Theories on Obesity and Type 2 Diabetes Mellitus: Predictors of Short-term Weight Loss and Disease Prevention in Elementary School Children

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Abstract

The following project is composed of two distinct parts. In the first part, a literature review was performed to explore the proposed mechanisms by which obesity leads to type 2 diabetes mellitus. While it is well-established that excess visceral fat is a strong risk factor for diabetes, there are alternative theories which attempt to explain this relationship. These include the portal hypothesis, the endocrine theory, and the ectopic fat theory. It was hypothesized that more than one theory could explain the link between obesity and diabetes. The results of the literature review supported this hypothesis, demonstrating an overlap between the endocrine theory and the ectopic fat theory underlying the diabetic-obesity relationship. The second part of this project examined data from the Activa y Sana pilot study to find child and parent-related predictors of short-term weight loss in elementary school children. Hours of TV watched per day and exercise intrinsic motivation are some examples of the variables explored. It was hypothesized that several predictors would be found using Pearson's Correlation Coefficients. The results showed that none of the correlations between child and parent data was statistically significant. Small sample size was a primary limitation of the analysis.

Introduction

Today in the United States, approximately 65% of the adult population is overweight or obese (NCHS 2002). This number has increased dramatically from the late 1970s, when prevalence was only 47% (NCHS 2002). The Centers for Disease Control and Prevention defines overweight and obesity based on a measurement called Body Mass Index, or BMI. It is a representation of a person's total body fat and, therefore, his or her chronic health risk. Adult BMI is calculated using both weight and height, but does not take into account age, sex, or body composition. Those with a BMI between 18.5 and 24.9 are considered normal weight, those between 25 and 29.9 qualify as overweight, and BMIs equal to 30 and above define obesity (CDC 2007). Child and teen weight status must be assessed differently because BMI varies significantly among different ages and sexes during youth. A child or teen is considered overweight when her BMI exceeds the 85th percentile for her age and sex, and obese when her BMI falls above the 95th percentile (CDC 2007). While more reliable methods of body composition assessment exist, such as skinfolds, BIA, and DXA, BMI is most often used in weight-related studies due to its cost-effectiveness and ease of measurement (CDC 2007).

As a result of the current obesity epidemic, 72 million American adults now live with increased risk for obesity-related disorders such as hypertension, dyslipidemia, type 2 diabetes mellitus, coronary heart disease, stroke, gallbladder disease, osteoarthritis, respiratory problems, and some cancers (CDC 2007). In addition, the 17% of overweight children and teens in the United States face increased risk for chronic disease later in life, reduced life span, and increased risk of adult obesity (CDC 2007). Some overweight children now suffer from disorders which have traditionally plagued adults. Specifically, type 2 diabetes mellitus—once referred to as adult-onset diabetes—has gained prevalence among children and teens (CDC 2005). Type 2 diabetes is characterized by insulin resistance, hyperglycemia, frequent urination, dehydration, and damage to the nerves, kidneys, eyes, and heart (WebMD). It is estimated that 1 in 3 children born in the US in 2000 will develop the disease (WebMD). In addition, Hispanic Americans, African Americans, and Native Americans are at especially high risk (CDC 2005). Now approximately 20.8 million people in the United States suffer from type 2 diabetes mellitus (CDC 2005), and obesity is the predominant risk factor. Hence, obesity prevention has become of increasing importance among public health officials, researchers, and physicians.

Scientific literature in the field of obesity has yet to distinguish the exact mechanism by which excess body fat leads to type 2 diabetes. Several theories exist, and research on diabetes continues to grow. Part 1 of this report will discuss those theories in order to comprehend the complex relationship between the two conditions. It is hypothesized that the link involves a combination of two or more physiological models, rather than a single theory. Nevertheless, a better understanding of the mechanism may eventually lead to more effective pharmacological treatment than the oral medications and insulin replacement which are used currently. In the meantime, it is important to study the efficacy of diet and exercise in preventing and treating diabetes and obesity. Researchers in obesity prevention struggle to develop the most effective treatment plan for obese patients.

Obesity prevention programs often include a variety of approaches to weight loss. Problem solving, social support, internet strategies, subject profiling, medication, and cognitive

behavioral therapy have been used in various studies to supplement healthy eating and exercise (Lohman 2007 lecture). These strategies also help elevate the rate of long-term weight management for participants in studies such as Healthy Weight 4 Life and Trevose (Lohman 2007 lecture). However, it has been particularly challenging to apply any of these approaches to childhood obesity prevention. School intervention studies have had little, if any, success in effecting significant weight loss in children (Lohman 2007 lecture). Researchers have found that family-based obesity interventions have been most successful in helping children lose weight (Lohman 2007 lecture). Studies show that the involvement of both children and their parents has helped reduce the prevalence of overweight even ten years later in the child's life (Epstein et al. 1994), suggesting that parental health behaviors have a significant impact on a child's behaviors and health status.

With this concept in mind, a university-based study called Activa y Sana has aimed to narrow down the specific parental characteristics and behaviors which influence the child's health status. Activa y Sana works with low-income Hispanic families in particular, hosting a weekly after-school information session with parents and concurrent physical activity interventions with children. Questionnaires and anthropometric measurements were used to gather data about each participant, and the weight of each individual was tracked on a weekly basis. Part 2 of this report will examine data from the Activa y Sana fall 2006 pilot study and attempt to distinguish significant correlations between parent and child data. For example, parental attendance at weekly sessions may be associated with degree of child weight loss from baseline. It is predicted that the Activa y Sana pilot study data will provide insight into the parent-related predictors of weight loss in children. With a better understanding of how family characteristics influence childhood obesity, it may be possible to develop more effective intervention programs for children and thus prevent their risk of developing dangerous diseases like type 2 diabetes mellitus.

Part One:

The Relationship between Obesity and Type 2 Diabetes Mellitus

Literature Review

Background Information

        In order to prevent the deleterious repercussions of obesity, it is important to understand the physiological mechanisms by which excess body fat leads to various health problems. In the case of type 2 diabetes mellitus, researchers and scientists have yet to agree upon the exact mechanism. Studies show that both diseases arise from increased adiposity, and that insulin resistance is the primary mechanism by which obesity leads to diabetes (Kelley and Goodpaster 2005). New technologies, such as computed tomography and magnetic resonance imaging, have allowed researchers to visualize more than a person's total fat percentage, but where fat is distributed as well (Jensen 2006). In recent studies, distribution of adipose tissue has been shown to correlate with various degrees of risk for type 2 diabetes mellitus. Specifically, visceral adipose tissue is often shown to be a strong predictor of insulin resistance (Jensen 2006).

        Studies show that patients with type 2 diabetes mellitus have more visceral adipose tissue than subcutaneous adipose tissue (Kelley and Goodpaster 2005). There are several theories for the mechanism by which visceral adipose tissue brings about insulin resistance. One of the first to emerge was the portal hypothesis, which explains that visceral adipose tissue releases fatty acids into the portal vein, and these fatty acids then travel to the liver where they affect insulin sensitivity (Kelley and Goodpaster 2005). However, this theory has been largely discounted due to its over-simplicity and the fact that visceral fat only accounts for a small percentage of circulating free fatty acids (Salmenniemi et al. 2005). Newly emerging studies propose that the adipocytes within visceral adipose tissue release adipocyte-derived hormones and cytokines that lead to insulin resistance (Ravussin and Smith 2002). Adiponectin is one such hormone under investigation (Kantartzis et al. 2006). More research seems to support the theory of ectopic fat storage, in which increased visceral adipose tissue leads to increased adiposity in other organs such as the liver and skeletal muscle (Ravussin and Smith 2002). There is growing investigation into this theory and increasing evidence that shows the dangerous impact of elevated liver and muscular fat content (Kelley and Goodpaster 2005). With so many conflicting studies, it seems impossible to speculate at this point whether one particular theory explains chronic disease variation among all individuals. Therefore, it is hypothesized that both the endocrine properties of visceral fat and the resulting propensity to store fat ectopically explain why type 2 diabetes mellitus arises most frequently in the obese with higher visceral adipose content.

The Risks Associated with Visceral Adipose Tissue

        Goodpaster and Kelley have put together a thorough review of the literature on obesity and type 2 diabetes mellitus. They begin by recognizing the strong correlation between body composition and metabolic disorders such as obesity and diabetes. The metabolic perturbations of diabetes include hyperglycemia, impaired postprandial insulin secretion, hyperinsulinemia, increased systemic free fatty acids and triglycerides, and most importantly, insulin resistance (Kelley and Goodpaster 2005). As the review states, "insulin resistance is the principle mechanism by which obesity heightens the risk for type 2 DM" (Kelley and Goodpaster 2005). In other words, insulin resistance (IR) found in the adipose tissue, muscle, and liver of obese individuals is the predisposing factor for type 2 DM. It is a condition characterized by impaired inhibition of splanchnic glucose output, impaired insulin suppression of lipolysis, and decrease in total body glucose disposal (Kelley and Goodpaster 2005).

It has become a paradigm in the field of body composition that visceral adipose tissue (VAT) is most strongly linked to insulin resistance of the skeletal muscle, dyslipidemia, increased risk for hypertension, and glucose intolerance (Kelley and Goodpaster 2005). However, some studies question the VAT-insulin resistance relationship. Does the location and type of fat truly account for variations among diseased individuals, or are researchers underestimating the importance of total body fat? A recent study by Salmenniemi and colleagues aims to clarify this relationship.

The goal of the Salmenniemi study was to assess the role of general versus intra-abdominal fat in inducing the metabolic perturbations of obesity and type 2 diabetes mellitus. It is a cross-sectional study of 129 non-diabetic subjects, who are also offspring of diabetic parents. Bioelectrical impedance was used to assess each subject's total fat mass (TFM), and computed tomography (CT) was used to measure intra-abdominal, or visceral, fat mass. The subjects were then grouped according to these measurements. Once in groups, each subject's insulin sensitivity was measured using a euglycemic insulin clamp, insulin secretion using the intravenous glucose tolerance test, and energy expenditure using indirect calorimetry. The authors of the study contend that few studies in the past have used such elaborate methods to assess the significance of intra-abdominal fat in inducing insulin resistance (Salmenniemi et al. 2005).

The results of the study show that individuals with high intra-abdominal fat had high levels of adiponectin and C-reactive protein, low rates of glucose uptake and energy expenditure, and high rates of lipid oxidation (Salmenniemi et al. 2005). Evidence shows that those with high intra-abdominal fat had more insulin resistance regardless of total body fat, suggesting that central obesity is indeed significant to perturbations in metabolism (Salmenniemi et al. 2005). In fact, high intra-abdominal fat was associated with each measured component of metabolic syndrome, except for high blood pressure (Salmenniemi et al. 2005). Ultimately, the Salmenniemi study supports the theory that visceral fat has a stronger correlation with insulin resistance than total body fat, but it does not test the importance of visceral fat versus subcutaneous fat.

The Role of Subcutaneous Fat

A study by Ross and colleagues was written in response to conflicting results about the role of subcutaneous adipose tissue. Some claim that it does not affect insulin sensitivity, and others say it does. It has been proposed that the various layers of subcutaneous adipose tissue may affect the results of these studies because each layer has different metabolic properties. Adipocytes deep in the subcutaneous layer – closer to the visceral layer – may resemble visceral adipose tissue metabolically and would be a stronger predictor of insulin resistance than superficial layers (Ross et al. 2002). The Ross article sets out to find whether or not subdivision of subcutaneous and visceral adipose tissue into more distinct layers strengthens the correlation between insulin resistance and either subcutaneous or visceral fat independently. The authors hypothesize that visceral fat will still correlate more strongly with insulin resistance than either subdivision of subcutaneous fat (Ross et al. 2002).

The study used a group of 89 abdominally obese, non-diabetic men at metabolic risk for developing insulin resistance. Standardized methods for measuring weight, height, and waist circumference were used to assess the anthropometric variables of the subjects. Magnetic resonance imaging was used to determine total adipose tissue and skeletal muscle. Total visceral and abdominal subcutaneous adipose tissues were also calculated. Abdominal subcutaneous fat

was divided into anterior and posterior compartments, and abdominal visceral fat was divided into intraperitoneal and extraperitoneal compartments. An oral glucose tolerance test was used to assess glucose tolerance. Insulin sensitivity was measured using a hyperinsulinemic euglycemic clamp (Ross et al. 2002).

The results of the study confirm the authors' hypothesis: visceral adipose tissue alone had a stronger correlation with insulin resistance and glucose intolerance than subcutaneous adipose tissue or nonabdominal adipose tissue (Ross et al. 2002). Dividing subcutaneous abdominal fat into two different layers did not provide any more reason to accept subcutaneous fat as a stronger correlate of insulin resistance (Ross et al. 2002). Instead, the study confirms previous observations that visceral adipose tissue is the stronger correlate—the paradigm that Kelley and Goodpaster speak of in their review. However, the reason why visceral adipose tissue causes more metabolic disturbances than subcutaneous fat is still under investigation. Researchers have yet to confirm a single mechanism responsible for this relationship.

Fat as an Endocrine Organ

In reference to theories on the mechanisms by which intra-abdominal fat has more significant effects on insulin sensitivity than total body fat, both the Salmenniemi article and the Ross article mention the portal hypothesis as a possible explanation. They recognize that insulin resistance and visceral obesity are tightly linked due to increased delivery of FFAs from visceral adipocytes to the liver, resulting in increased gluconeogenesis, VLDL production, and hyperinsulinemia (Salmenniemi et al. 2005, Ross et al. 2002). However, Salmenniemi discounts the portal hypothesis, saying that visceral adipose tissue only contributes a small portion of systemic FFAs. Other literature reviews, such as those by Despres, and Ravussin and Smith agree that the portal hypothesis does not sufficiently explain the risks of increased visceral adipose tissue. These authors also mention other theories, such as the influence of adipocyte size or endocrine factors which differ among visceral fat cells. As Goodpaster and Kelley point out in their review, the influence of adipocyte-derived hormones has become a popular new area of investigation. To address the theory of fat as an endocrine organ, Wexler and associates have put together a study to see if visceral fat does indeed induce greater levels of hormones that lead to insulin resistance.

Wexler and colleagues recognize the overwhelming evidence that suggests central adiposity has a greater affect on chronic disease than total body weight alone. They wish to test the hypothesis that inflammatory factors released from adipocytes may be the mechanism by which central adiposity leads to insulin resistance and type 2 diabetes mellitus. Their experiment considers the following biomarkers of inflammation and endothelial dysfunction: C-reactive protein, E-selectin, intracellular adhesion molecule-1, inerleukin-6, and TNF-a (Wexler et al. 2005).

The methods of the study were as follows: 510 non-diabetic women were grouped by BMI and waist circumference. This was meant to delineate body weight phenotypes. Four groups were orchestrated—low BMI and low WC, low BMI and high WC, high BMI and low WC, and high BMI and high WC. Each subject's level of insulin resistance was accounted for using mean fasting insulin levels. Assays were also performed to measure the level of biomarkers mentioned above. At the end of the study, it was found that fasting insulin was higher for women with greater waist circumference than in lean women (Wexler et al. 2005). BMI was not a factor in fasting insulin levels, suggesting that central adiposity is indeed more indicative of metabolic

risk factors (Wexler et al. 2005). Total body obesity affected IR independently of biomarkers. However, when looking at waist circumference alone, adjustment for biomarkers indicated that CRP, ICAM-1, IL-6, and E-selectin may mediate the relationship between central adiposity and IR (Wexler et al. 2005).

The report concludes that although the mechanism by which adiposity causes IR and type 2 DM is uncertain, their findings suggest the influence of inflammatory cytokines in this mechanism (Wexler et al. 2005). It attests that central adiposity acts to increase risk for type 2 DM through the effects of these inflammatory markers on insulin signaling pathways (Wexler et al. 2005). Inflammatory cytokines may impair insulin signaling through enhanced oxidative stress, increased FFA oxidation, impaired endothelial function, inhibition of glucose uptake by transporter 4, impaired glucose-stimulated insulin release by beta cells, or by the production of more cytokines (Wexler et al. 2005). Ultimately, increases in central adiposity lead to an increase in the severity of these factors, which may explain why visceral fat is a stronger risk factor than total body fat (Wexler et al. 2005). One of the major limitations of the Wexler article is that it does not look into the significance of the hormones adiponectin, leptin, and resistin, which are also suspected to explain the relationship between increased visceral adiposity and chronic disease.

Ectopic Fat Theory

In Goodpaster and Kelley's review of obesity research, muscle fat is cited as an important, and previously overlooked, source of insulin resistance. In the past, scientists discounted the theory that fat in the extremities could increase the risk of type 2 diabetes mellitus because of the weak correlation between gynoid adiposity and insulin resistance (Kelley and Goodpaster 2005). However, only subcutaneous fat of the extremities was measured, and it is now widely recognized that subcutaneous fat has a weaker correlation with insulin resistance than visceral fat. With computed tomography and magnetic resonance imaging, it is possible to measure not only the subcutaneous fat of the limbs, but the fat which lies within and between muscle cells. A new study by Goodpaster found a significant correlation between intermuscular adipose tissue and insulin resistance (Kelley and Goodpaster 2005). Other studies have also confirmed that the amount of lipid contained within skeletal muscle fibers is correlated with the severity of insulin resistance (Ravussin and Smith 2002).

Literature on obesity and type 2 diabetes mellitus has also illustrated a higher prevalence of liver fat among individuals with type 2 diabetes (Kelley and Goodpaster 2005). Some researches have proposed that this fat depot is an important predictor of insulin resistance. They have found a strong association between increased liver fat and increased visceral adipose tissue, high plasma free fatty acids, and insulin resistance (Kelley and Goodpaster 2005). The severity of dyslipidemia in patients with type 2 diabetes mellitus has been directly correlated with the amount of liver fat content (Kelley and Goodpaster 2005). Together, liver fat and muscular fat tissue account for what many researchers call ectopic fat. The ectopic fat theory speculates that fat stored in the liver and muscle is the main source of metabolic perturbations which lead to insulin resistance and type 2 diabetes mellitus (Ravussin and Smith 2002).

A review by Ravussin and Smith theorizes the reasons why fat begins to accumulate in sources outside visceral adipose tissue—mainly the liver and skeletal muscle. The authors first explain that the body's ability to store fat in adipose tissue is limited by the size and number of fat cells (Ravussin and Smith 2002). Obese patients are taking in an inordinate amount of dietary

fat, and their fat cells should proliferate in an attempt to store this excess. Instead, fat cells enlarge in many obese patients, and enlarged fat cells often lead to insulin resistance (Ravussin and Smith 2002). The failure of fat cells to proliferate results in ectopic storage of excess dietary fats (Ravussin and Smith 2002). Impaired whole-body fat oxidation is cited as another possible reason for the ectopic storage of fat (Ravussin and Smith 2002). Interestingly, Ravussin and Smith's article states that fat cells may not proliferate sufficiently due to alterations in the levels of transcription factors and extracellular signals such as prostaglandins, cytokines, and other hormones. As a result, enlarged visceral fat cells secrete an entirely different profile of hormones and adipokines than normal sized fat cells (Ravussin and Smith 2002).The information presented in the Ravussin and Smith article is convincing. However, it is suspected that adipocyte-derived hormones play a role in both the ectopic fat hypothesis and the theory of fat as an endocrine organ. A study by Kantartzis and colleagues has attempted to explain this observation, as described in the section below.

Overlap between Theories

The Kantartzis article brings into consideration the role of the adipocyte-derived hormone, adiponectin, in contributing to ectopic fat accumulation. Adiponectin is a protein active in the regulation of glucose and lipid metabolism and strongly indicative of metabolic perturbations (Kantartzis et al. 2006). Therefore, it is a useful marker to measure in assessing the effect of adiposity on insulin resistance. Effectively, adiponectin is beneficial to metabolism by lowering insulin resistance, raising HDL, lowering triglycerides and several inflammatory factors (Kantartzis et al. 2006). Thus it is protective for type 2 diabetes and cardiovascular disease. By studying adiponectin patterns, it is also possible to examine the relationship between visceral fat and ectopic fat stores in the liver and muscle.

The study used a group of 242 Caucasian, non-diabetic individuals. These participants were divided into two groups according to total body fat percentage. The experimenter measured the plasma adiponectin concentrations, serum lipids, serum markers of inflammation, atherosclerosis, and endothelial function of each subject. In addition, magnetic resonance imaging (MRI) was used to measure visceral adipose tissue, and ^{1}H magnetic resonance was used to measure liver and intramyocellular fat. Then correlations were performed in order to see adiponectin's relationship with these various factors and perhaps gain insight into its metabolic effects.

Results showed that adiponectin was positively correlated with HDL cholesterol, and negatively correlated with LDL cholesterol, triglycerides, high-sensitivity C-reactive protein, interleukin-6, apolipoprotein B, and various other inflammation markers (Kantartzis et al. 2006). These associations were stronger in the obese group than the lean group (Kantartzis et al. 2006). Essentially, the study confirmed previous assumptions about adiponectin regarding inflammation and lipid profiles. The relevant finding in this paper was that adiponectin was also negatively associated with liver and intramyocellular fat, but not independently of visceral fat (Kantartzis et al. 2006). This suggests that visceral fat is indeed a strong correlate of ectopic fat storage. However, adiponectin may be the mechanism by which the effects of increased visceral fat are mediated. The Kantzartzis article also makes up for the limitations of the Wexler article in explaining the relationship between visceral adipocytes and adiponectin.

Discussion of Theories

Across almost all studies, it is clear that central adiposity has a stronger correlation with insulin resistance than total body or subcutaneous adiposity. Many studies, such as the Salmenniemi article, illustrate this pattern. However, there are some which claim there is no difference in the risk between subcutaneous and visceral fat. The Ross article examines the various layers of subcutaneous fat under closer examination, finding that visceral adipose tissue still poses greater risk for insulin resistance and type 2 diabetes mellitus than any subdivision of subcutaneous fat.

Having confirmed this relationship repeatedly, numerous studies have tried to explain the mechanism of it. The first theory to emerge, the "portal hypothesis," has been largely criticized. Wexler's article, on the other hand, tests the theory of fat as an endocrine organ. It finds that, indeed, the inflammatory factors released by adipocytes increase the severity of the metabolic syndrome. Yet another hypothesis serves to convolute these results—the theory of ectopic fat storage as the mechanism by which obesity leads to type 2 diabetes mellitus. Ravussin and Smith find a strong correlation between insulin resistance and intramuscular, intermuscular, and liver fat storage. According to their review, changes in transcription factors, cytokines, and other hormones may lead to impaired fat cell proliferation and fat oxidation, thus promoting ectopic storage of fat. It seems that the endocrine and ectopic fat theories may be intertwined due to the relevance of endocrine factors in each. The Kantartzis article attempts to explain this phenomenon with a study on adiponectin. It can only conclude that visceral fat is strongly associated with ectopic fat storage, and that adiponectin is one possible mechanism by which excess visceral adiposity leads to insulin resistance. This conclusion supports the original hypothesis that both the ectopic fat theory and the endocrine theory—more than just the visceral adipose theory—can explain the relationship between obesity and type 2 diabetes mellitus.

Nevertheless, researchers continue to search for a single explanation. While it seems they are looking for a silver bullet, none may actually exist. The human body is incredibly complicated, and obesity is a relatively new area of research. Many more studies are needed to find the mechanism we are looking for. The problem with much of the existing research is that it is mainly correlation-based data. A cause and effect relationship cannot be deduced. For example, does increased visceral fat lead directly to insulin resistance through an imbalance in inflammatory factors? Or does this imbalance cause fat to be stored ectopically, which then leads to insulin resistance? There are many questions that remain unanswered. To challenge the ectopic fat theory, one could present the fact that athletes store more intramuscular and intramuscular fat than normal. Are the metabolic profiles of athletes different from those of the obese, and how? Much more data is needed to make any conclusions, but this data is difficult to acquire due to the expense and invasiveness of reliable body composition assessment methods. Every method, even those as advanced as computed tomography and magnetic resonance imaging, has limitations which must be taken into account. With more and more trials, and increasingly reliable technology, it may be possible to deduce an accurate theory. Once the link between obesity and type 2 diabetes mellitus is fully explained, researchers can focus on developing pharmacological to help treat and prevent both conditions.

As some scientists ponder the physiological mechanisms which control disease risk due to obesity, other researchers are trying to prevent such diseases from another approach—weight loss. Indeed, countless weight loss studies are underway to circumvent the growing damages of the obesity epidemic. In the next portion of this report, one such study will be examined for relevant factors which may or may not contribute to weight loss in children.

Part Two

The Activa y Sana Childhood Obesity Prevention Study

Research Study

Background

It is evident from part one of this report that a conclusive theory on the mechanism between obesity and type 2 DM has yet to exist. The research involved in developing a better understanding will no doubt be expensive, lengthy, and beneficial to only future generations. Therefore, it is important that health researchers also focus on developing nutritional and physical activity interventions which can help reduce the number of obese adults and children today. As discussed earlier, several approaches to weight loss have succeeded; however, many have also failed. Inducing long term weight loss in children has been extremely challenging for health researchers. It is suspected that perhaps children are far too influenced by the behaviors of their parents and society for researchers to make effective strides in child weight loss. The Activa y Sana study seeks to establish predictors of parent health behaviors, child health behaviors, and weight loss. With knowledge of which behaviors most influence child weight loss, it will then be possible to develop a tailored intervention program for obese children, consequently reducing their risk for type 2 DM.

Activa y Sana

Activa y Sana is an NIH-funded, university-based study which examines associations of child health behaviors and the health behaviors of their parents. It includes three elementary schools with three different interventions related to weight loss of children. The first school participated in a school-based program that met state standards for nutrition and physical activity. The second school underwent this program in addition to an after-school program for physical activity and nutrition. The third school received both the school-based and after-school programs, plus additional family-based instruction. In this report, it is the data from School Three which will be used to examine the relationship between parent and child health behaviors. All three schools were matched based on student demographics; hence, each is composed of a predominantly Mexican-American population. The eight-week family program emphasized behavior modification, nutrition education, and physical activity education.

When the Activa y Sana pilot study began in the fall of 2006, each student and parent underwent standard, non-invasive anthropometric assessments. Height and weight were measured in order to calculate BMI. Attendance was taken for all participants, and weight was re-measured each week throughout the duration of the study. Any missing data due to participant absence was accounted for by taking the average of the two screenings before and after the missing data point.

In addition to anthropometric data, questionnaire data was also included for each participant. Children received different questions than parents, and questionnaires were in English only. The following questionnaires were used in the study: Children's Physical Activity Questionnaire, Children's Diet Assessment Questionnaire, Children's Habits Questionnaire, Weight Management Self-Efficacy, Self-Efficacy and Exercise Habits Survey, Exercise Intrinsic Motivation, Arizona Food Frequency Questionnaire, Arizona Activity Frequency Questionnaire, and Dishman Questionnaire. Only certain questions from the parent and child questionnaires were explored as variables. A total of 12 children and 11 adults are included in the data under examination.

It is hypothesized that several child and parent factors will correlate significantly with child short-term weight loss.

Variables Under Investigation

Using both anthropometric and questionnaire data, several parent and child variables were tested for significant correlation with child weight loss or weight regain. To begin with, weight loss over the fall 2006 period and weight regain over the break between the fall of 2006 and spring of 2007 were measured for every participant. These numbers were used as the basis for most correlations. Child weight loss and regain were compared with other child variables, as well as parent weight loss, parent weight regain, and other parent variables. Below are the factors that were examined as possible determinants of child weight loss or weight regain:

Children: Initial BMI Z-score, initial weight, attendance, hours of television watched, self-efficacy of diet and exercise

Parents: Initial BMI, initial weight, attendance, self-efficacy of weight management, self-efficacy of exercise habits, and exercise intrinsic motivation

For example, child weight loss was tested against both initial BMI Z-score of the child, as well as parent weight loss and parent initial BMI, to find significant correlations. In addition, child initial BMI Z-score was correlated with parent initial BMI in the case study included later in this report.

Lastly, parent weight loss and weight regain were compared against other parent variables to find any significant correlations. While the focus of this pilot study was to find child weight loss variables, a secondary objective was to study parent behavior as well.

Statistical Methods

Weight loss for each participant was calculated by subtracting his or her last weight measurement from the initial measurement for fall 2006 (Weight Change 1). Since this number did not always constitute a decrease in weight, it is termed "Weight Change 1" for clarification. Similarly, weight regain was calculated by subtracting the first measurement of spring 2007 from the last measurement of fall 2006, hence accounting for any weight change during the break from diet and exercise intervention (Weight Change 2). BMI values were calculated using the formula in the introduction of this report. Attendance was measured based on the percentage of sessions in which the subjects participated. Other information, such as self-efficacy scores or hours of TV watched, was determined based on questionnaire answers. Some of these variables were based on the average score of several questions on one questionnaire. For example, parents' exercise intrinsic motivation was calculated from the average score of 16 questions on the Exercise Intrinsic Motivation Questionnaire. The participants answered each question according to a scale from 1 to 5, and all 16 answers were averaged to obtain a single exercise intrinsic motivation score.

Child BMI Z-scores are based on statistical data from the National Center for Health Statistics at the CDC (RCH, 2007). They represent how many standard deviations above or below the mean a child's BMI falls based on age. Children within one Z-score above or below the mean account for 68% of the population of children for that age, and those within two Z-scores account for 98% (RCH, 2007). Child BMI Z-scores were used rather than simple BMI
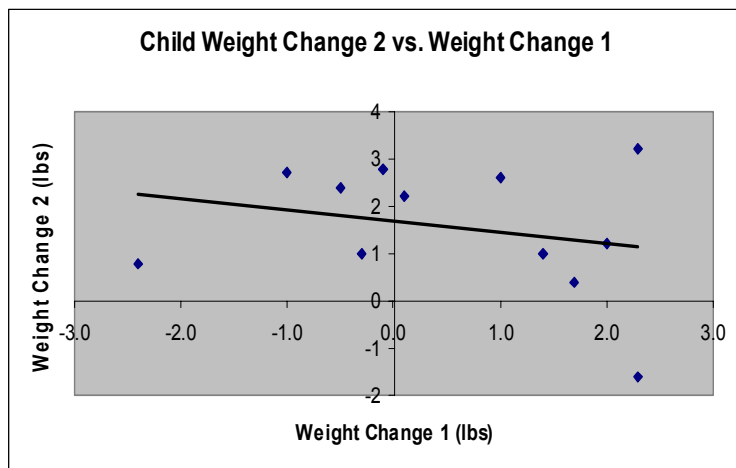
because they take into account age and sex and are therefore more representative of a child's overweight status.

For each set of anthropometric and questionnaire data, averages and standard deviations were calculated. Correlations were calculated using Pearson's Correlation Coefficient, r, as well as r-squared. Scatter-plots were used to visualize a regression line amongst the data. All of the raw data can be found in the appendix in table format; however, it is also displayed in graph form within the Results section below. A p-value of 0.05 was used to ascertain statistical significance at (n-2) degrees of freedom for each tested correlation (Fisher and Yates, 1974).

Results

First, child Weight Change 1 and Weight Change 2 were tested against other child characteristics to find non-parental based correlations. The following scatter plots display a regression line for each correlation, along with R, R-squared, and n. Below the graphs is a summary table of correlation values, along with testing for statistical significance.
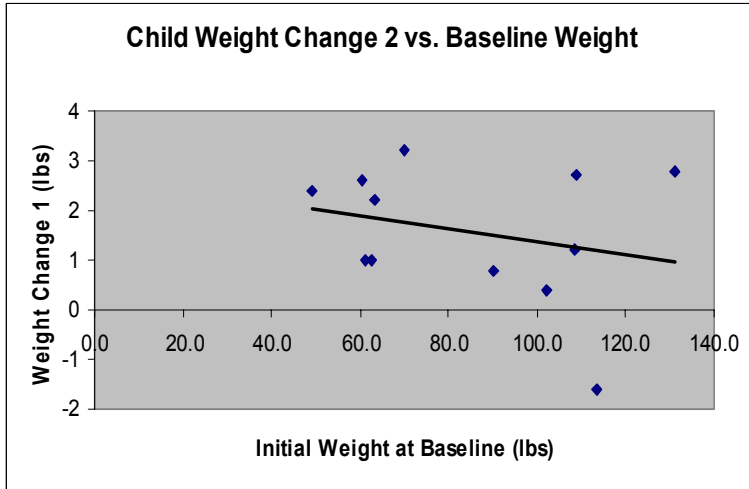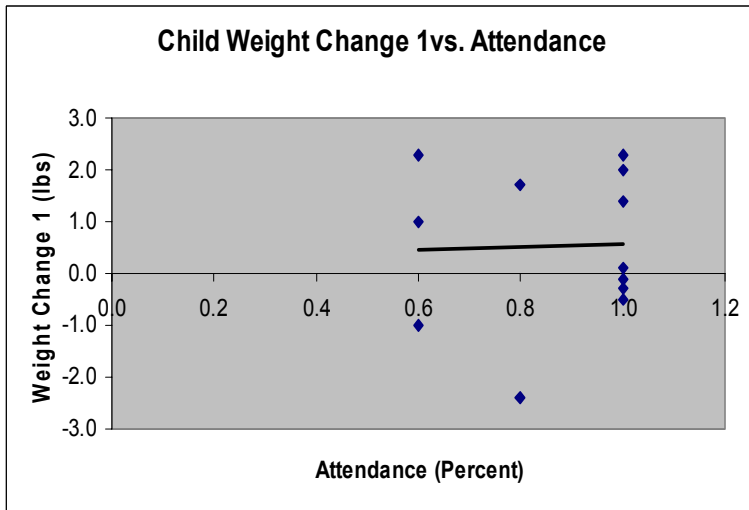
Figure 1

r	-0.25
r^2	0.063
n	12

Figure 2

r	0.07
r^2	0.005
n	12

Figure 3

Child Weight Change 2 vs. Baseline Weight

r	-0.26
r^2	0.069
n	12

Figure 4

Child Weight Change 1vs. Attendance
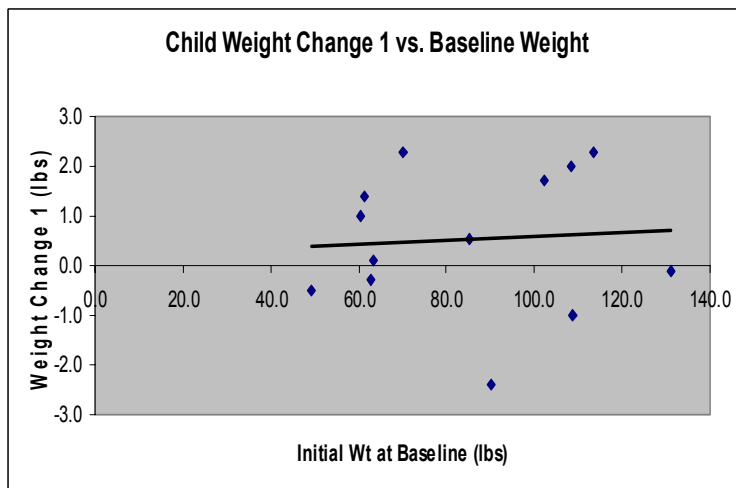
r	0.030
r^2	0.0009
n	12

Figure 5

Child Weight Change 2 vs. Attendance

r	-0.43
r^2	0.19
n	12

Figure 6

Weight Change 1 vs. Hrs TV Watched per Day
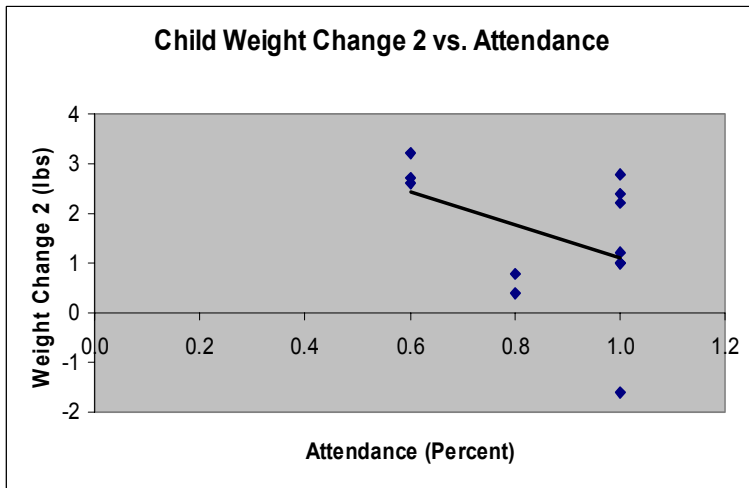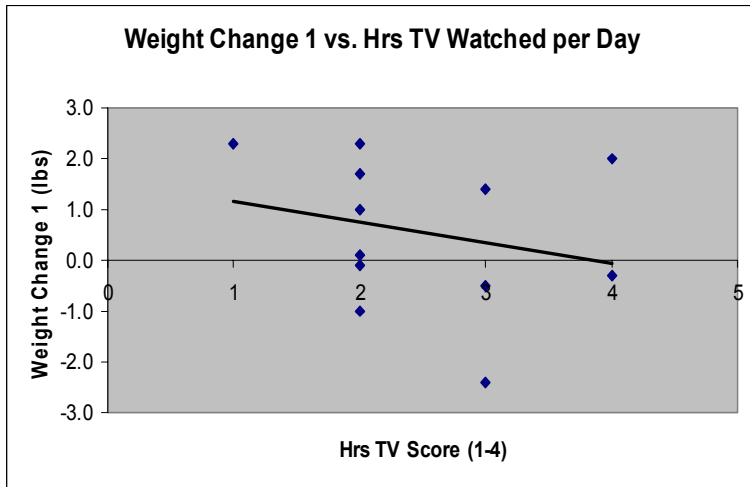
Score	Response
1	None
2	1-2 hrs/day
3	3-4 hrs/day
4	5-6 hrs/day
5	7+ hrs/day

r	-0.25
r^2	0.062
n	12

Figure 7

Weight Change 2 vs. Hrs TV Watched per Day
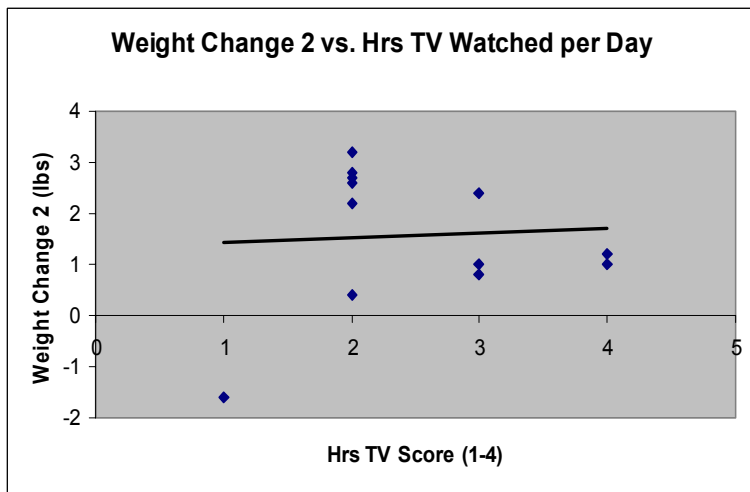
Score	Response
1	None
2	1-2 hrs/day
3	3-4 hrs/day
4	5-6 hrs/day
5	7+ hrs/day

r	0.063
r^2	0.004
n	12

Figure 8

Weight Change 1 vs. Self-efficacy of Exercise and Nutrition

Score	Ability to eat right/exercise
1	I know I can
2	I think I can
3	I'm not sure I can
4	I know I can't

r	0.25
r^2	0.061
n	12

Figure 9
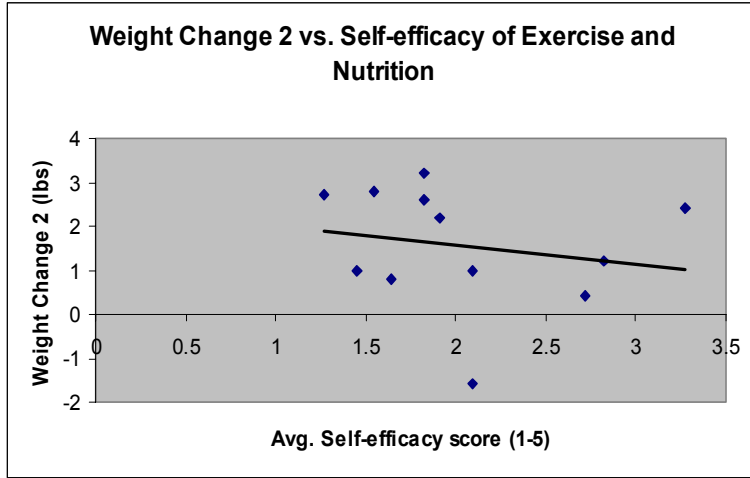
Weight Change 2 vs. Self-efficacy of Exercise and Nutrition

Weight Change 2 (lbs) vs. Avg. Self-efficacy score (1-5)

Score	Ability to eat right/exercise
1	I know I can
2	I think I can
3	I'm not sure I can
4	I know I can't

r	-0.19
r^2	0.037
n	12

Figure 10

Child Weight Change 1 vs. BMI Z score

Weight Change 1 (lbs) vs. Child BMI Z score
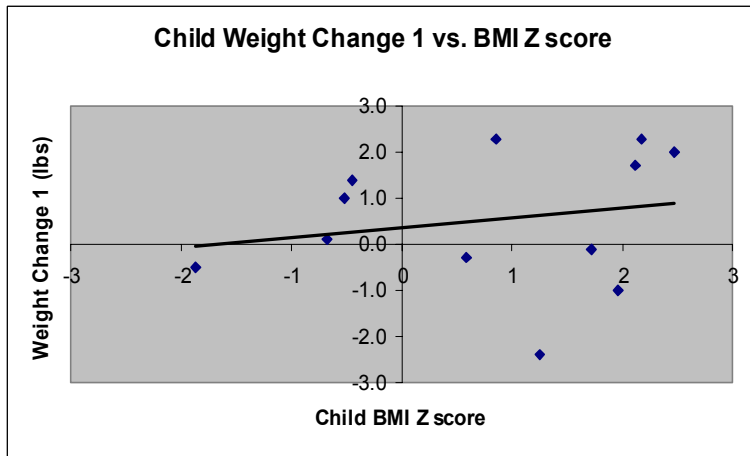
r	0.21
r^2	0.042
n	12

Figure 11

Child Weight Change 2 vs. BMI Z score

Weight Change 2 (lbs) vs. Child BMI Z score
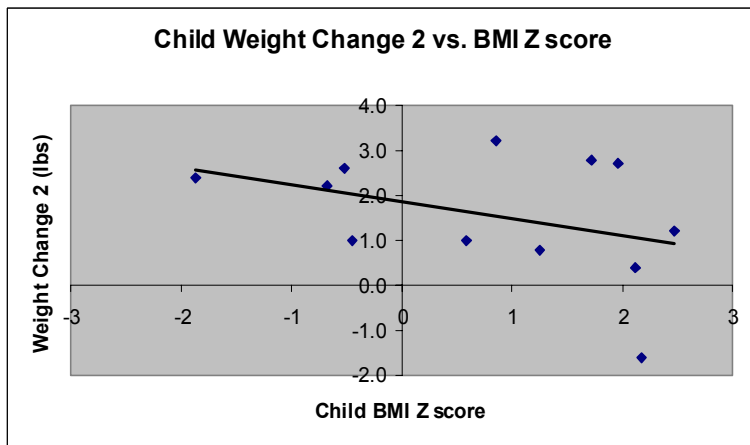
r	-0.38
r^2	0.15
n	12

Since all correlations have 12 participants (n), an r value of at least 0.576 is needed for the correlation to be significant at df=10 and p<0.05 (Fisher and Yates, 1974). Figure 14 summarizes the correlation values of all variable pairs in descending order.

Figure 14: Summary of Child-only Correlations

Variable 1	Variable 2	r	[r]	Significant at p<0.05?
Attendance	Weight Change 2	-0.43	0.43	NO
Child BMI Z-score	Weight Change 2	-0.38	0.38	NO
Baseline Weight	Weight Change 2	-0.26	0.26	NO
Weight Change 1	Weight Change 2	-0.25	0.25	NO
Hours TV/day	Weight Change 1	-0.25	0.25	NO
Self-Efficacy	Weight Change 1	0.25	0.25	NO
Child BMI Z-score	Weight Change 1	0.21	0.21	NO
Self-Efficacy	Weight Change 2	-0.19	0.19	NO
Baseline Weight	Weight Change 1	0.070	0.070	NO
Hours TV/day	Weight Change 2	0.063	0.063	NO
Attendance	Weight Change 1	0.030	0.030	NO

It is evident from Figure 14 that none of the correlations among child weight change and other characteristics was significant. However, by displaying the absolute values of r in descending order, it is possible to see which correlations were strongest or weakest. By looking at the sign of the r values, one can also see whether Variable 1 was correlated with positive or negative weight changes.

Next, Child Weight Change 1 and Child Weight Change 2 were tested against all parent variables. In this case, the number of participants (n) was equal to the number of child-parent pairs and was limited by the number of parents with complete data sets for each variable. Below are the charts for all child-parent correlations, followed by a summary table.
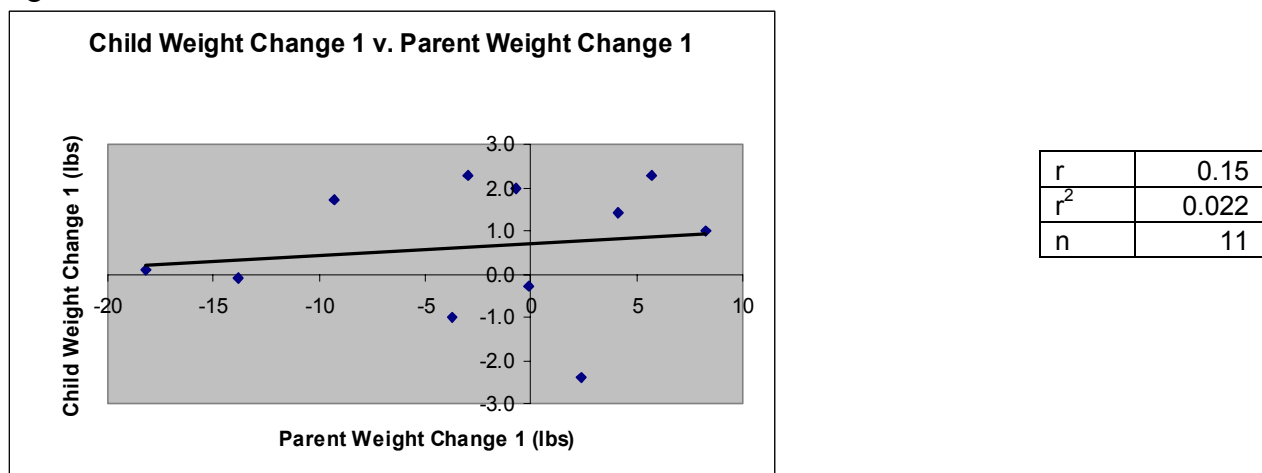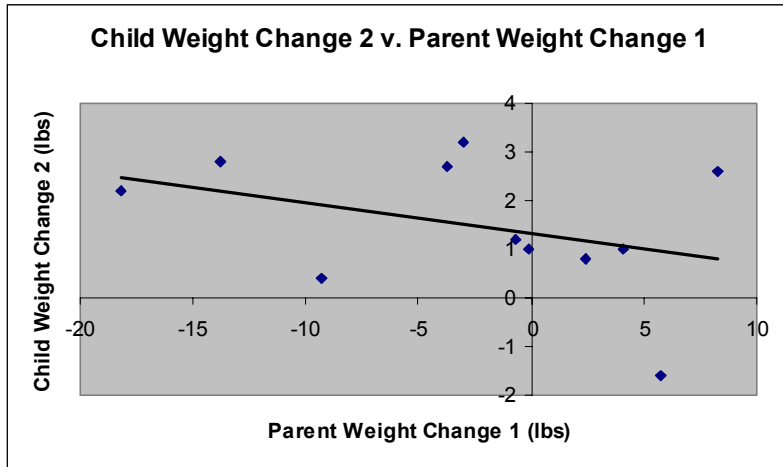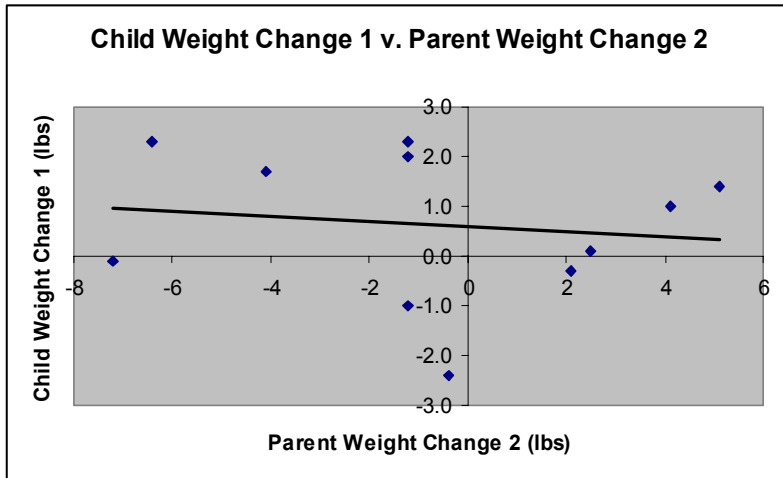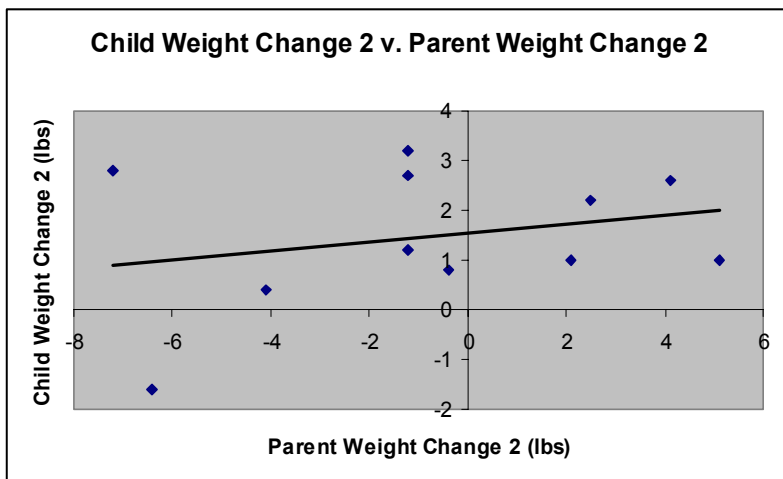
Figure 15

r	0.15
r^2	0.022
n	11

Figure 16

Child Weight Change 2 v. Parent Weight Change 1

r	-0.37
r^2	0.14
n	11

Figure 17

Child Weight Change 1 v. Parent Weight Change 2

r	-0.13
r^2	0.018
n	11

Figure 18

Child Weight Change 2 v. Parent Weight Change 2

r	0.26
r^2	0.068
n	11

Figure 19

Child Weight Change 1 vs. Parent BMI

r	-0.40
r^2	0.16
n	11

Figure 20

Child Weight Change 2 vs. Parent BMI

r	-0.47
r^2	0.22
n	11

Figure 21

Child Weight Change 1 vs. Parent Baseline Wt
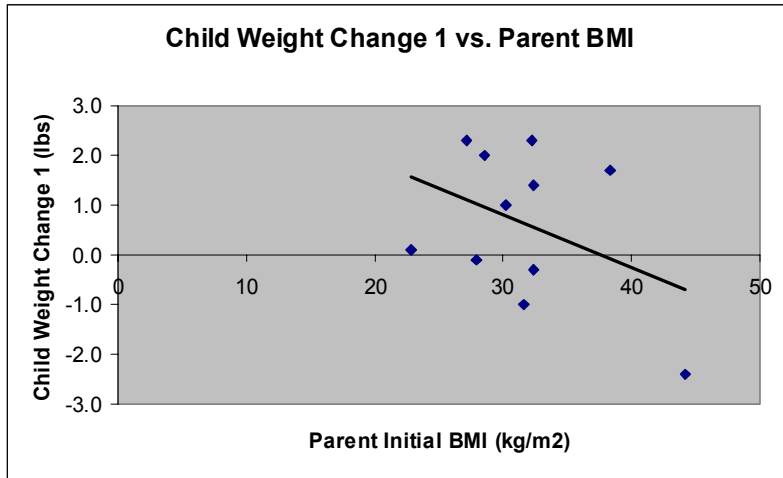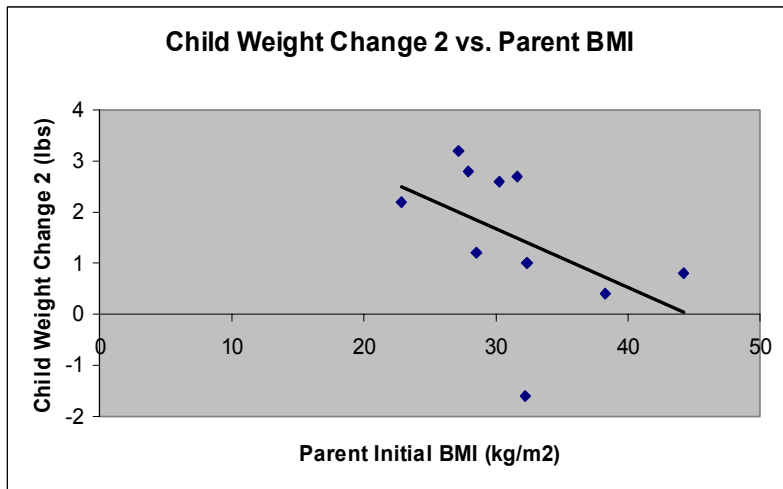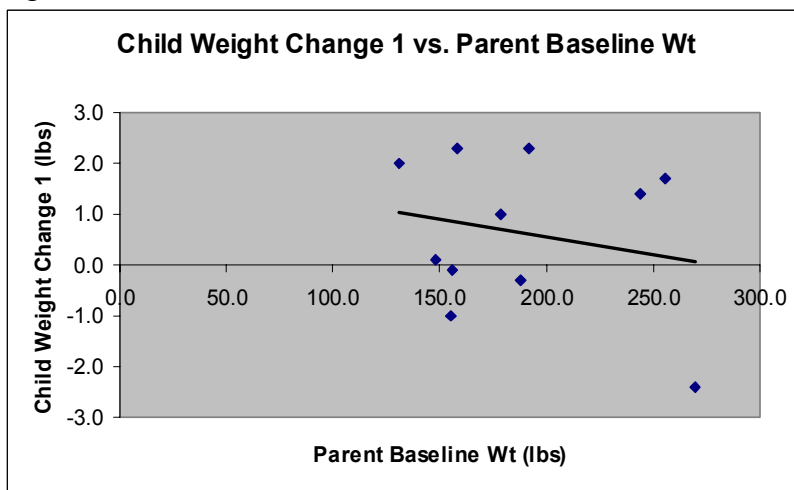
r	-0.22
r^2	0.050
n	11

Figure 22

Child Weight Change 2 vs. Parent Baseline Wt

r	-0.49
r^2	0.24
n	11

Figure 23

Child Weight Change 1 vs. Parent Attendance
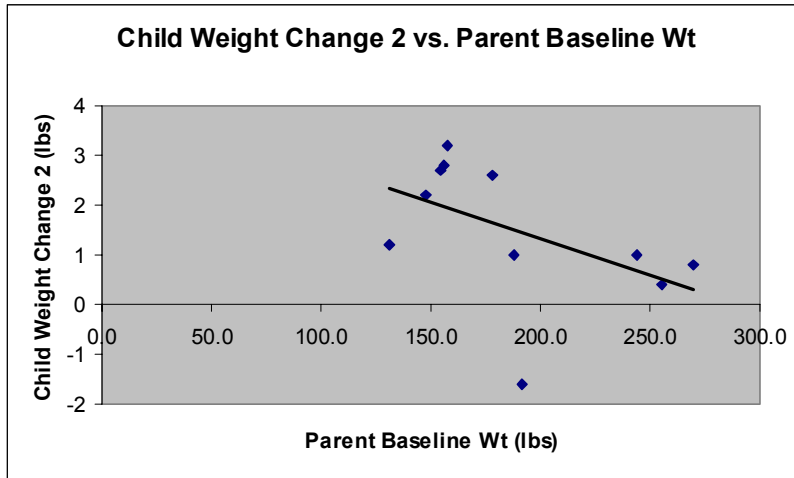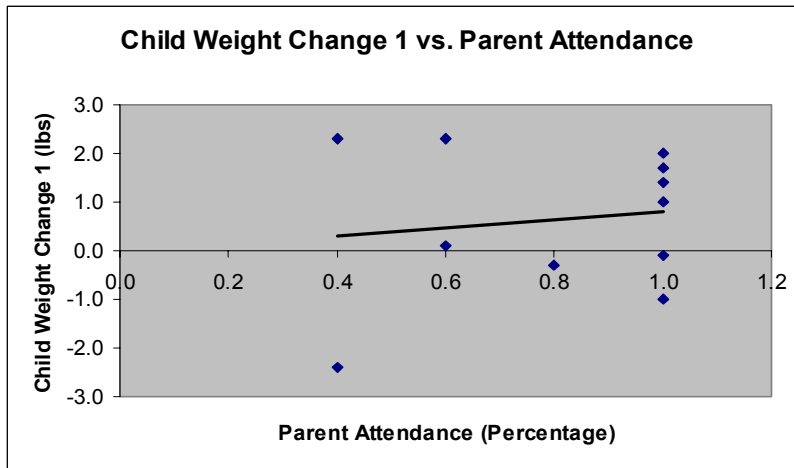
r	0.15
r^2	0.021
n	11

Figure 24

Child Weight Change 2 vs. Parent Attendance
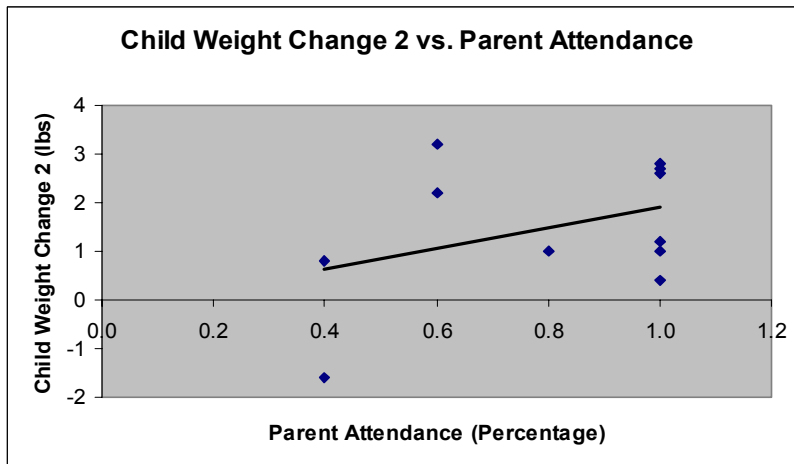
r	0.39
r^2	0.15
n	11

For parent Exercise Intrinsic Motivation in the two graphs below, the following table depicts the meaning of each parent's numerical score:
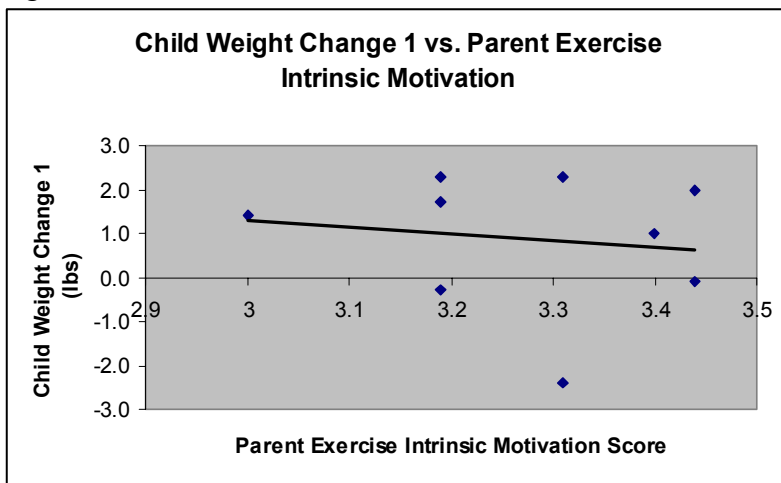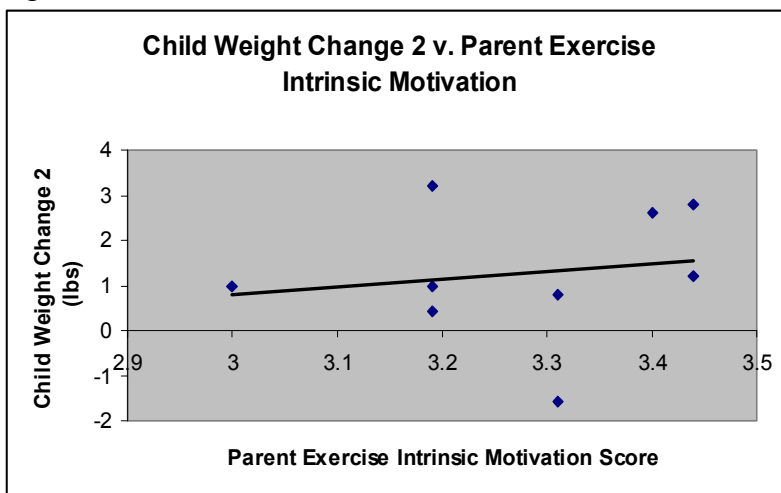
Exercise Intrinsic Motivation - higher score means more EIM	
Score	Response
1	Strongly Disagree
2	Disagree
3	Unsure
4	Agree
5	Strongly Agree

Figure 25

Child Weight Change 1 vs. Parent Exercise Intrinsic Motivation

r	-0.14
r^2	0.021
n	9

Figure 26

Child Weight Change 2 v. Parent Exercise Intrinsic Motivation
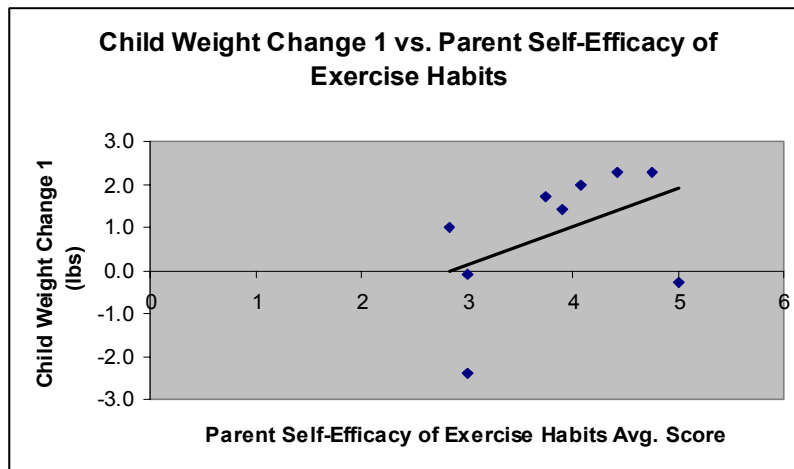
r	0.17
r^2	0.030
n	9

For parent Self-Efficacy of Exercise Habits, the following legend explains how the numerical score for each parent is translated:
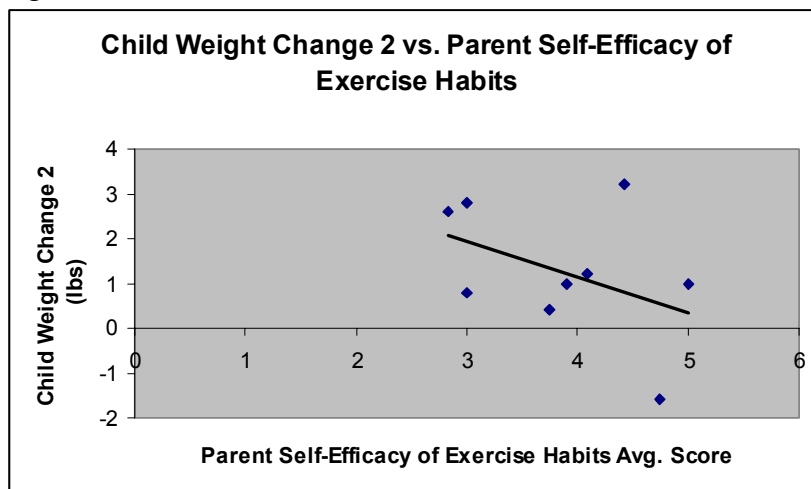
Self-Efficacy and Exercise Habits Survey - higher score means more SE*	
Score	Response
1	I know I cannot
2	
3	Maybe I can
4	
5	I know I cannot
6	Does not apply (score not included

Figure 27

Child Weight Change 1 vs. Parent Self-Efficacy of Exercise Habits

r	0.45
r^2	0.20
n	9

Figure 28

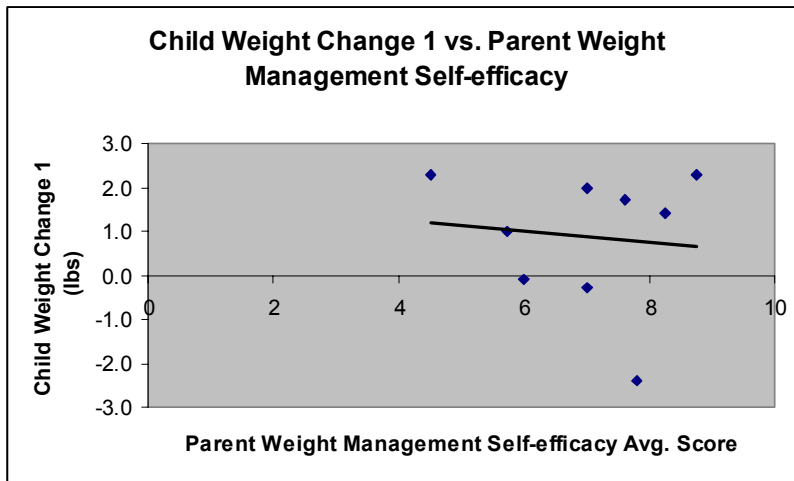Child Weight Change 2 vs. Parent Self-Efficacy of Exercise Habits

r	-0.43
r^2	0.19
n	9

In the following two graphs comparing child weight change to parent Weight Management Self-efficacy, the score for each parent is based on a scale from 1 to 10. Parents answered each question according to this scale, with 1 being least confident and 10 being most confident.

Figure 29

Child Weight Change 1 vs. Parent Weight Management Self-efficacy

r	-0.10
r^2	0.011
n	9

Figure 30

Child Weight Change 2 vs. Parent Weight Management Self-Efficacy

r	0.39
r^2	0.15
n	9

Figure 31

Child BMI Z score vs. Parent BMI
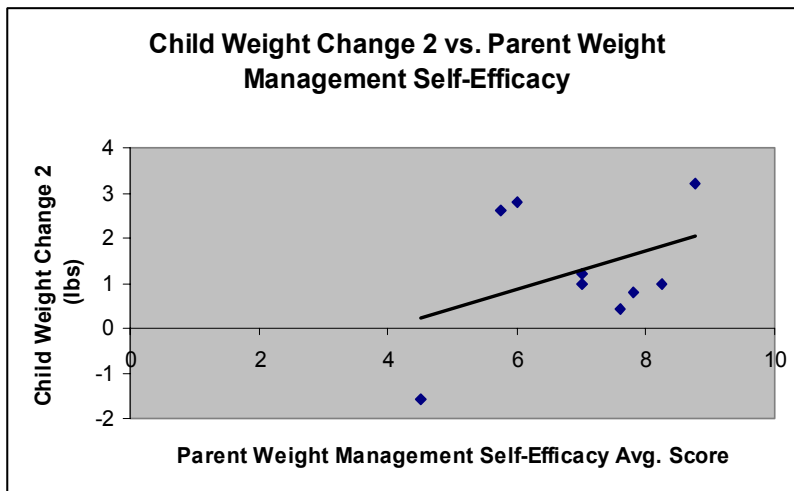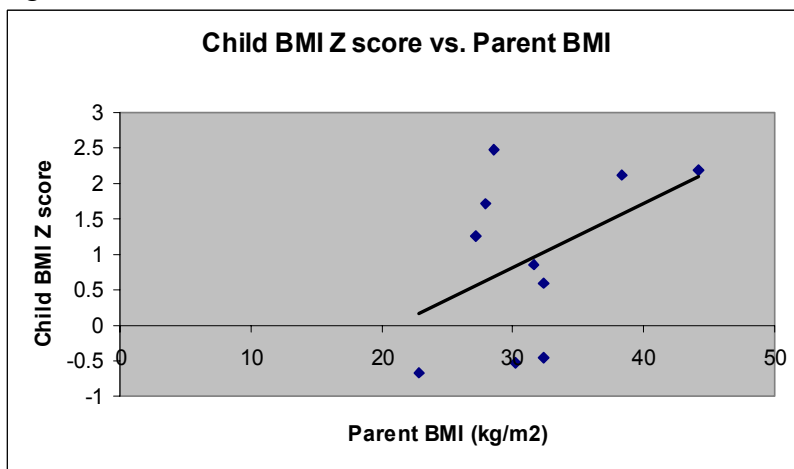
r	0.45
r^2	0.21
n	10

Figure 32: Summary of Child-Parent Correlations

Parent Variable	Child Variable	r	[r]	n	df	Significant at p<0.05?
Baseline Weight	Weight Change 2	-0.49	0.49	11	9	NO
Initial BMI	Weight Change 2	-0.47	0.47	11	9	NO
Initial BMI	BMI Z score	0.45	0.45	10	8	NO
SEEH	Weight Change 1	0.45	0.45	9	7	NO
SEEH	Weight Change 2	-0.43	0.43	9	7	NO
Initial BMI	Weight Change 1	-0.40	0.40	11	9	NO
Attendance	Weight Change 2	0.39	0.39	11	9	NO
WMSE	Weight Change 2	0.39	0.39	9	7	NO
Weight Change 1	Weight Change 2	-0.37	0.37	11	9	NO
Weight Change 2	Weight Change 2	0.26	0.26	11	9	NO
Baseline Weight	Weight Change 1	-0.22	0.22	11	9	NO
EIM	Weight Change 2	0.17	0.17	9	7	NO
Weight Change 1	Weight Change 1	0.15	0.15	11	9	NO
Attendance	Weight Change 1	0.15	0.15	11	9	NO
EIM	Weight Change 1	-0.14	0.14	11	9	NO
Weight Change 2	Weight Change 1	-0.13	0.13	11	9	NO
WMSE	Weight Change 1	-0.10	0.10	9	7	NO

Again, it is evident that no correlations between parent and child data were significant. However, the table allows us to see which correlations were strongest and whether or not the parent variable was correlated with positive or negative weight changes (except in the case of Child BMI Z-score vs. Parent BMI).

For the last part of the results, parent weight loss and weight regain during the pilot study were compared with other parent variables of particular interest. These are attendance, exercise intrinsic motivation, self-efficacy of exercise habits, and weight management self-efficacy. Those correlations are displayed below in graph form, once again followed by a summary table.
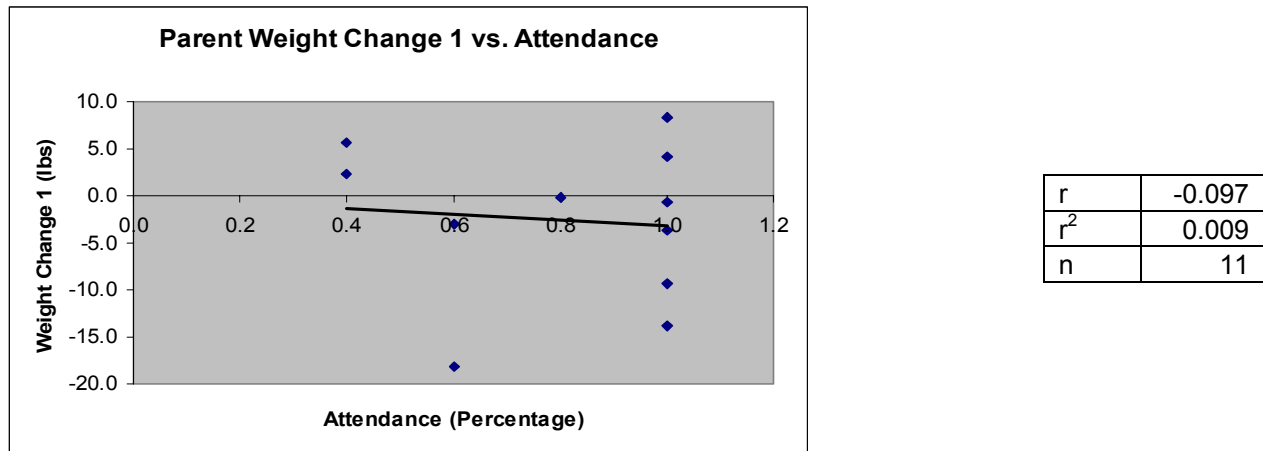
Figure 33

r	-0.097
r²	0.009
n	11

Figure 34

Parent Weight Change 2 vs. Attendance
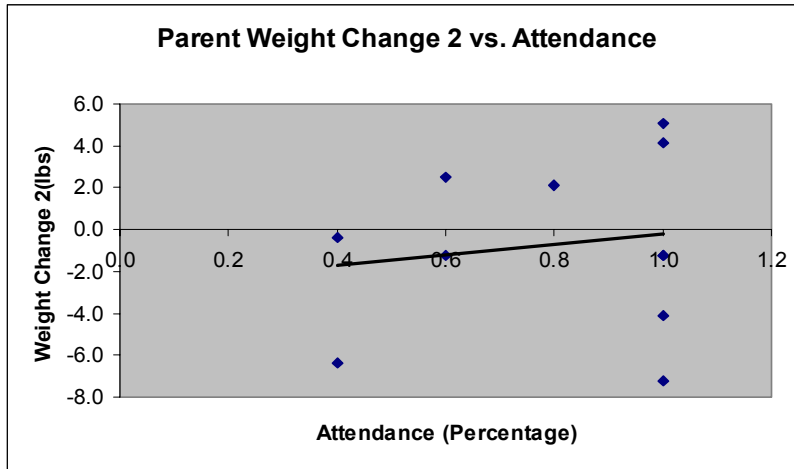
r	0.15
r^2	0.023
n	11

Figure 35

Parent Weight Change 1 vs. Exercise Intrinsic Motivation

r	-0.36
r^2	0.13
n	11

Figure 36

Parent Weight Change 2 vs. Exercise Intrinsic Motivation
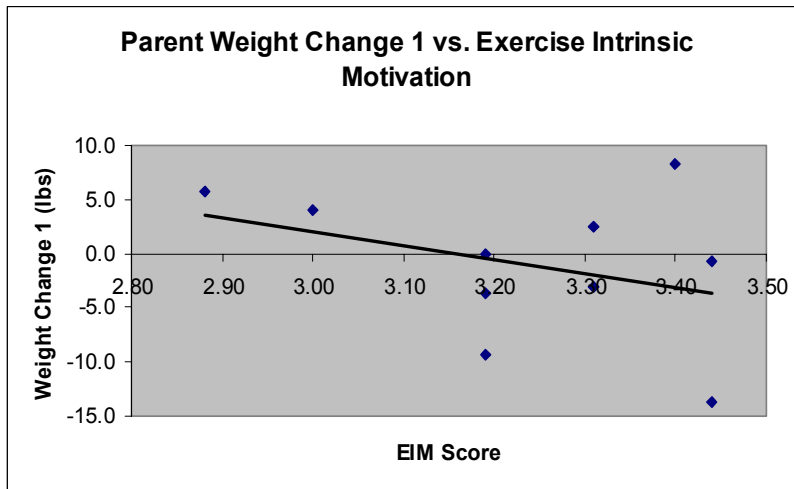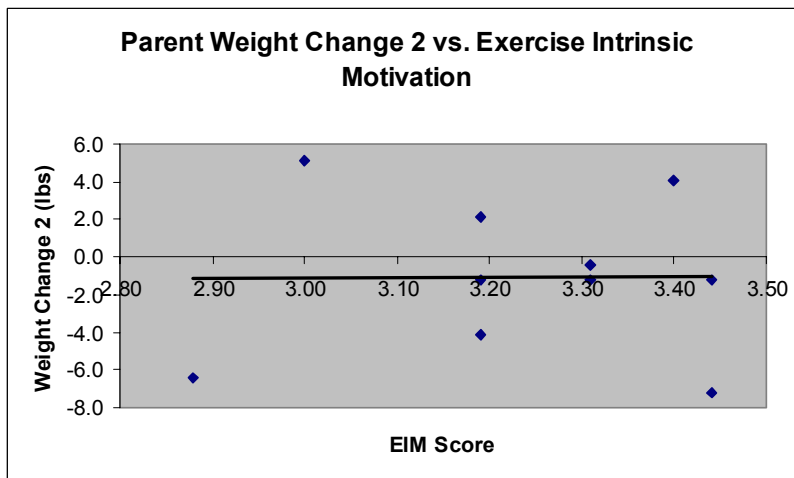
r	0.0076
r^2	5.8E-05
n	10

Figure 37

Parent Weight Change 1 vs. Self-Efficacy of Exercise Habits
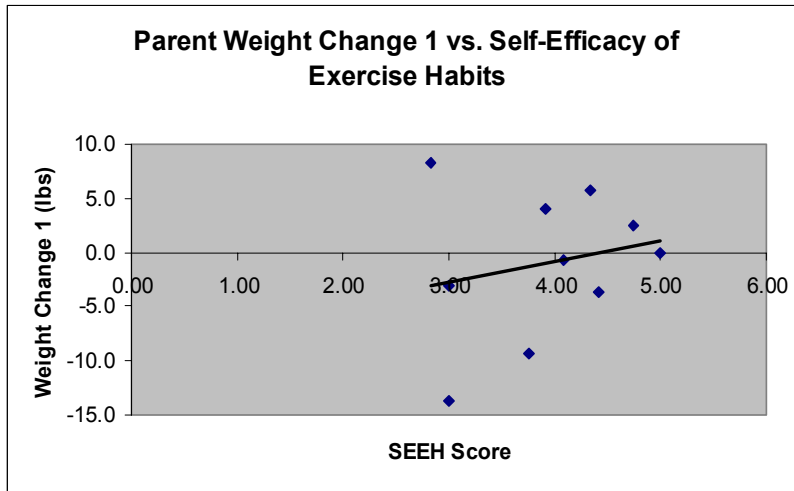
r	0.22
r^2	0.047
n	10

Figure 38

Parent Weight Change 2 vs. Self-Efficacy of Exercise Habits
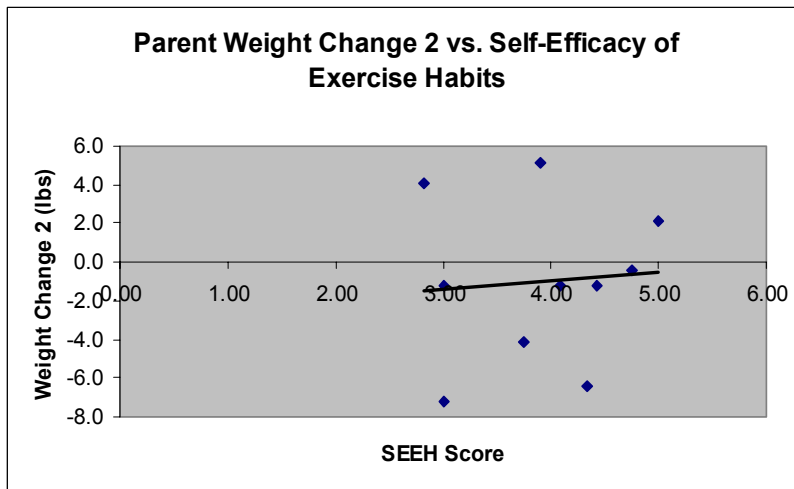
r	0.082
r^2	0.0067
n	10

Figure 39

Parent Weight Change 1 vs. Weight Maintenance Self-Efficacy

r	-0.20
r^2	0.039
n	10

Figure 40

Parent Weight Change 2 vs. Weight Maintenance Self-Efficacy

r	0.13
r^2	0.016
n	10
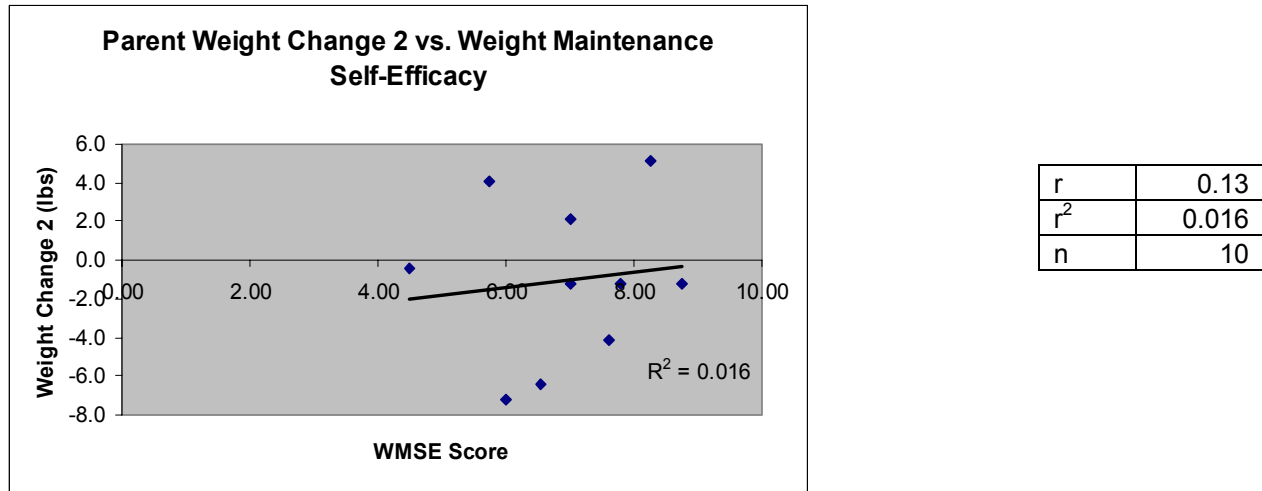
Figure 41: Summary of Parent-only Correlations

| Variable 1	Variable 2	r	[r]	n	df	Significant at p<0.05?
EIM	Weight Change 1	-0.36	0.36	10	8	NO
SEEH	Weight Change 1	0.22	0.22	10	8	NO
WMSE	Weight Change 1	-0.20	0.20	10	8	NO
Attendance	Weight Change 2	0.15	0.15	11	9	NO
WMSE	Weight Change 2	0.13	0.13	10	8	NO
Attendance	Weight Change 1	-0.097	0.097	11	9	NO
SEEH	Weight Change 2	0.082	0.082	10	8	NO
EIM	Weight Change 2	0.0076	0.0076	10	8	NO

As is the case with both child-only and child-parent correlations, there are no significant relationships between parent weight change and other parent variables.

Given the results in all of the scatter plots above, it is clear that there is much variation among this small group of participants. Upon closer investigation, it may be possible to draw inferences which can explain this considerable variation. Therefore, we will examine the data more closely using a case study of two different children with vastly different BMI Z scores, but similar-weight parents.

Case Study

In the graph below, two children (c-SAN02 and c-SAN41) have parents with similar BMIs (a-SAN02: 28.5 kg/m^2; a-SAN41: 30.2 kg/m^2); however, these two children have drastically different BMI Z-scores. This case study will compare other, non-parental data between these two children which may explain differences in their anthropometric data.

Figure 42: Case Study

Child BMI Z score vs. Parent BMI
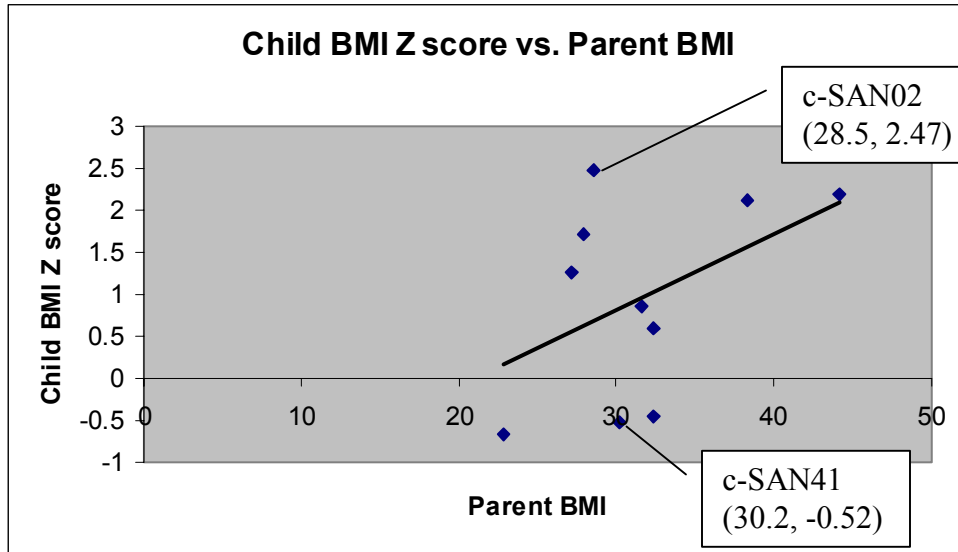
Parents a-SAN02 and a-SAN41 both have BMIs within 1.5kg/m2 of 30kg/m2 (average equal to 29.35 ± 1.2). However, child c-SAN02 has a BMI Z-score of 2.47, while child c-SAN41 has a BMI Z-score of -0.52. It is evident that the difference in BMI status between these two children is not associated with correspondingly different parental BMIs. Therefore, it is suspected that variables other than parental health status contribute to their very different BMI Z-scores. Such variables include exercise intrinsic motivation, self-efficacy of diet and exercise, number of hours TV per day, etc. In order to compare these variables among the two children of this case study, questionnaire data will be assessed. The following tables present this data.

Figure 43

ANTHROPOMETRIC DATA	c-SAN02	c-SAN41
BMI	27.2	15.3
BMI Z-score	2.47	-0.52
Weight Change During Fall 2006 Intervention	+1.0 lbs	+2.0 lbs
Weight Regain from November to January	+1.2 lbs	+2.6lbs

Figure 44

QUESTIONNAIRE DATA	c-SAN02	c-SAN41
Children's Diet Assessment Questionnaire		
Yesterday, did you eat French fries or chips?	No	No
Yesterday, did you eat any vegetables? Vegetables are salads; boiled, baked, and mashed potatoes; and all cooked and uncooked vegetables.	Yes, I ate vegetables 1 time yesterday	Yes, I ate vegetables 3 or more times yesterday
Yesterday, did you drink fruit juice? Fruit juice is a drink which is 100% juice, like orange juice, apple juice or grape juice. Do not count punch, kool-aid, sports drinks, or other fruit-flavored drinks.	No	Yes, I drank fruit juice 1 time yesterday
Yesterday, did you drink milk?	No	Yes, I drank

		milk 2 times yesterday
Yesterday, did you eat doughnuts, sweet rolls, cookies, brownies, pies, or cake?	No	No answer
Yesterday, did you drink soda pop?	No	No answer
Yesterday, did you eat ice cream, an ice cream bar, or other frozen dairy dessert? Examples of a frozen dairy dessert are: ice cream cone, ice cream sundae or milkshake.	No	No answer
Yesterday, did you eat candy or a candy bar? Examples of candy are: candy pieces like M&M's, hard candy (like lollipops, Jolly Ranchers or tamarindo), and regular gum. This does NOT include sugar-free gum or candy.	Yes, I ate these foods 1 time yesterday	No
Yesterday, did you eat out in a restaurant or take food "to go" from a restaurant?	No	No
Children's Habits Questionnaire		
How many times do you drink each of these beverages in a day or week?		
Water	Everyday	Everyday
Juice	1-3 times/wk	1-3 times/wk
Soda Pop	1-3 times/wk	Never
Diet Soda Pop	Never	Never
Whole Milk	4-6 times/wk	1-3 times/wk
1% or 2% Milk	1-3 times/wk	4-6 times/wk
Skim Milk	Never	Never
How many hours of television do you watch everyday?	5-6 hrs/day	1-2 hours/day
How many hours of video games and computer games do you play every day?	3-4 hrs/day	None
How sure are you? (Self-efficacy of physical activity and eating habits based on average score for 11 questions) Scores: 1 = I know I can; 2 = I think I can; 3 = I'm not sure I can; 4 = I know I can't	2.82	1.82
Children's Physical Activity Questionnaire		
What did you do TODAY after school? (Composite score based on the average score for 31 different questions) Ex.: Bicycling/scootering: 1 = None; 2 = a little; 3 = a lot	1.613	1.968

Figures 43 and 44 allow us to examine the differences in health habits between these two children as a way to explain their difference in weight status. The first child, c-SAN02, is overweight, while the second child, c-SAN41, is underweight. The first child appears to eat fewer vegetables than the second child, while the second child drank more fruit juice and milk than the first child. When asked if they ate candy yesterday, only the first child answered yes. The first child also drinks more soda pop and whole milk than the second child, who mostly drinks 1 or 2% milk and never soda pop. The first child spends more time watching television and playing video and computer games than the second child. According to the self efficacy score, the first child has less confidence in his/her ability to eat right and exercise. Lasly, the second child appears to participate in a greater amount and variety of physical activities. The

differences in these two children amount to an obviously healthier lifestyle in the lower-weight child than the higher-weight child, with no factors to suggest otherwise.

Discussion

In this study, I searched for possible predictors of child weight loss based on anthropometric and questionnaire data gathered from the Activa y Sana pilot study. In doing so, I hoped to determine which parent health behaviors most influence child health behaviors. The results of my investigation showed that there were no significant correlations ($p < 0.05$) between child weight loss and any other child or parent characteristics examined in the study. As a secondary goal, determinants of parent weight loss were investigated using the same data. Again, there were no significant correlations ($p < 0.05$). Another important finding was that not all variables correlated with weight change in the direction expected. Rather, some characteristics traditionally thought to promote weight loss actually correlated with weight gain. The factors that unexpectedly correlated with child weight *gain* during the intervention include fewer hours of TV watched per day, increased self-efficacy of diet and exercise, lower parent BMI, lower parent attendance, and lower parent self-efficacy of exercise habits. In addition, increased self-efficacy of weight management was associated with weight gain in parents during the intervention.

Activa y Sana utilized family-based intervention, a specialized area of child weight-loss research currently under investigation. Several studies have been performed in which parent involvement in the child's weight-loss regimen was analyzed for significance. For example, findings from Epstein et al. support the efficacy of family-based behavioral treatment for childhood obesity, even at ten-year follow up (1994). The Epstein study used a weekly treatment regimen, similar to Activa y Sana, and also used BMI and questionnaire data. However, rather than an eight-week long school-based intervention, the Epstein study used volunteer participants in a clinical setting for a three-month long intervention. Also, these participants were randomly chosen and predominantly middle-class; whereas, those in Activa y Sana came from primarily Hispanic, low-income backgrounds. Currently, there are virtually no weight loss studies which focus on family-based interventions among Hispanic, low-income groups. Therefore, the end results of the Activa y Sana study should provide a benchmark for weight loss research in this population. Unfortunately, my analysis of the data from the pilot study alone did not present any parent or child health behaviors which have a significant effect on child weight loss.

The lack of significant predictors of child weight loss may be due to the apparent limitations of my analysis. Mainly, the low number of participants in the study was a considerable hindrance to producing any significant correlations. In several of the scatter plots, it is visible that very few of the data points fall close to the regression line. Many fall far above or below the line. This demonstrates how a very small number of participants can result in a wide variation in the data, making it difficult to draw legitimate conclusions. Another significant limitation to this study was the short duration of the intervention. As a result, weight changes among the participants were not always substantial, and therefore, correlations were not very strong. Other statistical limitations include the fact that participation in the project was voluntary and depended on weekly attendance to meetings. Also, this particular elementary school under investigation may not necessarily represent the general Hispanic population. And lastly, although translators were present during the weekly meetings, questionnaires were in English only, which may have affected the responses of non-English speakers. All in all, it is important to consider

that none of the data could account for genetic variation among the participants in the study—an important facet of an individual's ability to lose weight.

It is possible that the limitations discussed above may have confounded some of the trends seen in the data. Nevertheless, I was still able to make speculations about the results based on the direction of correlations. To begin with, the very first correlation calculated (Figure 1) showed that weight loss during the intervention period was associated with increased weight regain during the break between November and January. This may be indicative of the efficacy of the intervention, as those who experienced most success also saw most recession when the guidance of the program was removed. Such a pattern is also evident in many other correlations; typically, a factor that promoted weight gain or loss during the intervention promoted the opposite during the break. For example, parent exercise intrinsic motivation was associated with child weight loss during the intervention, but weight regain during the break. Adversely, a high initial BMI z-score was correlated with more weight gain during the intervention and less weight regain during the break. Referring to this last correlation, it is possible that those who were already gaining weight during the intervention experienced a less dramatic spike in weight gain during the break. Another very important trend to consider is that of parent Weight Change 1 compared to child Weight Change 1. It appears that, based on the regression line in Figure 15, weight decreases in parents were associated with weight decreases in their children. This trend, although not statistically significant, supports the foundation of family-based interventions, which is that parent health behaviors have a strong impact on their children.

One of the more unexpected trends seen in the data was that high initial parent weight and BMI were correlated with greater child weight loss and decreased regain (Figures 19-22). It would seem more likely that parents with lower BMIs would have healthier lifestyles, as well as a better influence on their children. However, it is possible that these lower-BMI parents were less concerned about losing weight and therefore offered less encouragement toward their children. Parents with higher BMIs may have taken the intervention more seriously in an effort to lose weight, thus having a positive impact on their children's health. While there were several other variables which correlated unexpectedly with child weight gain, the R values were very small. It can only be speculated that in these cases, the considerable limitations of the study were responsible for confounding the results.

Because of the wide variation among participants, I found it helpful to examine a case study of two individual children whose parents had similar BMIs. It allowed me to search for differences between them which could not be explained by parental characteristics. Upon close scrutiny of the children's questionnaire responses, I found that the lower-weight child practiced health behaviors that are generally viewed as more beneficial for maintaining a healthy weight (Figure 44). Meanwhile, the health habits of the overweight child coincide with a less nutritious and physically active lifestyle. Ultimately, the case study revealed that there are several micro-level variables which may account for the difference in these two children's weight statuses. Everyday behaviors, such as drinking two-percent milk versus whole milk, have the potential to impact a child's entire health profile. A review article by Nicklas and colleagues discusses how small, additive changes that have occurred in society over time may be responsible for driving the obesity epidemic among children. For example, the authors find that children today drink more soft drinks and consume breakfast less frequently than they have in the past (Nicklas et al. 2001). Also, children now eat at home with their families less often and out at restaurants more (Nicklas et al. 2001). These daily behaviors, while minor in scale, seem to amplify the effects of

our current obesogenic environment. My individual assessment of these two children appears to demonstrate this concept.

Furthermore, this case study was useful in prompting new objectives for future research. It is speculated that the weight disparity between the two children could be attributed to differences in how they were raised rather than their parents' own health behaviors. It is possible that the overweight child had more access to unhealthy foods and sedentary activities because his or her parents allowed it. On the other hand, maybe the lower-weight child has parents who are strict about physical activity and healthy eating. Hence, research in this area should address family values and child discipline in addition to health behaviors specific to the parents. Questionnaires could include topics such as, "how much soda do you allow your child drink each day?" or "do you allow your child to watch television during most of his or her free time?" Future family-based interventions could search for significant correlations between these parenting characteristics and child weight status.

With new ideas such as these, childhood obesity researchers can tackle the epidemic from various approaches. Regardless of the approach taken, it is hopeful that studies like Activa y Sana will be extremely helpful in advancing research on child weight loss. It and other obesity interventions underway will play a crucial role in identifying effective weight loss methods and factors that promote weight maintenance. At stake are the lives of countless children and adults, and most at risk are underserved populations such as the low-income, Hispanic participants in Activa y Sana. It is important to remember that the obesity epidemic transcends all groups in terms of age, sex, race, and gender. Therefore, it is in the nation's best interest to continue pursing interventional studies in an effort to thwart the deleterious effects of obesity and prevent diseases like type 2 diabetes, cardiovascular disease, and cancer.

Conclusion

This study examined data from the Activa y Sana nutritional and physical activity intervention to find predictors of child weight loss. Specifically, it aimed to identify significant correlations between child weight loss and the health behaviors of his or her parents during an eight-week long family intervention. The results of the study showed that there were no significant correlations between child health variables and parent health variables

Several limitations—mainly the small sample size—help explain the lack of significant findings in the study. Speculations were made regarding the child-parent patterns that were observed in the comparisons made. For example, it was noted that child weight loss during the intervention was associated with parent weight loss, thus supporting the main theory behind family-based interventions. Additionally, a case study was performed to assess possible predictors of child weight status outside the realm of parent data. This analysis added value to the study in its scrutiny of everyday health behaviors of the child and its relevance to the future of weight loss studies. Despite the challenges researchers face in determining effective weight loss strategies for both children and adults, the outlook for future research remains hopeful.

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