ABSTRACT

RELATIONSHIP BETWEEN CHANGES IN MAXIMAL AEROBIC CAPACITY AND METABOLIC PROFILES IN OBESE YOUTHS

by Laura M. Wellbery

The increasing prevalence of obesity in youths is linked with the metabolic syndrome which greatly increases disease risk. The present study was done to assess the relationship between changes in aerobic fitness and changes in the metabolic profiles in obese youths after a clinical weight management intervention program. There were favorable changes in weight, VO2max, triglycerides, body composition, fasting insulin, total and LDL cholesterol levels. Additionally, weight change was a significant predictor of change in insulin, change in total cholesterol, and change in LDL cholesterol. In a subgroup of African American females, there were favorable changes in body composition and VO2max. In this subgroup, VO2max was a significant predictor of the change in insulin level. The results of the present study suggest that changes in weight and changes in aerobic fitness are related to more favorable metabolic profiles in obese youths.
RELATIONSHIP BETWEEN CHANGES IN MAXIMAL AEROBIC CAPACITY AND
METABOLIC PROFILES IN OBESE YOUTHS

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CHAPTER I

INTRODUCTION

Current estimates of the prevalence of obesity and overweight in youths are approximately 11% and 22% based on the 95th and 85th percentile for age and weight, respectively (54). Even more problematic is that overweight and obesity have been rapidly increasing in the United States in the last decade (54). Among African American youths, the estimates are even higher as obesity and overweight in African American females is estimated at 16.2-30.7% in ages 6-11 and 14.4-29.9% in ages 12-17 while obesity and overweight in African American males is estimated at 13.4-27.2% in ages 6-11 and 9.4-23.3% in ages 12-17 (54). This suggests that childhood obesity is a significant problem that needs to be addressed (54). Childhood obesity is associated with obesity in adulthood while obesity in adulthood is associated with an increased disease risk (54). Obesity among adults as well as among children is clearly linked with the metabolic syndrome, a clustering of hypertension, dyslipidemia, insulin resistance, and glucose intolerance which can lead to cardiovascular disease (CVD) and non-insulin dependent diabetes mellitus (NIDDM) (1,2,4-7, 16-17,22-37).

Insulin resistance is believed to be a main trigger in the development of the metabolic syndrome and appears to be more prevalent in overweight/obese individuals with central or abdominal obesity more strongly linked than overall obesity (1,2,4-8). Various studies in younger populations have shown that like adult obesity, obesity in childhood is associated with higher insulin levels suggestive of insulin resistance, the metabolic syndrome, and the eventual development of significant health consequences
(22-28). With adults, it appears that lifestyle modifications that include weight loss and increases in physical activity have positive impacts on metabolic abnormalities associated with the metabolic syndrome (39-47). Studies on children and the role of physical activity on metabolic factors have also been conducted which suggests that physical activity is beneficial in targeting facets of the metabolic syndrome even in youths (48-53). Since obesity in childhood is becoming more common along with these associated health consequences, the more physical activity’s role and specific mechanisms for improvement are explored, the greater the likelihood health risk can be reduced in this population. Therefore, the purpose of this study is to assess the relationship between changes in aerobic fitness (VO2max) and changes in body composition, fasting insulin levels, triglyceride levels, total cholesterol levels, LDL levels, and HDL levels in obese children.
CHAPTER II

REVIEW OF LITERATURE

The term insulin resistance describes the decreased ability of endogenous or exogenous insulin to regulate blood glucose levels as a result of a decreased response to the insulin (1,2). As tissue, particularly skeletal muscle which is responsible for 70-90% of glucose clearance, becomes more resistant to the insulin, hyperglycemia and or hyperinsulinemia can result (3). The inability to maintain glucose homeostasis due to insulin resistance is associated with the clustering of hypertension, dyslipidemia, and obesity which lead to the development of non-insulin dependent diabetes mellitus (NIDDM) and cardiovascular disease (CVD) with insulin resistance as a main impetus of this metabolic syndrome or insulin resistant syndrome (1,2,4-7). Debate exists however regarding the specifics of the relationship between insulin resistance with these various conditions. One theory holds that insulin resistance causes hyperinsulinemia which causes high blood pressure and high lipid levels by means of increased triglyceride synthesis, sodium retention, and increased sympathetic nervous system activity while another theory contends that altered lipid metabolism causes insulin resistance and the associated problems (2). Regardless of the nature of the relationship with these various conditions, insulin resistance is a multifaceted problem associated with health consequences that afflict numerous individuals and thus it is important to examine avenues of treatment for the insulin resistant syndrome.

Insulin sensitivity varies greatly across the population and even healthy individuals with normal glucose levels have different sensitivity levels to insulin and thus
different levels of insulin production (1,5). The explanation for this lies in the fact that insulin sensitivity is influenced by interactions of genetic and environmental factors (1,8). Genes that are implicated include genes related to glucose metabolism, insulin signaling, lipid metabolism, and insulin sensitization/desensitization (8). Environmental factors are significant on their own as well as in their interaction with genetic factors and include obesity, diet, and physical activity (1,8). Obesity, which is the most common clinical feature of the metabolic syndrome, is strongly linked with the development of insulin resistance with central or abdominal obesity more significantly linked than overall obesity (2,8). As the adipocyte hypertrophy that occurs in obese individuals progresses, insulin receptor density decreases, the cells become more resistant to insulin as they are deficient in $\alpha$-glycerophosphate, and plasma free fatty acids accumulate (3). The elevation in plasma free fatty acids stimulates gluconeogenesis, increases hepatic glucose output, inhibits muscle glucose clearance, and causes a high level of insulin to be produced which is essentially ineffective in lowering the plasma glucose level due to the accumulation of free fatty acids (3).

Central obesity is related to the release of the fatty acids which stimulates gluconeogenesis and hepatic glucose output and increases the supply of fatty acids to the periphery (2). This is combined with higher muscle triglycerides stores and impairment in the insulin-signaling pathway which impairs glucose oxidation and glycogen synthesis in muscle. Decreased insulin stimulated glucose uptake may result in hyperinsulinemia which causes downregulation of insulin receptors and results in peripheral and hepatic insulin resistance (1,2). The role of diet in insulin resistance relates to findings that reductions in caloric intake and weight loss in overweight individuals have a positive
impact on insulin resistance particularly when weight loss is from central abdominal fat (2,10,11).

Physical activity has been shown to have a major impact on insulin action which is why a sedentary lifestyle is associated with insulin resistance (2,12,13). Insulin resistance is impacted by physical activity indirectly by increasing body fat loss (12). Directly, physical activity acutely increases the effectiveness of muscle glucose transporters and can increase insulin sensitivity at least 16 hours after a single bout of exercise (13). Repeated training may prevent physiological and cellular changes associated with insulin resistance such as those related to abnormal hepatic glucose production, blood flow kinetics, and cellular events linked to glucose intolerance (3). Additionally, physical activity training that results in a large amount of energy expenditure can result in decreased visceral fat which will result in decreased release of free fatty acids and the eventual resultant hyperinsulinemia in adults as well as in children (1-2, 20, 53). These positive effects of physical activity highlight how physical inactivity figures in to the development of insulin resistance.

As the result of various factors affecting insulin sensitivity, insulin sensitivity levels can vary greatly even among healthy individuals. Goaly et al. found that glucose uptake varied 3-fold in non-diabetics with normal fasting plasma glucose levels (14). Additionally, some subjects with normal glucose levels had similar glucose uptakes as subjects with impaired glucose tolerance (fasting plasma glucose between 110-126 mg/dL) and subjects with NIDDM (fasting plasma glucose above 126 mg/dL) (3,4,14,15). The explanation for why some apparent insulin resistant subjects are able to maintain glucose homeostasis while others cannot lies in the fact that some individuals
can secrete enough insulin to compensate for the insulin resistance (4). Individuals with varying levels of insulin resistance who maintain normal glucose levels have β-cells that can effectively modify how much insulin is secreted to keep glucose levels normal. Individuals who are glucose intolerant or have NIDDM also vary in their glucose tolerance, which can be explained by this ability or inability to maintain and utilize high levels of insulin (4). When the higher levels of circulating insulin cannot be maintained, circulating free fatty acid levels rise, hepatic glucose production rises, and the insulin resistant subject becomes hyperglycemic (4). It appears to be best to have a higher sensitivity to insulin as insulin resistance is a precursor to the development of various diseases, but when an individual is actually insulin resistant, the inability to maintain high levels of circulating insulin due to β-cell impairment causes reduced plasma insulin levels and eventually NIDDM (3,4).

Insulin resistance is also associated with hypertension, dyslipidemia, and CVD. Hypertension is common in obese and NIDDM individuals which suggests a defect in glucose uptake in hypertensive individuals (4,5). The mechanism behind the hypertension appears to be related to hyperinsulinemia which induces kidney sodium retention and increases sympathetic nervous system activity (4,5). Hyperinsulinemia is also implicated in the development of lipid abnormalities as it is associated with increased VLDL, increased LDL, and decreased HDL which are all risk factors for coronary artery disease (5). Insulin affects arterial tissue by causing proliferation of smooth muscle cells, by enhancing cholesterol synthesis and LDL receptor activity, by increasing the formation of lipid plaques, by stimulating connective tissue synthesis, and by stimulating growth factors which all contribute to the atherogenic process (5).
Looking at the role of insulin in the atherogenic process, Ronnemaa et al. examined the association between fasting plasma insulin levels and coronary heart disease in healthy and NIDDM adults ages 45-64 (16). All subjects who had various manifestations of coronary heart disease had higher fasting insulin levels suggesting insulin’s contribution to atherosclerosis (16). A related population based study of younger, healthy individuals known as the CARDIA study examined the relationship between fasting insulin levels and CVD risk factors in healthy black and white adults (17). In all race-sex groups (black, white, ages 18-24, ages 25-30), insulin was positively correlated with blood pressure, triglycerides, total cholesterol, and LDL cholesterol while it was negatively correlated with HDL cholesterol. The magnitude of each of these relationships was relatively small, but the fact that insulin levels in a healthy, young population was associated with several CVD risk factors before abnormalities exist suggests that even at a young age, higher levels of insulin may have an impact on CVD risk. The higher levels of insulin indicative of insulin resistance as seen in these two studies is believed to have an indirect role in atherosclerosis by means of the effects on blood glucose levels, lipid metabolism, and blood pressure while it directly causes damage to the arterial walls (17). Regardless if hyperinsulinemia leads to atherosclerosis indirectly or directly, non-pharmacological treatments that reduce insulin resistance by reducing hyperinsulinemia such as weight loss, physical activity, and proper diet may impact CVD risk even at a young age (17).

While Manolio et al. found correlations between insulin levels and CVD risk factors in all groups, Haffner et al. examined differences between ethnic groups in insulin secretion and insulin resistance to determine if certain groups were more at risk (18).
Based on the fact that African Americans and Hispanics have two to three times the risk of developing NIDDM when compared to non-Hispanic whites, the study looked at the insulin profiles of the three ethnic groups. The African Americans and Hispanics had higher fasting insulin concentrations, higher 2-hour insulin concentrations, higher acute insulin responses (insulin secretion) and lower insulin sensitivity levels than the non-Hispanic whites. However, after adjustments were made for obesity, body fat distribution, and behavioral factors, it was only the African Americans who had higher fasting insulin concentrations, higher 2-hour insulin concentrations, higher acute insulin responses, and lower insulin sensitivity levels than the non-Hispanic whites (18). It was concluded that the differences for the Hispanics were due to obesity and body fat distribution but it was unclear what accounted for the differences with the African Americans as differences still existed even after adjustments were made for obesity and body fat distribution. Osei et al. compared blacks and whites and also found higher secretions of insulin in the blacks when compared to the white subjects (19). The study reasoned that the differences are due to decreased hepatic insulin extraction and clearance in the black subjects, which helps explain the higher incidence of obesity and NIDDM in this population (19).

Visceral fat accumulation also appears to be an important determinant of insulin resistance in adults in various ethnic groups. Higher levels of visceral fat and not just overall obesity are related to hyperinsulinemia, insulin resistance, glucose intolerance, hypertriglyceridemia, reduced HDL cholesterol, increased apolipoprotein B levels, and increased LDL cholesterol that is very small and dense (20). Yamashita et al. found that subjects with greater visceral fat obesity as compared to subcutaneous fat obesity had
higher triglycerides, higher total cholesterol, higher glucose levels, and higher blood pressures (21). Visceral fat accumulation was also associated with coronary artery disease as it was related to more risk factors and it was associated with complications even in non-obese subjects (21). The study concluded that accumulation of visceral fat is related to sex hormones, aging, genetic factors, dietary factors, and inactivity which goes along with other researchers’ speculations regarding visceral fat and the resultant metabolic profiles (20,21). Visceral fat affects insulin resistance as the high levels of free fatty acids increase hepatic triglyceride synthesis and induce insulin resistance, which causes glucose intolerance and atherosclerosis (21).

The relationship of fat distribution and disease risk has also been studied in younger populations. Several studies have examined childhood weight and the development of metabolic abnormalities later in adulthood (22-24). Vanhal et al. examined height and weight at age 7 compared to adult height and weight and the occurrence of the metabolic syndrome (obesity, hypertension, hypercholesterolemia, and insulin resistance). While low birth weight was not associated with the metabolic syndrome as an adult as suggested in the past, half of the obese children became obese adults and childhood obesity increased the risk of metabolic abnormalities as an adult. The most interesting finding was that adults who became obese as adults and not in childhood had a lower risk for the metabolic syndrome when compared to obese adults who were obese as children. Steinberger et al. found that childhood BMI was positively correlated with BMI in adulthood, total cholesterol in adulthood, and LDL cholesterol in adulthood but BMI in childhood was negatively correlated with glucose utilization in adulthood (23). It appears that hyperinsulinemia as a child is related to obesity that
persists into adulthood as insulin is a growth factor and the genes that influence lipid storage and mobilization in the abdominal area in particular may contribute to the problem (24,25).

In examining the role of body fat distribution in hyperinsulinemia in children, the Bogalusa Heart Study collected data on glucose and insulin levels after an oral glucose tolerance test in black and white children (26). The study found that insulin response is greater in overweight children with more central body fat as compared to more peripheral body fat which the researchers proposed puts these individuals at more of a risk for hyperinsulinemia and associated conditions in adulthood (26). Legido et al. found similar results in obese prepubertal girls implicating central obesity in metabolic abnormalities (27). Possible explanations for why central obesity is associated with more metabolic abnormalities include increases in cortisol and insulin secretion and decreases in growth hormone and testosterone secretion. Cortisol and insulin increase lipid accumulation while growth hormone and testosterone aide in lipid metabolism (28). The effects of these hormones on visceral adipose tissue are greater as visceral fat has more cells per unit mass and increased blood flow when compared to subcutaneous fat tissue (28).

Several studies have examined ethnic differences in children regarding body fat distribution and metabolic complications (29-31). Goran et al. found that visceral fatness in children varies a great deal with a lower amount of visceral fat in African American children compared to white children (29). The researchers of this study reasoned that differences in sex hormones may contribute to the differences in the ethnic groups and that other research should explore this issue. Another study by Gower et al. found that differences in body fat or fat distribution in African American children when compared to
white children could not explain the greater fasting and postchallenge insulin concentrations of the African American children (30). The study additionally found that insulin levels were related to adiposity regardless of ethnicity, suggesting that obesity is related to disease in both groups (30). A subsequent study that found that obese and African American children were more insulin resistant which could not be explained by body fat distribution (31). Schuster et al. also found that African American adolescents had greater insulin resistance levels than white adolescents (32). Visceral fat was however related to triglyceride levels and fasting insulin levels which was the same for both ethnic groups in the Gower et al. study (31).

In a longitudinal study, Ronnemaa et al. collected insulin data on Finnish subjects ages nine to 24 (33). Insulin levels peaked during puberty and declined after age 21 while insulin levels were positively correlated with BMI, triglycerides, and blood pressure and were inversely correlated with LDL cholesterol (33). Obesity was related to insulin levels and CHD risk factors, which the authors reasoned illustrated the point that obesity at a young age can have serious health consequences later on as marked by the CHD risk factors present (33). Data from the Bogalusa Heart Study was also used in a cross-sectional approach to examine individuals from young childhood to early adulthood for the metabolic markers of fasting insulin levels, serum lipids levels, and serum lipoprotein levels (34). In all ages studied, fasting insulin levels were again positively correlated with LDL cholesterol and triglycerides while insulin levels were negatively correlated with HDL cholesterol (34). Additionally, this study found that insulin levels were associated with adverse lipoprotein levels increasingly among obese individuals.
with stronger associations existing in young adults, suggestive of an increased cardiovascular disease risk in adulthood (34,35).

To examine why hyperinsulinemia and obesity are so frequently associated as noted by many of the above studies, Odeleye et al. studied Pima Indian children, a culture that is at an increased risk of NIDDM (36). Researchers found that in the sample studied, fasting plasma insulin levels in the boys and girls (ages 5-9 when first tested) correlated with the rate of weight gain (ages 15-19 when later tested) (36). The hyperinsulinemia is believed to lead to weight gain by promoting rapid fat storage as noted by the thrifty genotype hypothesis (36). Hoffman et al. however showed that by decreasing weight by caloric restriction in obese children, increases in insulin sensitivity could be achieved (37).

When examining insulin resistance in children, it is important to consider the fact that puberty is associated with a decline in insulin sensitivity (38). In a unique longitudinal study, Goran et al. followed 60 pre-puberty black and white children for two years to determine how insulin sensitivity changed in this group with the onset of puberty (38). The researchers found that when progression from Tanner stage I to Tanner stage III or IV (indicative of pubertal development) occurred, insulin sensitivity decreased by 32% and the pubertal transition was associated with increases in fasting glucose, fasting insulin, and acute insulin response (38). As the results were consistent across sex, ethnicity, and obesity level, the findings suggest that early and pubertal development and a longer period of development may leave a child with a greater risk of significant insulin resistance and associated metabolic complications due in part to possible beta cell burnout (38). The study found that in the more obese subjects, puberty occurred earlier
which may be a risk factor for NIDDM as hypothesized by the authors (38). Dietary and exercise interventions are cited in the study as possible steps to minimize the risk of pubertal insulin resistance developing into significant metabolic health consequences such as decreased insulin sensitivity, increased fasting glucose levels, increased fasting insulin levels, and increased acute insulin response (38).

Exercise intervention studies regarding adults and facets of the insulin resistance syndrome are in abundance (39-47). In a recent study of elderly men, individuals with just 30 min a day or more of moderate physical activity such as bicycling or gardening had a lower incidence of glucose intolerance independent of body fatness (47). Additionally, men who maintained activity for the five-year follow up had a higher glucose tolerance (47). In a group of middle aged and older men, a study by Prately et al. found that with nine months of moderate intensity aerobic exercise along with caloric increase to prevent weight loss, insulin responses to an oral glucose tolerance test declined by 16% (39). Also important was the finding that changes in percent body fat and waist circumference were independent predictors of the changes in insulin response partly due to a decrease in abdominal fat (39). The authors note that the mechanisms may not be fully understood yet, but the results suggest that regular aerobic exercise in this group can improve metabolic profiles and subsequently lessen the risk of NIDDM and atherosclerosis (39). A study by Tuomilehto et al. confirmed that lifestyle modifications (diet and exercise) can in fact lessen the risk of the development of NIDDM in at risk individuals as members in the intervention group had fewer cases of NIDDM and were more likely to have improvements in glucose tolerance (46). Oppert et al. in a study of identical twins also reasoned that changes in abdominal visceral fat
induced by a negative energy balance with exercise are associated with reductions in plasma insulin levels and improvements in insulin sensitivity (42).

In a study of obese men compared with age matched non-obese sedentary men, six months of aerobic exercise coupled with weight loss yielded declines in blood pressure and improvements in glucose and lipid metabolism for the obese men (40). The mean number of abnormalities associated with insulin resistance declined from 4.4 to 2.2 in each individual (40). Additionally, the study found that subjects with the greatest improvements in VO2max had the greatest increases in insulin sensitivity and the greatest declines in plasma insulin levels while the subjects with the greatest loss in body fat had the greatest improvements in glucose tolerance (40). A similar study that was conducted earlier by Dengel et al. found that both aerobic exercise training and weight loss are necessary to achieve maximal metabolic improvements which agrees with the later findings mentioned above (40,41). Out of the four training groups in the study (weight loss alone, exercise alone, weight loss and exercise, control group), only the weight loss and exercise group had improvements in glucose tolerance and insulin sensitivity which suggests weight loss and aerobic training affect metabolic control through different mechanisms (41). The researchers reasoned that the weight loss reduced fat tissue which increased insulin sensitivity and that the aerobic training enhanced delivery of insulin and glucose to muscle tissue, enhanced insulin-mediated muscle blood flow, enhanced glucose extraction, and increased the amount of glucose transporters in the muscle (41).

In a later study of exercise training and diet, it was found that diet combined with exercise induced weight loss by aerobic or resistance training lowered fasting insulin level and insulin response to a five hour 75 gram oral glucose tolerance test greater than
by diet alone by means of reductions in visceral and abdominal subcutaneous adipose
tissue (45). The study concluded that a 1,000 kcal energy restriction by means of either
resistance or aerobic exercise along with diet will aide in aspects of insulin resistance for
obese men (45). However, a study of forty-five obese women found that weight
reduction did improve hyperinsulinemia, but exercise training did not yield any additional
benefits (43). The lack of improvement from exercise was attributed to a function of two
factors including a low frequency of training at a low intensity and the rapid weight
reductions induced by the very low calorie diet of the study (925 kcal/day) which may
have hidden any possible effects of the exercise (45). The Oslo Diet and Exercise Study
however found a beneficial effect on insulin resistance with diet and exercise for a group
of both men and women with significant cardiovascular risk factors (44). The
intervention study produced declines in insulin resistance and there was a positive
correlation between changes in insulin resistance and changes in BMI (44).

Similar studies of the role of physical activity on metabolic factors have been
conducted with children and adolescents (48-53). In a study of black and white children
ages 7-11 with a wide range of fatness, the relationship between fitness level and body fat
percentage was assessed and a multitude of insights were yielded (51). The percentage of
body fat measured by a DEXA scan was positively related to insulin level and the
atherogenic index (comprised of total cholesterol, HDLC cholesterol, apoprotein B, and
apoprotein A-1) while VO2max was negatively correlated with the atherogenic index
(51). Fatness was a significant predictor of the atherogenic index and insulin levels but
VO2max by itself was not a significant predictor of the atherogenic index and insulin
levels which suggested that VO2max exerts its influence on these variables through its
impact on body fat (51). Body fat distribution did not have an impact which the authors attributed to the fact that the population studied was very young and that fat distribution may have more of an impact in later years of childhood and adolescence (51). Finally, the study found that black children had higher insulin levels when compared to the white children even after body fat was accounted for (51). When a limited number of black girls in the 7-11 age group were studied on an exercise and lifestyle education intervention, the physical activity improved fitness and body fat while the lifestyle education improved diet (52). As risk factors for both NIDDM and CAD were impacted in this group, the researchers concluded that exercise training and lifestyle education may be helpful in impacting risk factors in this group and that further study in this area is warranted to determine the ability to generalize the results to various other clinical settings (52).

In another group of obese black and white 7-11 year old children, the roles of exercise training and detraining in the absence of dietary intervention on body composition and CAD/NIDDM risk factors were assessed in a modified cross-over design intervention study (53). While in the exercise training group, the children engaged in 4 months of group exercise (HR at 150bpm) 5 days a week for 40 minutes a session (53). The physical training was associated with improvements in body fat percentage, visceral adipose tissue, subcutaneous adipose tissue, insulin levels, triglyceride levels, leptin levels, and cardiac parasympathetic activity (53). Following 4 months of detraining, there were increases in leptin levels and negative changes in percent body fat (53). The researchers reasoned that the impact of the physical training primarily occurred by means of its influence on body fat (53). When a similar design
with a similar population was employed to explore the role of exercise training on components of the insulin resistance syndrome without dietary intervention in obese children, results indicated that the exercise training for four months decreased triglyceride levels, decreased insulin levels (with increased insulin sensitivity), and decreased body fat percentage (50). This study also indicated that with the detraining period of four months, the improvements were lost (50). Studies such as the ones discussed here (50-53) that include exercise training are important as it has been documented that children exhibiting characteristics of the metabolic syndrome have low cardiorespiratory performance (49). By seeking to improve these low fitness values in the children, it is clear that health benefits can be achieved which will decrease the risk of various serious diseases.

Obesity in childhood and the related complications associated with it are part of an increasing problem that needs to be addressed as current estimates of the prevalence of obesity and overweight in youths are approximately 11% based on the 95th percentile and 22% based on the 85th percentile with an increasing trend in the United States in the last decade (54). In agreement with adult studies, it appears that visceral fat, which accumulates early in childhood, is associated with a higher disease risk than subcutaneous fat in obese children and adolescents than non-obese and younger children and is more closely associated with insulin resistance, dyslipidemia, CVD, and NIDDM (55,56). In a study conducted in the greater Cincinnati, OH area, a dramatically increasing incidence of NIDDM among adolescents was found which was related to a strong family history of NIDDM and obesity (57). This agrees with a more recent study in the UK where increases in the occurrence of NIDDM in children, though not as
dramatic as in the United States, is taking place with family history and obesity again as
the main culprits (58). Physical activity and fitness level may also be an important
variable related to the increased incidence of NIDDM in youths as is the case with adults.
Yet, research is lacking on these relationships with youths and needs to be addressed to
better understand the development of NIDDM in younger populations.

Obesity as an adult is associated with increased disease risk, and as obesity as a
child is associated with increased risk of obesity as an adult (54). Consequently, reducing
the problem in childhood should receive attention. As illustrated by the review of the
exercise intervention studies provided in this paper, physical activity does appear to have
a significant role in targeting components of the insulin resistance syndrome even in
young children. The more that this area is explored and the better the understanding of
the mechanisms of physical activity in improving the multifaceted phenomena of the
insulin resistance syndrome, the greater hope there is for the improvement in the short
term and long term complications of the increasing metabolic abnormalities in children.

**Study Purpose**

The purpose of the present study is to assess the relationship between changes in
aerobic fitness (VO₂max) and changes in body composition, fasting insulin levels,
triglyceride levels, total cholesterol levels, LDL levels, and HDL levels in obese children.
As there is an alarming increase in the occurrence of obesity as well as the development
of NIDDM in childhood, it is important to determine what can be done to optimize health
among these individuals which will lessen the development of acute and long-term health
complications associated with excess weight and/or low fitness and physical activity
levels. Based on the abundance of the existing literature on adult populations, it is clear that increasing physical activity can have a beneficial effect on metabolic abnormalities in a multitude of ways. While research on this topic in younger populations does exist, the dramatic increase in childhood obesity and related complications is still a relatively recent phenomena and one that is not fully understood. By increasing knowledge on what can be done to improve health in this population, it is conceivable that the knowledge will translate into practice to target the existing short-term and long-term metabolic complications that exist in many children.

Hypothesis

The hypothesis of the present study is that with increased aerobic fitness (VO₂ max) by means of an exercise and weight management program including group exercise and lifestyle education, there will be favorable changes in the following: body composition, fasting insulin levels, triglyceride levels, total cholesterol levels, LDL levels, and HDL levels. Additionally, with decreases in body weight and body mass index, there will be favorable changes in the following: body composition, fasting insulin levels, triglyceride levels, total cholesterol levels, LDL levels, and HDL levels.
CHAPTER III

METHODS

Research Design

The present study is classified as a longitudinal and retrospective study. During the course of the study which involves Phase I of the Healthworks Weight Management Program, all subjects complete pre- and post-tests which includes a graded maximal exercise test to measure VO₂max, a dual energy x-ray absorptiometry scan (DEXA scan) to measure body fatness and lean mass percentage, fasting blood analysis of glucose, insulin, triglycerides, total cholesterol, LDL cholesterol, HDL cholesterol, and a general physical exam. Phase I of the HealthWorks program, which is the time span of the present study, consists of a diet and exercise intervention program that lasts approximately 16-20 weeks. The time of the intervention period (Phase I) varies somewhat because during this time, the youth and a parent/guardian must meet with a case manager every other week for a total of six meetings. It is not until all of the meetings have taken place that a youth can complete all of the post-testing and thus, complete Phase I of the program and the intervention period of the present study.

Participants

Participants in the study include children and adolescents enrolled in the Cincinnati Children’s Hospital HealthWorks Weight Management Program. The HealthWorks Weight Management Program is an outpatient, family-centered, behavioral weight management program for overweight and obese youths in which patients and families are seen by a physician, a registered dietician, a psychologist, an exercise
physiologist, and a nurse throughout the duration of the program (68). There were a total of 186 participants, yet the number of participants measured for each variable in the study varies as seen in Table 2 and Table 4 as the measures were conducted clinically and it was not possible to ensure that all participants were measured on every variable. The distribution of participants included males and females as well as African American and Caucasian youths in the program with an age range of 6-18 years as shown in Table 1.

Entrance into the HealthWorks program occurs by means of a physician referral for a youth who meets all of the criteria outlined for the program. To be eligible for the program, the youth must have a body mass index greater than the 95th percentile for age and sex or a body mass index greater than the 85th percentile if another co-morbidity is present. The body mass index values for these percentiles are described elsewhere (59).

Measurement of body composition

Measurement of body composition is obtained by means of dual energy x-ray absorptiometry (DEXA scan) which estimates the percent body fat and lean mass by dividing the body into fat, bone, and fat-free soft tissue compartments. The test is conducted at the Children’s Hospital by a qualified x-ray technician using a Hologic 4500 (Waltham, MA) whole body scanner. The DEXA scan is a good technique for the purposes of this research as it is easy to administer and has high reliability (60).

Assessment of Aerobic Fitness Level

Aerobic fitness level is assessed for each participant by means of a standard maximal treadmill exercise test with a 12-lead EKG. Participants are informed of the
procedures prior to the start of the exercise test and are acquainted with all of the
equipment. A Modified Balke protocol is used as it allows for a more gradual warm-up
period and may be more tolerable by youths as it is a walking test. The protocols are as
follows:

<table>
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<th>Duration (Min)</th>
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<th>Elevation (%)</th>
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<tr>
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<td>3</td>
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<td>20</td>
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<td>4</td>
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<tr>
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<td>3</td>
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<tr>
<td></td>
<td>3</td>
<td>3.7</td>
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The 12-lead EKG uses usual placement with RA, LA, RL, LL, and V1-V6. Heart
rate and EKG tracings are recorded during supine, standing, standing with
hyperventilation, supine with hyperventilation, during each minute of exercise and
immediately post-exercise, 1, 3 and 5 minutes post-exercise. Blood pressure is recorded
during supine, standing, the last minute of each exercise stage, immediately post-
exercise, 1,3, and 5 minutes post-exercise. VO$_2$, VCO$_2$, RR, and RMV are measured at
rest while standing and during each stage of exercise beginning at the first minute until
the end of the stage. These variables were measured with standard open circuit spirometry procedures on a Parvomedics True Max 2400 metabolic cart (Sandy, UT). Additionally, RPE is measured at 1.5 minutes into each exercise stage. A test is completed after at least two of the following criteria is reached: estimated maximal heart rate is reached, there is no additional increase in VO₂ despite increases in workload (plateau), there are signs of exertional intolerance, an RER of 1.0-1.2 is reached, volitional failure is reached. If the test is determined to not be a true maximal test, further testing on another occasion is conducted (From Healthworks Weight Management Program Exercise Test Protocol).

Measurement of Lipids and Insulin

After a twelve hour fast, blood was drawn on each participant to obtain an insulin and lipid profile; insulin level, total cholesterol level, HDL cholesterol level, LDL cholesterol level, and triglyceride level were measured. The analyses were conducted in the Cincinnati Children’s Hospital Medical Center clinical laboratory. Insulin levels above 15 µU/ml were considered abnormal and were based on pediatric age range standards. Standards from the National Cholesterol Education Program Pediatric Panel report were utilized to determine abnormal lipids and lipoproteins (69).

Exercise Intervention Program

Children and adolescents attend group exercise sessions that take place at the Cincinnati Children’s Hospital Medical Center, which consists of 60-minute exercise sessions with moderate physical activity (aerobic and light resistance training). All participants continue in the group exercise sessions for approximately 16-20 weeks and
are required to attend group exercise sessions at least once a week. The sessions have been offered 3 to 5 nights a week and subjects are encouraged to attend as many nights as possible as well as to increase physical activity outside of the program through the development of individualized behavioral goals. The goal of the HealthWorks program is to make physical activity fun which will encourage the children to continue to be physically active when away from group exercise to increase daily energy expenditure and aerobic fitness.

Data Analyses

Analyses of the data include: paired t-tests to examine changes in the desired outcome variables for the population studied (percent body fat, insulin levels, log of insulin levels, body mass index, percent lean mass, VO₂max (ml (min)^{-1}), VO₂max (ml (kg · min)^{-1}), total cholesterol, triglyceride levels, LDL cholesterol, and HDL cholesterol). Additionally, stepwise linear regression analyses were completed to determine which variables were related to a particular outcome variable such as how changes in VO₂max levels are predictive and related to changes in insulin levels.
CHAPTER IV

RESULTS

Table 2 is a table of means and paired t-tests and shows that for the children and adolescents studied, there were significant changes in all variables measured except for HDL cholesterol (Table 2). These changes are illustrated in Figures 1-11 which exhibit the changes in pre-test values and post-test values for the total population studied for the following variables: weight, absolute VO2max, relative VO2max, total cholesterol level, triglyceride level, LDL cholesterol level, HDL cholesterol level, percent body fat, body mass index, percent lean mass, and insulin level (Figure 1-Figure 11).

A stepwise linear regression analysis was conducted for the total population studied to determine which variables were potentially related to the change in insulin level (log insulin level, linsulind), total cholesterol level (dtc), triglyceride level (dtg), LDL cholesterol level (dldl), HDL cholesterol level (dhdl), body mass index (bmid), percent lean mass (pleand), and percent body fat (pbfd). This analysis suggested that the weight difference (wtd) or the change in weight entered into the model as a significant predictor of the log change in insulin (linsulind) (p<.05) and accounted for approximately 5% of the variability ($r^2 = .0463$) (Table 3). Race entered into the analysis as a second significant predictor of the log change in insulin (linsulind) (p<.05) accounting for 4.2% of the variability ($r^2 = .0417$) (Table 3). Age, gender, and VO2max were not significant predictors of this analysis and therefore were not a part of this model.

A stepwise linear regression analysis suggested that weight difference was significantly related to the change in total cholesterol (dtc) (p<.001) and the change in
LDL cholesterol (dldl) (p<.01) accounting for 12% of the variability and 9.2% of the variability, respectively \((r^2=0.1202, r^2=0.0918)\) (Table 3). Age, race, gender, and VO2max were not significant predictors of the change in total cholesterol and the change in LDL cholesterol in this analysis and therefore were not a part of this model.

In a subgroup of African American females studied \((N=38)\), there were significant changes in percent body fat, body mass index, percent lean mass, absolute VO2 max \((\text{ml} \text{(min)}^{-1})\), and relative VO2 max \((\text{ml} \text{(kg·min)}^{-1})\) as indicated in Table 4. There were no significant changes in insulin level, log of insulin level, total cholesterol level, triglyceride level, LDL cholesterol level, or HDL cholesterol level (Table 4). These changes are illustrated in Figures 13-23 which exhibit the changes in pre-test values and post-test values for the subgroup of African American females studied for the following variables: weight, absolute VO2 max, relative VO2 max, total cholesterol level, triglyceride level, LDL cholesterol level, HDL cholesterol level, percent body fat, body mass index, percent lean mass, and insulin level (Figure 14-Figure 25).

In a subgroup of African American females, a stepwise linear regression analysis was used to determine which variables were potentially related to the change in insulin level (log insulin level, linsulind), total cholesterol level (dtc), triglyceride level (dtg), LDL cholesterol level (dldl), HDL cholesterol level (dhdl), body mass index (bmid), percent lean mass (pleand), and percent body fat (pbfd). This analysis suggested that the difference in relative VO2 max \((\text{dvo2ml} \text{(kg·min)}^{-1})\) entered into the model as a significant predictor of the log change in insulin \((p<.01)\) accounting for 36.7% of the variability \((r^2=0.3667)\) (Table 5). Figure 12 illustrates how in this subgroup, as relative VO2 max \((\text{ml} \text{(kg·min)}^{-1})\) increases, insulin levels decline. Age and weight were not
significant predictors of the changes in log insulin levels as part of this analysis and therefore were not a part of this model. The stepwise linear regression analysis suggested that the change in weight and initial absolute VO₂max (ml (min)⁻¹) together were significantly related to the change in total cholesterol (p<.05) accounting for 33.2% of the variability (r²=.3667) (Table 5). Age was not a significant predictor of the changes in total cholesterol as part of this analysis and therefore was not a part of this model.
CHAPTER V

DISCUSSION

As illustrated in Table 2, there were favorable significant changes in the total population studied in percent body fat, insulin level, body mass index, percent lean mass, absolute VO$_2$ max (ml (min)$^{-1}$), relative VO$_2$max (ml (kg · min)$^{-1}$), triglyceride level, LDL cholesterol level, and body weight (Table 2). This is consistent with previous research that shows that lifestyle intervention programs with physical activity positively affect health related variables such as the variables in this study that are components of the metabolic syndrome in adults and youths (10-12, 20, 27, 39-48, 50, 52-53). There were no significant changes in HDL cholesterol in this group which can be supported by previous research that indicates that changes in HDL cholesterol from physical training are dependent on the length of the training program, the volume of training completed, changes in body composition, dietary intake, and weight loss (61). Sasaki et al. demonstrated that HDL cholesterol can increase with a two-year aerobic training program, but this study on obese children consisted of a protocol of running at 70% of maximal oxygen uptake for each child and the intensity was carefully monitored (62). There appears to be a dose-response relationship with activity and changes in HDL cholesterol and the research shows that HDL cholesterol may not be elevated with lower volume training programs such as the one employed in the present study (61).

Weight loss was significantly related to changes in insulin levels, changes in total cholesterol, and changes in LDL cholesterol. As it is plausible that the weight loss represented some loss in central abdominal fat, it is likely that these improvements in
insulin, cholesterol, and LDL cholesterol were due in part to improvements in insulin-signaling pathway, glucose oxidation, glycogen synthesis mechanisms which impacted insulin sensitivity, insulin level and subsequently cholesterol levels as the lower insulin levels caused decreased triglyceride synthesis (1-2,10-11, 63).

It is interesting to note that with the total population studied, the change in fitness level was not related to the change in insulin level as was originally hypothesized. One possibility may be that the initial insulin were not elevated enough for the changes in fitness to have a significant effect on the insulin levels. Perhaps for moderate physical activity and subsequent changes in physical activity to have an impact on insulin levels, the level of insulin needs to be at a higher threshold initially than what was observed for the total participant group studied. In a recent study by Kang et al., obese adolescents who were engaged in high intensity physical activity had significant improvements in triglyceride level, VLDL cholesterol level, and the total cholesterol to HDL ratio where subjects with the least favorable pre-intervention values had the most significant improvements (64). The high intensity physical activity did not however have an effect on insulin level in the group of obese adolescents and while the authors of this study did not speculate as to why a relationship was not discovered, it may be due to pre-intervention insulin levels that were not at a high enough threshold (64). Race was the only variable related to the change in insulin level when the regression analysis on the entire population studied was completed. Despite the fact that this relationship was not found with the statistical analyses run, physical activity most likely did have a role in impacting insulin level as increased physical activity levels did result in increased energy expenditure and thus occurred along with weight loss.
In the subgroup of African American females however, the change in fitness level was significantly related to the change in insulin level as illustrated in Figure 12. These results need to be examined in light of the fact that African American females are a particularly at-risk group as they tend to have higher insulin levels, lower fitness levels, and higher incidences of all aspects of the metabolic syndrome (19, 29-30, 32, 51). African Americans have a 2-3 times greater risk of developing NIDDM as they have been shown to have decreased hepatic insulin extraction, decreased glucose clearance, greater insulin resistance, and greater fasting and post-challenge insulin concentrations (18-19, 30, 32). In pre-puberty, African Americans have higher insulin levels than white children, yet after puberty which is characterized by temporary insulin resistance, black adolescents have a lower insulin sensitivity and higher insulin levels than white adolescents (65). In African American females in particular, when compared to males or whites, insulin concentrations are higher (66). These higher insulin concentrations observed in African American females appear to be due initially to decreased hepatic insulin clearance from impaired insulin extraction from reduced hepatic insulin receptor function rather than hyperinsulinemia, but this decreased clearance is what eventually leads to elevated insulin levels and subsequent complications (66). Genetic defects with genes such as those associated with glucose metabolism, insulin signaling, lipid metabolism, and insulin sensitization may play an equally important role along with various environmental factors such as diet and physical activity increasing the risk for the African American females. In this light, this finding is extremely significant as it shows that moderate physical activity in this group can impact fitness levels and insulin levels which can subsequently lower the occurrence of the metabolic syndrome. This is in line
with findings by Arslanian et al. that with increased physical fitness levels in African American children, differences in insulin secretion and sensitivity between the African Americans and white children disappear, suggesting the impact that fitness has on the African American subjects (65).

Possibilities why these relationships were found in the African American females and not in the total population studied include the fact that the African American females had higher insulin levels to begin with while the total population studied may not have had high enough values to see an effect over such a short time span (Table 6). Along the same lines, the African American females had lower fitness values than the total pool of participants as a whole and thus, the moderate physical activity may have been able to exert more of an effect on the African American females than on the other participants (Table 6). Additionally, these results suggest that changes in fitness level may have more of an impact on insulin levels when insulin levels are at more of an elevated state whereas changes in fitness may have less of an effect when insulin levels are at lower levels at the start of the intervention (Table 6).
CHAPTER VI

FUTURE CONSIDERATIONS

From the findings of the present study, future research can be directed to examine the impact of a longer intervention period than the 16-20 week period utilized in this study. Perhaps a longer intervention with higher intensity exercise as that employed by Sasaki et al. or Kang et al. would yield more significant changes especially in the total subject pool as opposed to just the at risk subgroup of African American females (62,64). The rationale behind the Kang et al. study was that 2-3 months may be too short of a time period to discover changes in components of the insulin resistance syndrome and thus, with a longer intervention period with the subjects in the present investigation there may be more significant improvements in the measured outcome variables (64). Additionally, attendance to the exercise sessions would also be useful in understanding the role of the amount of physical activity in changing components of the metabolic syndrome in obese children and adolescents. Other directions for future research from the present study include different ways of analyzing the data. Methods employed could include multivariate multiple regression analysis where several predictor variables would be used with several dependent variables to get a better understanding of how the predictor variables relate to the dependent variables. In this type of analysis, predictor variables such as age, race, gender, fitness, weight change, relative VO\textsubscript{2}max (ml (kg \cdot min)\textsuperscript{-1}) change, and body mass index change could be used with the outcome dependent variables of insulin, total cholesterol, triglyceride level, HDL cholesterol level, and LDL cholesterol level. The fact that a wide age range was used may have caused problems as
subjects were at different pubertal stages which is known to impact insulin levels and insulin sensitivity (38). Controlling for this could allow for the discovery of more significant relationships.

Overall, the findings from the present study are important in understanding the metabolic syndrome through body weight and fitness changes in obese children and adolescents. In the total population studied, the intervention program resulted in significant improvements in percent body fat, insulin level, body mass index, percent lean mass, absolute VO$_2$max (ml (min)$^{-1}$), relative VO$_2$max (ml (kg · min)$^{-1}$), total cholesterol level, triglyceride level, and LDL cholesterol. Change in weight was significantly related to change in insulin level, change in total cholesterol, and change in LDL cholesterol. In the subgroup of African American females, there were significant changes in percent body fat, body mass index, percent lean mass, absolute VO$_2$max (ml (min)$^{-1}$), and relative VO$_2$max (ml (kg · min)$^{-1}$) and the change in relative VO$_2$max (dVO$_2$ml (kg · min)$^{-1}$) was significantly related to the change in insulin level. The relationships discovered were in a clinical setting and most likely would have been even stronger in a controlled research environment, which speaks to the practicality of the present findings and how it is likely that similar clinical intervention programs can have similar positive outcomes. Obesity and the resultant health consequences that have been long observed in adults are increasing rapidly in younger populations and discovering how to reduce these health problems is essential. The longer obesity persists in childhood, the more likely it is that the obesity will persist into adulthood and thus, interventions need to occur as soon as possible to limit health consequences (67). It is clear that by engaging in a lifestyle and exercise intervention program, obese children and adolescents can improve several health
related variables and in particular, African American females who are a higher risk for developing the metabolic syndrome can decrease insulin levels by increasing fitness levels and thus decrease the associated problems of the metabolic syndrome.
REFERENCES


## Table 1. Demographic Characteristics of the Participants

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<td>VO2 max difference (dvo2 ml (kg·min)^{-1})</td>
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<td>LDL cholesterol difference (Dldl)</td>
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<td>HDL cholesterol difference (Dhdl)</td>
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<td>Weight difference (Wtd)</td>
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Table 3. Stepwise Linear Regression Analyses for the Total Population Studied

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<td>Change in total cholesterol (dtc)</td>
<td>&lt;.001</td>
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<td>Weight difference (wtd)</td>
<td>Change in LDL cholesterol (dlldl)</td>
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<td>.0918</td>
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<td>Race</td>
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Table 5. Stepwise Linear Regression Analyses for the African American Female Subgroup

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<th>Dependent Variable</th>
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<tr>
<td>Change in Relative (VO_2\text{max} (dvo_2\text{ml (kg \cdot min)}^{-1}))</td>
<td>Log change in insulin (linsulind)</td>
<td>&lt;.01</td>
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<td>Weight difference (\text{wtd}) and Initial absolute (VO_2\text{max (ml (min)}^{-1}))</td>
<td>Change in total cholesterol (dtc)</td>
<td>&lt;.05</td>
<td>.3317</td>
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Table 6. Initial values of the Total Population Studied Compared to the African American Females

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<th>Pre-Test Insulin levels</th>
<th>Pre-Test (VO_2\text{max levels(ml (kg \cdot min)}^{-1}))</th>
<th>Pre-Test (VO_2\text{max levels (ml (min)}^{-1}))</th>
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<td>Total Population</td>
<td>28.84 (N=158)</td>
<td>25.35 (N=161)</td>
<td>2119.73 (N=161)</td>
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<tr>
<td>African American Females</td>
<td>30.89 (N=32)</td>
<td>22.41 (N=37)</td>
<td>2111.97 (N=37)</td>
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Figure 1. Graph of Pre-Test and Post-Test Values with Standard Deviations for Weight Change for the Total Population Studied

* Significant difference from Pre-Test to Post-Test, (p<.0001)
Figure 2. Graph of Pre-Test and Post-Test Values with Standard Deviations for VO\textsubscript{2}Max Change (ml(min\textsuperscript{-1})) for the Total Population Studied

*Significant difference from Pre-Test to Post-Test, (p<.0001)

Figure 3. Graph of Pre-Test and Post-Test Values with Standard Deviations for VO\textsubscript{2}max Change (ml(kg·min\textsuperscript{-1})) for the Total Population Studied

*Significant difference from Pre-Test to Post-Test, (p<.0001)
Figure 4. Graph of Pre-Test and Post-Test Values with Standard Deviations for Total Cholesterol Change for the Total Population Studied

*Significant difference from Pre-Test to Post-Test, (p<.0001)

Figure 5. Graph of Pre-Test and Post-Test Values with Standard Deviations for Triglyceride Level Change for the Total Population Studied

*Significant difference from Pre-Test to Post-Test, (p<.0001)
Figure 6. Graph of Pre-Test and Post-Test Values with Standard Deviations for LDL Cholesterol Change for the Total Population Studied

*Significant difference from Pre-Test to Post-Test, (p<.0001)
Figure 7. Graph of Pre-Test and Post-Test Values with Standard Deviations HDL Cholesterol for the Total Population Studied

*No significant difference from Pre-Test to Post-Test, (p>.05)

Figure 8. Graph of Pre-Test and Post-Test Values with Standard Deviations for Percent Body Fat Change for the Total Population Studied

*Significant difference from Pre-Test to Post-Test, (p<.0001)
Figure 9. Graph of Pre-Test and Post-Test Values with Standard Deviations for Body Mass Index Change for the Total Population Studied

*Significant difference from Pre-Test to Post-Test, (p<.0001)

Figure 10. Graph of Pre-Test and Post-Test Values with Standard Deviations for Percent Lean Mass Change for the Total Population Studied

*Significant difference from Pre-Test to Post-Test, (p<.0001)
Figure 11. Graph of Pre-Test and Post-Test Values with Standard Deviations for Insulin Level Change for the Total Population Studied

*Significant difference from Pre-Test to Post-Test, (p<.0001)

Figure 12. Graph of Changes in Relative VO₂max (dvo₂ml (kg · min)⁻¹) and log insulin (linsulind) in African American Females
Figure 13. Graph of Pre-test and Post-test Values for Weight Change for the African American Female Subgroup

*No significant difference from Pre-Test to Post-Test, (p>.05)

Figure 14. Graph of Pre-test and Post-test Values for VO\textsubscript{2}max Change (ml/(min)\textsuperscript{-1}) for the African American Female Subgroup

*Significant difference from Pre-Test to Post-Test, (p<.0001)
Figure 15. Graph of Pre-test and Post-test Values for VO$_2$max Change (ml(kg·min)$^{-1}$) for the African American Female Subgroup

*Significant difference from Pre-Test to Post-Test, (p<.0001)

Figure 16. Graph of Pre-test and Post-test Values for Total Cholesterol Change for the African American Female Subgroup

*No significant difference from Pre-Test to Post-Test, (p>.05)
Figure 17. Graph of Pre-test and Post-test Values for Triglyceride Level Change for the African American Female Subgroup

*No significant difference from Pre-Test to Post-Test, (p>.05)*

Figure 18. Graph of Pre-test and Post-test Values for LDL Cholesterol Change for the African American Female Subgroup

*No significant difference from Pre-Test to Post-Test, (p>.05)*
Figure 19. Graph of Pre-test and Post-test Values for HDL Cholesterol Change for the African American Female Subgroup

*No significant difference from Pre-Test to Post-Test, (p>.05)

Figure 20. Graph of Pre-test and Post-test Values for Percent Body Fat Change for the African American Female Subgroup

*Significant difference from Pre-Test to Post-Test, (p<.05)
Figure 21. Graph of Pre-test and Post-test Values for Body Mass Index Change for the African American Female Subgroup

*Significant difference from Pre-Test to Post-Test, (p<.05)

Figure 22. Graph of Pre-test and Post-test Values for Percent Lean Mass Change for the African American Female Subgroup

*Significant difference from Pre-Test to Post-Test, (p<.05)
Figure 23. Graph of Pre-test and Post-test Values for Insulin Level Change for the African American Female Subgroup

*No significant difference from Pre-Test to Post-Test, (p>.05)