeck, 2001).

The depression scores for participants in both schools resulted in referrals of 10% of students to a school-based mental health worker. Mental health screening tools are intended to over-identify individuals actually meeting diagnostic criteria for a mental health disorder; however it is not known what percentage of referred students met the DSM-IV-TR criteria for depression. The 10% rate of students scoring above the cutoff for depression risk was only slightly higher than the estimated 8.5% of adolescents nationally who suffer from depression (National Institute of Mental Health, 2000). The referral rate also was lower than the 14% rate found for the normative sample for the RADS-2. Given the reliability estimate in the study of the RADS-2 ($r = .92$), it appears

that the rate of depression in the sample may have been lower than that found in the general population. The sample was demographically representative of the school populations; however it is possible that the students who volunteered for the study tended to have less depressive symptoms than their school peers who did not volunteer for the study.

According to participant responses on their history of dieting and bingeing, one out of three or 33% of students in the study had been on a diet, and one out of eight or 12% had experienced a binge-eating episode in the past. Comparing the 12% bingeing prevalence in the present study to other rates in the research is difficult due to the various definitions of binge-eating that have been measured in other studies (Bulik et al., 2002; Rosenvinge et al., 1999; Ross & Ivis, 1999). The most useful prevalence rates to compare would be those for adolescents meeting the criteria for binge-eating disorder, rather than sub-clinical levels of binge-eating. Prevalence estimates of adolescent binge-eating based on sub-clinical criteria have ranged as high as 18.9% (Ross & Ivis, 1999), while one study that used a stricter set of criteria for adolescent binge-eating disorder found a prevalence of 1% (Rosenvinge et al., 1999). It should also be emphasized that binge-eating disorder onset usually occurs in late adolescence or early 20's, and the sample in this study had fewer participants in their late teens, therefore the prevalence rate may be an underestimate.

The correlation matrices for BMI, physical activity, depression, dieting, and binge-eating showed four statistically significant correlations for the aggregated data set, none for the High School 1 data set, and one for the High School 2 data set. However, the correlations were weak and of questionable clinical value, with the highest correlation

explaining only 6% of the variance in the variables. These weak correlations were consistent with the output of the path analysis, results of which indicated that the data failed to fit the revised path model (MODEL2), or the final path model (MODEL3). While the non-normality of the dieting and bingeing variables compromised the validity of the chi-squared results and increased the likelihood of rejecting a true model, the weak correlations in the correlation matrices substantiate the judgment that the model did not fit the data.

Several factors may have negatively impacted the fit of the model to the data. Although the literature supports the individual relationships between pairs of variables (Berkowitz & Stunkard, 2002; Decaluwe et al., 2002; Goodman & Whitaker, 2002; Price, 2002), there is no existing analysis that looks at the relationships among a constellation of variables around obesity, and it is possible that the model is incorrect. Alternatively, prior to abandoning the current model, it would seem prudent to investigate the influence of sample size on the outcomes. In addition, it is possible that a modified sample selection procedure, targeting participants for which the literature suggests the model relationships hold especially true (i.e., extremely obese or depressed individuals), may yield more promising results. For example, one study focused on adolescents who were diagnosed with depression and found that depression was predictive of binge-eating disorder (Zaider et al., 2002). In addition, obese bingers have been found to have more severe obesity, earlier onset and more frequent dieting, and higher levels of psychopathology including depression (Stunkard, 2002). It should also be noted that one large study on obesity and depression controlled for many of the variables in the present

model and still failed to find a correlation at baseline, although it did find that depression predicted obesity at followup (Goodman & Whitaker, 2002).

Two other factors related to sample selection may have impacted the goodness of fit of the model. The sample for the current study included student athletes whose BMI indicated they were obese but who most likely had a lower level of body fat than indicated by BMI. The study did not exclude students who may have had a biologically-based cause of obesity, such as a thyroid disorder or diabetes. In short, the small size of the sample in the present study and the use of a convenience sample which included primarily non-obese, non-depressed participants without binge-eating problems, may have made it difficult for the path model to perform well in the data analysis.

When interpreting the poor fit of the model to the data, it is important to recollect that the model excluded heritability, the variable long known to have the strongest influence on obesity. The biologically-based factor of heritability has been shown to explain approximately two thirds of the variance in weight (Grilo & Pogue-Geile, 1991; Price, 2002). The path model in the current study excluded heritability due to the extent of the literature already documenting this variable as the most significant factor influencing obesity. Assuming that 66% of the influence on weight has already been accounted for by heredity, it would be reasonable to expect the model to account for no more than a total of about 30% of the variance, so that weight would show R^2 less than or equal to .30. However, the current path model accounts only for 6% ($R^2 = .06$) of the variance in weight. Combined with the 66% estimated influence of heredity, the variables would explain approximately 72% of the variance in weight in the sample.

As previously suggested, hypotheses for the failure of the model to explain a higher percentage of the variance in weight fall into two categories: a) the model is correct but the sample size was too small with selection criteria not sufficiently focused on significantly obese and depressed adolescents, and b) the model is incorrect. Future research should continue to explore the current model. Subsequently, if the model ultimately is rejected then future studies might improve on the model by incorporating additional variables such as parent weight, consumption of sugar-sweetened beverages (i.e., soft drinks), or a combined variable for gender and ethnicity. These and other suggestions for future research are discussed in a later section.

Question 2: For the populations of obese and non-obese adolescents, a) what is the rate of binge-eating symptoms, and b) what is the strength of the relationship between binge-eating and depression?

The hypotheses for research question 2 proposed: (a) similar levels of binge-eating in obese vs. non-obese groups, and (b) a positive correlation between binge-eating and depression. The data provided limited support for hypothesis (a), and no support for hypothesis (b). With regard to the levels of binge-eating for obese and non-obese groups, this part of the question was addressed in two ways: comparing the responses to the Yes/No question on bingeing ("Have you ever experienced a binge-eating episode?"), and comparing the EDI-2 scores for the two groups. The first comparison failed to support the hypothesis that the groups did not differ in bingeing behavior, while the second comparison did support the hypothesis. In the first analysis, there was a statistically significant difference between the obese and non-obese groups in responses to the Ever-Binged question, with obese participants (27%) more than three times as

likely as non-obese participants (8%) to report a previous bingeing experience. These findings are consistent with previous research which has placed the rate of binge-eating behavior at 36.5% for obese adolescents (Decaluwe et al., 2002), although a lower rate of 6.1% was found for obese adolescents meeting the criteria for binge-eating disorder. These rates compare to previously estimated rates of binge-eating in the overall population ranging from 1% for adolescents meeting criteria for binge-eating disorder (Rosenvinge et al., 1999) to 18.9% for adolescents who engaged in binge-eating at least once in the past year (Ross & Ivis, 1999).

In the next assessment related to hypothesis (a), which involved the comparison of EDI-2 bingeing responses between obese and non-obese groups, no significant difference in EDI-2 responses was detected between the two groups. This analysis supported the hypothesis that similar levels of binge-eating would be found in obese vs. non-obese groups. The data appear to contradict each other given the statistically significant difference found between the groups on the Ever-Binged question and the lack of a significant difference between the groups on the EDI-2 bingeing questions. This discrepancy could be explained by the explicit and implicit time frames of the items: the Yes/No question pertained to whether a bingeing episode had occurred at any time in the student's past, however most of the EDI-2 items utilize present-tense wording (i.e., "I stuff myself with food.").

In summary, the results for research question 2(a) could be interpreted as follows: obese students in the sample were more likely than non-obese students to have experienced bingeing in the past, but no more likely than non-obese students to be currently experiencing binge-eating symptoms. It is important to note that neither the

Ever-Binged question or the EDI-2 scale is sufficient to make a diagnosis of binge-eating disorder, criteria for which include at least two bingeing episodes a week for six months. Accordingly, the rate of obese participants who met the criteria for binge-eating disorder would be lower than the rate who simply confirms at least one prior bingeing episode. It would be expected that the group of adolescents who has either binged in the past or engages in bingeing currently, will have different needs than non-bingeing obese adolescents in terms of recommended obesity interventions.

The hypothesis for Research question 2(a) was based upon prior research findings that the majority of binge-eaters are not obese, such as one study that found 12% of women with binge-eating disorder were obese (Kinzl et al., 1997). In the present study, a much higher proportion of binge-eaters was obese: 13 students or approximately 45% of the 29 participants who answered "Yes" to the Ever-Binged question were obese. In addition, it is important to understand and conceptually differentiate the history of binge-eating experience for obese individuals, 27% in this study, from the proportion of all binge-eaters made up by obese individuals, or 45% in this study.

Results pertaining to hypothesis (b), the relationship between binge-eating and depression, suggested a small level of statistical significance for the overall sample and for the non-obese group, with little clinical significance for either group, despite being the groups with the largest N. No other groups had statistical significance. It is possible that the relationship between binge-eating and depression is one that is better detected over time in a longitudinal study, similar in design to previous research which has found depression to predict binge-eating (Zaider et al., 2002). For example, in the present study, participants reported current symptoms for both binge-eating and depression.

Consequently if a student previously experienced symptoms of depression which then faded and were replaced by current binge-eating symptoms, that student's data would appear low in depression and high in binge-eating. Similarly, if a student with a high level of depressive symptoms had participated in the study, and then later the depressive symptoms disappeared and binge-eating behavior increased, the data also would not reflect this scenario.

Question 3: For the population of adolescents who engage in both binge-eating and dieting behavior, what is the order of precedence of binge-eating and dieting (i.e., which occurs first, binge-eating or dieting)?

It was expected that a higher percentage of students who had both dieted and binged would report having dieted before they had their first bingeing experience. The bingeing and dieting data did not support this hypothesis. The research is somewhat mixed on the order of precedence for bingeing and dieting, and whether or not dieting is a risk factor for binge-eating disorder (Santonastaso et al., 1999; Stunkard, 2002). Dieting is accepted as a risk factor for bulimia nervosa and anorexia nervosa, and this provided the basis for the hypothesis that dieting would precede bingeing behavior in the majority of cases. However, dieting has not been firmly established as a risk factor for binge-eating disorder (Stunkard, 2002). Possible reasons for this mixed view could be an insufficient amount of research to show the directionality of the relationship, or that there is no true rule that determines which behavior usually occurs first. It is conceivable that there is no rule that determines which behavior is a risk factor for the other, and that the order of precedence varies with the individual, or that perhaps there is another, stronger risk factor that predisposes an adolescent to binge-eating behavior rather than dieting.

If the results of the present study could be generalized to the population of adolescents who have both binged and purged, they would support the contention that bingeing is more often a risk factor for dieting than the other way around. However, given the small number of participants who reported both dieting and bingeing experiences, it is difficult to generalize these results to the population. The wide confidence intervals shown in Table 34 underscore this problem, suggesting, for example, a 95% likelihood that the true percentage of dieters/bingers who binged first ranges anywhere from 46% to 95%. Even at the low end of the confidence interval, these results could suggest that some adolescents may begin bingeing behavior and then initiate dieting as a compensatory behavior, while others begin dieting first and then begin bingeing as a reaction to the restrictiveness of dieting.

Implications

Perhaps the most critical implication that may be drawn from this investigation is that, if the results of this study can be generalized to the population of American adolescents, the rate of obesity in adolescents continues to rise. In this study, 19% of all participants were classified as obese and 20% as overweight, with a total of 39% of adolescents either overweight or obese. These rates are based upon cutoff scores published in 2000, which originally by definition identified only the highest 5% of adolescent BMI's as obese. Given the continual increase in the rate of obesity and the many negative health and psycho-social outcomes of adolescent and adult obesity, prevention and treatment should continue to be a priority in our society. Similarly, research to enlighten obesity prevention and treatment should continue to be paramount. Another implication of this study concerns the finding that 12% of adolescents had

experienced a binge-eating episode, 33% had been on a diet, and some children started dieting and binge-eating as young as 9 and 10 years of age. Results of this study also suggest that a sizeable portion of students who report bingeing behavior are not obese.

The results of the path analysis indicate that there is much still to be understood about the non-hereditary factors that contribute to obesity. It remains likely that the rise in obesity in the United States and elsewhere stems from some form of gene-environment interaction which has taken shape in the last several decades of history, with culturally determined lifestyle changes playing a key role. Many causal theories and intervention programs are discussed in the popular press and in the research literature, however the rates of child and adolescent obesity continue to rise unabated. It is understandable that institutions and individuals urgently desire to implement solutions to the obesity epidemic, given the serious medical and psycho-social outcomes of obesity. However, the results of the current study highlight the importance of understanding the forces at work behind the obesity puzzle before implementing effective, research-based interventions. In fact, one could argue that the lack of understanding of precisely what has caused the rise in obesity explains why there has been so little success in controlling or reversing it. In this context of urgency, the value of action research which could be conducted in the context of intervention, might be paramount.

School psychologists and others involved in the education of children must be mindful that obesity is not an individual problem but a societal one, and an obese child or adolescent should not be assigned the "blame" for his or her obesity or be the subject of negative stereotypes or discrimination. Even in the absence of clear data on the causal factors of obesity, the results of this study point to the importance of research-based

interventions that teach positive messages about nutrition, health, and body image to children at home and at school. These are messages that can be conveyed to children by parents, teachers, guidance counselors, school psychologists, and school nurses. For example, mental health professionals can work with health professionals to design and implement primary, secondary, and tertiary prevention and intervention programs for obesity. Again, action research on effectiveness of these programs can be conducted to contribute to the knowledge base on obesity. Action research programs could address motivating children and adolescents to initiate lifestyle changes in areas that have individually been found to influence obesity, such as physical activity and time spent on sedentary activities.

Assumptions and Limitations

Several limitations related to reliability and validity are evident for the present study. For example, the test-retest reliability of physical activity self-report items on the Demographic Physical Activity Questionnaire (DPAQ), as estimated by Brener et al. (1999), was quite low at .55. However, it is unclear what time interval was used in this assessment, and it would be expected that reliability would decrease as the time interval increases due to the participants' changing habits and behavior. In addition, the short length of the bulimia scale of the EDI-2, with only five items, would appear to contribute to lower reliability for the binge-eating variable. Although previous research estimated the reliability for the five bingeing items at .82, the internal consistency reliability calculated in the current study was lower at .69. In addition, the EDI-2 test manual cautions that although studies have used the bulimia scale in isolation, validity can not be assumed if a subscale is used in isolation as the bulimia scale was used in the present

study. Another concern relates to the bingeing and dieting history questions, "Have you ever been on a diet…?" and, "Have you ever experienced a binge eating episode…?" No validity or reliability data exist on these questions which require Yes/No answers.

Another limitation related to validity of BMI should be noted. No secondary measurement of body fat was used for student athletes whose BMI fell within the obese range. Consequently, some students with a high level of muscle mass may have been identified in error as obese. Ideally, a nurse should provide a backup or secondary measurement of body fat, such as skin fold thickness, to avoid wrongly identifying students who engage in rigorous exercise as obese. This limitation of BMI as a measurement tool is common in obesity research and is considered to be outweighed by its convenience.

One final limitation regarding reliability concerns the socio-economic status variable and the indicator for free or reduced lunch. This study used the indicator for free or reduced lunch eligibility to determine socio-economic status. Although students who participate in the free and reduced lunch plan must apply and qualify for the program, there is no requirement that students do this. Thus, it is conceivable that some participants may be of low SES but not be taking advantage of the free and reduced lunch program, resulting in an underestimate of those from low SES backgrounds. However, 40% of High School 1 students and 49% of High School 2 students receive free or reduced lunch, suggesting that many students are participating in the program.

Other limitations of the study relate to the sample. First, the recruitment methods at the two schools differed due to requirements of the school administration, suggesting that different types of students may have been recruited at the two schools. This concern

was addressed by statistically comparing the two school samples, as discussed previously in Chapter 4, and while the groups were not statistically identical, results suggested minor differences in the groups. Another limitation of the study concerns sample size. The number of participants in the study was 251. A sample size of 200 is considered by some to be relatively small for a structural equation modeling study (Chou & Bentler, 1995). A smaller sample size means that power will be lower and it will therefore be more difficult to reject the null hypothesis. However, offsetting this decreased power to some extent is the reliability and validity of the measurement tools for depression (RADS-2) and dieting (DEBQ). Another limitation with regard to the sample is the fact that all participants are from a single school district and state, which limits external validity of any results and the ability to generalize results to the larger population of American adolescents. In addition, the demographic makeup of both high schools is less diverse than for the state as a whole, limiting external validity of the findings. Another limitation related to the sample relates to the opinion by some researchers that individuals with eating disorders are underrepresented in groups that participate in eating disorder studies (Wilson et al., 1996). If students with eating disorders chose not to participate in the present study, the sample would be biased and consequently the data would be less than representative of the high school population. Finally, the sample by definition did not include students who had dropped out of school. To combat these limitations, students were recruited in a variety of ways and incentives were provided for participation. In addition, some participants were recruited from the performance-based diploma program and were not enrolled in the regular high school program.

An additional limitation of the study concerns the self-report measures used to assess depression, dieting, bingeing, and physical activity. The most accurate assessment of both depression and binge-eating would be through individual diagnostic interview, rather than through a questionnaire. As in any study utilizing self-report measures rather than direct assessment of the variable, there is an inherent risk that participants are not truthful or results will not be valid or reliable. These concerns were mitigated by the procedures documented and explained to participants, the presence of the investigator during completion of questionnaires, and the ordering of the questionnaires within each packet.

In another limitation of the study, the key assumption in the path analysis is the fact that heredity was excluded from the model. As a result, as much as 66% of the variance in weight was unexplained by the model at the outset. One additional caution on interpreting the results of the path analysis should be mentioned. Although it is clear that the relationships in the path model are directional and significance may have been found in the data analysis, we must keep in mind that the predictor variables were not manipulated and the study was not an experimental one. Therefore, had there been significant results of the path analysis, we would be careful with statements of causality and limit any interpretations to correlational discussions.

Directions for Future Research

An opportunity remains for future research to better understand the relationships among the variables that influence weight and obesity. Ideally future research will begin with further investigation of the viability of the current path model by using a modified sample selection process and a larger sample. Many of the research studies that provided

the basis for the model focused upon participants who were obese (Decaluwe et al, 2002; Stunkard, 2002), depressed (Zaider et al., 2002), or had binge-eating disorder (Santonastaso et al, 1999). Similarly, future investigation into the current path model should focus on participants who are significantly obese, have clinical depression, and/or have binge-eating disorder. Such a sample population may be easier and more appropriate to recruit through pediatricians or clinics than through the school setting. The sample selection process would also exclude participants who had a medical cause of obesity.

Future path analysis research utilizing a much larger sample size would provide greater power and opportunity for statistical significance. A larger sample size would allow the inclusion of the gender/ethnicity variable in the model rather than breaking them into two variables. In addition, future research should attempt to mirror the general population in ethnic diversity, to provide the most useful data and allow generalization to the population.

In order to avoid having athletes incorrectly identified as obese, an alternate, direct method of body fat analysis should be employed such as skin fold thickness, whereby individuals with a high BMI would be directed to the secondary measurement station. Alternatively, the direct method of body fat measurement could be used for all participants. In addition, future researchers could consider other means of measuring the physical activity and bingeing behaviors. Actual physical activity might be tracked by a participant on a daily log, or with a pedometer worn each day. Binge eating could be assessed directly by interview as a followup to the bulimia scale of the EDI-2 or other screening tool.

In the event that future research cannot validate the current path model even with the use of a larger sample and modified sample selection process, changes to the path model should be considered. For example, a path model that includes parent weight as one of the variables might be found to have significant results, given the known impact of heredity on weight and obesity (Grilo & Pogue-Guile, 1991; Price, 2002). Heredity also bears influence upon several other variables in the current model, therefore its inclusion may produce a model with a better fit. Parent depression and parent binge-eating might be considered as additional variables since heredity is considered a risk factor for depression (Smith et al., 2003) and binge-eating (Bulik et al., 2002). Future research might consider inclusion of soft drink consumption as a variable in the path model. Authors of a recent review of 30 experimental and epidemiological studies, conducted between 1966 and 2005, concluded that greater intake of sugar-sweetened beverages, particularly carbonated soft drinks, is associated with weight gain and obesity in both children and adults (Malik, Schulze, & Hu, 2006). Time spent watching television and playing video games represents another variable that might be considered for a future path analysis. These sedentary activities can be viewed as types of physical inactivity, and they have become integral to American lifestyle over the last several decades. Many researchers have linked them in theory to the rise in child and adolescent obesity (Stunkard, 2002), and have promoted reductions of child sedentary activities (Steinbeck, 2001).

Obesity research in general, and path analysis research in particular, remain important and could shed light on the interrelationships of variables contributing to the continuing upsurge in obesity in adolescents. Genetic research should continue, along

with recently emerging lines of research in possible biochemical and viral causes of obesity. Heredity alone cannot explain the increase in obesity in the last 20 years, and further research can help to uncover the combination of influences that has led to the obesity epidemic. Such understanding is critical in order to properly design effective programs for prevention and treatment. The current lack of understanding of the causes of obesity, and the tendency to blame each individual, have allowed the obesity problem to progress to now epidemic proportions.

Conclusion

In conclusion, this study investigated the relationships between variables that have been found to influence weight and obesity. Results indicate that the proposed path model and its revisions were a poor fit for the data. The level of binge eating reported by groups of obese and non-obese students was similar, however a higher percentage of obese students than non-obese students reported a previous binge eating experience. For participants in the study who reported a history of both dieting and binge eating, onset of bingeing preceded onset of dieting more often than dieting preceded bingeing. However, the wide confidence intervals and the small number of participants with a history of both dieting and bingeing prevent generalizing this result to the U.S. adolescent population. In the pursuit of effective prevention and treatment interventions, future academic research and action research in the schools should continue to investigate the causes and the relationships between the variables that work together to produce the country's epidemic of obesity.

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Appendices

Appendix A

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Dear Parent:

The following information is to help you decide whether or not you want to allow your teenager to be a part of a low risk research study. Please read this carefully. If you do not understand anything, ask the person in charge of the study: **Angela T. Sheble**. The title of the research study is **Correlates of Weight in Adolescents: A Path Analysis**. The study will be done at 2 high schools. Other high schools also may be included in the study if necessary. Your teenager is being asked to participate because he/she attends one of these schools. The study will have about 400 participants altogether.

The purpose of this research study is to learn more about the things that may have to do with weight in adolescents, like physical activity, eating habits, depression, dieting, socio-economic status, special education, gender, and ethnicity/race. The study will take place from **January through May, 2005**. Your teenager will visit the clinic **one time for 30-40 minutes,** during one class period**,** on a Friday, to participate. During this visit, your teenager will be asked to complete 4 short questionnaires on dieting, depression, eating habits, and physical activity, and he/she will have their height, weight, and Body Mass Index (BMI) measured. Your teenager will also be entered in a **free raffle** to win a gift for participating. **Raffle prizes** may include gift certificates for items like **CD's, clothing, movies, and restaurants**. You and your teenager will not be paid for participation in this study. However, you and/or your teenager may benefit by learning his/her Body Mass Index. Also, your teenager will be entered in a raffle. There is no known risk to your teenager from being a part of this research study.

You and your teenager's privacy and research records will be kept **confidential** to the full extent required by law. Authorized research personnel, employees of the Department of Health and Human Services, and the USF Institutional Review Board and its staff and any other individuals acting on behalf of USF, may inspect the records from this research project. If your teenager's depression questionnaire indicates that he or she may be depressed, you will be notified promptly by a school professional such as a counselor, social worker, or psychologist. The results of this study may be published. However, the data obtained from your teenager will be combined with data from other teenagers in the publication. The published results will not include your teenager's name or any other information that would personally identify your teenager in any way. Packet ID numbers will be used, so your teenager's name will not appear on the questionnaires or in the computer data. Only the person in charge of the study will have access to the packet ID numbers and names.

Allowing your teenager to participate in this research study is voluntary. You are free to allow your teenager to participate in this research study or to withdraw him/her at any time. If you choose not to allow your teenager to participate or if you remove your teenager from the study, there will be no penalty. Your decision to allow your teenager to participate (or not to allow your teenager to participate) will in no way affect his/her status at school.

If you have any questions about this research study, contact **Angela T. Sheble**. If you have questions about your rights as a person who is taking part in a research study, you may contact the **Division of Research Compliance of the University of South Florida at (813) 974-5638**.

Thank you in advance for allowing your teenager to participate in this research study. Please sign the Parent Consent form on page 2. Your teenager will also be asked to sign (at school) the Child's Assent form on page 3.

Sincerely,

Angela T. Sheble, Ed.S.

Consent for Child to Take Part in this Research Study

I freely give my consent to let my child take part in this study. I understand that this is research. I have received a copy of this consent form.

_____	_____	_____
Signature of Parent of child taking part in study	Printed Name of Parent	Date

Investigator Statement

I certify that participants have been provided with an informed consent form that has been approved by the University of South Florida's Institutional Review Board and that explains the nature, demands, risks, and benefits involved in participating in this study. I further certify that a phone number has been provided in the event of additional questions.

_____	Angela T. Sheble, Ed.S. _____	
Signature of Investigator	Printed name of Investigator	Date

Child's Assent Statement

Angela Sheble has explained to me this research study called Correlates of Weight in Adolescents: A Path Analysis.
I agree to take part in this study.

_____	_____	_____
Signature of Child taking part in study	Printed Name of Child	Date
_____	Angela T. Sheble, Ed.S.	_____
Signature of person obtaining consent	Printed name of person obtaining consent	Date
_____	_____	_____
[Optional] Signature of Witness	Printed Name of Witness	Date

If child is unable to give assent, please explain the reasons here:

_____	Angela T. Sheble, Ed.S	_____
Signature of person obtaining consent	Printed name of person obtaining consent	Date
_____	_____	_____
[Optional] Signature of Witness	Printed Name of Witness	Date

El espacio abajo es para el cuño de la Junta de Revisión Institucional- Por favor de dejar en blanco

Estimados Padres:

La siguiente información es para ayudarles a decidir si usted quiere o no dejar que su adolescente participe en un estudio de investigación de bajo riesgo. Por favor lea esto cuidadosamente. Si usted no entiende nada, por favor pregúntele a la persona encargada de este estudio: **Angela T. Sheble**. El titulo de este estudio es **Factores Relacionados con el Peso en los Adolescentes.** El estudio será hecho en 2 escuelas superiores. Otras escuelas superiores podrán ser incluidas también si hay necesidad. Su adolescente ha sido elegido a participar porque el o ella van a esta escuela. Este estudio tendrá alrededor de 400 participantes en total.

El propósito de este estudio de investigación es para saber mas acerca de las cosas que pueden tener que ver en el peso de los adolescentes como actividad física, hábitos alimenticios (lo que comen), estado de animo (depresión), dieta, clase socio-económica, educación especial, sexo, y raza. El estudio se llevara a cabo de **Enero a Mayo del 2005**. Su adolescente visitara la clínica **una vez por 30-40 minutos** un Viernes durante un periodo de clase para participar. Durante esta visita, su adolescente tendrá que llenar 4 cuestionarios cortos sobre dieta, depresión, lo que comen, y actividad física. También se obtendrá la estatura, el peso, y se le medirá el índice de la masa del cuerpo. Su adolescente podrá participar en una **rifa gratis** para poder ganarse premios. Estos pueden incluir **discos compactos, ropa, entradas para el cine, y restaurantes**. Su adolescente no recibirá pago por participar en este estudio, pero se podrá beneficiar sabiendo su índice de masa y el poder participar en la rifa. Que se sepa, no hay ningún riesgo en que su adolescente participe en este estudio.

La privacidad de usted y la de su adolescente además de los datos obtenidos en este estudio serán todos confidenciales según lo requiere la ley. El personal investigativo autorizado, empleados del Departamento de Salud y Servicios Humanos y la Junta de Revisión Institucional de La Universidad del Sur de la Florida y sus empleados u otras personas representando a la Universidad, pueden inspeccionar los datos de este estudio investigativo. Si el resultado del cuestionario de depresión de su adolescente indica que el o ella puede estar deprimido, usted será notificado rápidamente por un profesional de la escuela como un consejero, trabajador social o sicologo. Los resultados de este estudio podrán ser publicados pero, los datos obtenidos de su adolescente serán combinados con los datos de los otros participantes. Los resultados publicados no incluirán el nombre de su adolescente o cualquier otra información que pueda identificar a la persona en alguna forma. Un numero de identificación será usado y de esa forma, el nombre de su adolescente no aparecerá en le cuestionario ni en los datos de computadora. Solamente la persona encargada de este estudio tendrá acceso al numero de identificación y a los nombres de los participantes.

Dejar que su adolescente participe en este estudio es voluntario. Usted esta libre de permitir de que su adolescente participe en este estudio o puede dejar de participar en cualquier momento. Si usted decide que su adolescente no participe o si usted quita a su adolescente de este estudio, no habrá ningún tipo de penalidad. Su decisión de participar o no participar no afectaran a el o ella en su escuela.

Si tiene alguna pregunta acerca de este estudio, favor de ponerse en contacto con **Angela T. Sheble.** Si usted tiene preguntas relacionadas con sus derechos como persona quien esta tomando parte en un estudio de investigación, puede llamar a La División de Cumplimiento de Investigación de La Universidad del Sur de la Florida a (813) 974-5638.

Muchas gracias por dejar que su adolescente participe en este estudio. Por favor firme la forma de Permiso de Padres en la página 3. Su adolescente tendrá que firmar también en la escuela la forma de Consentimiento de Hijo que aparece en la pagina 4.

Sinceramente,

Angela T. Sheble, Ed.S.

Permiso para que su hijo o hija tome parte en este estudio de investigación

Yo doy mí consentimiento libremente para que mí hijo o hija tome parte en este estudio. Yo entiendo que esto es un estudio de investigación. Yo he recibido una copia de esta forma de permiso o consentimiento.

_____	_____	_____
Firma del padre del hijo/hija que toma parte en este estudio	Nombre del Padre en letra de molde	Fecha

Declaración del Investigador

Yo certifico que los participantes has sido proveídos con la forma de permiso la cual ha sido aprobada por Junta de Revisión Institucional de La Universidad del Sur de la Florida y explica este estudio incluyendo las demandas, riesgos, y beneficios relacionados con este estudio. Yo certifico además que un numero de teléfono ha sido dado en caso de que haya preguntas adicionales.

_____	Angela T. Sheble, Ed.S.	_____
Firma del Investigador	Nombre del Investigador	Fecha

Declaración de Consentimiento del Adolescente Participante

Angela Sheble me ha explicado este estudio llamado Factores Relacionados con el Peso en los Adolescentes.

Yo estoy de acuerdo en participar en este estudio.

_____	_____	_____
Firma del Adolescente que toma parte en este estudio	Nombre del Adolescente en letra de molde	Fecha
_____	Angela T. Sheble, Ed.S.	_____
Firma de la persona que obtiene el consentimiento	Nombre de la persona que obtiene el consentimiento	Fecha
_____	_____	_____
[Opcional] Firma del Testigo	Nombre del Testigo en letra de molde	Fecha

Si el adolescente no puede dar su consentimiento, por favor explique las razones aquí:

_____	Angela T. Sheble, Ed.S	_____
Firma de la persona que obtiene el consentimiento	Nombre de la persona que obtiene el consentimiento	Fecha
_____	_____	_____
[Opcional] Firma del Testigo	Nombre del Testigo en letra de molde	Fecha

Appendix B

Demographic and Physical Activity Questionnaire (DPAQ)

For School Use:

Survey Number: _____	BMI:_____	SES:_____	ESE:_____

Demographic and Physical Activity Questionnaire (DPAQ)

Part I. Student Information: Please provide the following information.

1. Sex ☐ Male ☐ Female	**3. What grade are you in?** ☐ 9th ☐ 10th ☐ 11th ☐ 12th
2. Ethnicity: Do you think of yourself as…? __ African American/Black __ Asian __ Caucasian/White __ Latino/Hispanic __ Native American/Alaskan __ Other:_____	**4. How old are you?** ☐ 13 ☐ 14 ☐ 15 ☐ 16 ☐ 17 ☐ 18 ☐ 19 ☐ 20

Part II. Physical Activity: The following questions are about physical activity. Circle only one answer for each question.

1. On how many of the past **7 days** did you exercise or participate in physical activities for at least **20 minutes that made you sweat and breathe hard,** such as basketball, jogging, swimming laps, tennis, fast bicycling or similar aerobic activities?

0 days	1 day	2 days	3 days	4 days	5 days	6 days	7 days

2. On how many of the past **7 days** did you participate in physical activity for at least **30 minutes that did not make you sweat or breathe hard**, such as fast walking, slow bicycling, skating, pushing a lawn mower, or mopping floors?

0 days	1 day	2 days	3 days	4 days	5 days	6 days	7 days

3. On how many of the past **7 days** did you do exercises to strengthen or tone your muscles, such as push-ups, sit-ups, or weight lifting?

0 days	1 day	2 days	3 days	4 days	5 days	6 days	7 days

4. On how many of the past **7 days** did you participate in at least 20 minutes of physical activity outside the normal school day?

0 days	1 day	2 days	3 days	4 days	5 days	6 days	7 days

5. In an average **week** when you are in school (Monday-Friday), on how many **days** do you go to physical education (PE) classes?

0 days	1 day	2 days	3 days	4 days	5 days

6. During an average physical education (PE) **class**, how many **minutes** do you spend actually exercising or playing sports?

I do not take PE	Less than 10 minutes	10 to 20 minutes	21 to 30 minutes	More than 30 minutes

7. In an average **school day** (Monday-Friday), how many **hours** do you spend doing any of the following activities: watching television/movies, playing video games, reading, homework, or on the computer/Internet?

0 (No time on these activities)	Less than 1 hour per day	1 hour per day	2 hours per day	3 hours per day	4 hours per day	5 or more hours per day

About the Author

Angela T. Sheble received her B.S. in Commerce in 1983 from the University of Virginia in Charlottesville, Virginia. She worked for 18 years in the computer software industry in various management, consulting, and technical capacities, in both the private and public sector. Ms. Sheble entered the University of South Florida's School Psychology doctoral program in 2000 where she chose pediatric school psychology as her area of emphasis. She earned her Master's degree in 2001 and her Education Specialist degree in 2004. Ms. Sheble won the Florida Association of School Psychologists Graduate Studies Award in 2001, and the University Involvement Award in 2003 from the Tampa Bay Academy of Hope. As part of her clinical training, Ms. Sheble worked in early intervention in 2003 and 2004. Ms. Sheble completed her internship in 2004 and currently works as a school psychologist. Ms. Sheble has a 13-year old son with autism spectrum disorder.