CARDIORESPIRATORY RESPONSES IN HEALTHY-WEIGHT AND OBESE WOMEN AND CHILDREN

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CARDIORESPIRATORY RESPONSES IN HEALTHY-WEIGHT AND OBESE WOMEN AND CHILDREN

DISSERTATION

A dissertation submitted in partial fulfillment of the requirements for the degree of Doctor of Philosophy in the College of Education at the University of Kentucky

By
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Lexington, Kentucky

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Lexington, Kentucky

2013

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ABSTRACT OF DISSERTATION

CARDIORESPIRATORY RESPONSES IN HEALTHY-WEIGHT AND OBESE WOMEN AND CHILDREN

A criterion method to evaluate cardiorespiratory health is measuring peak oxygen consumption (VO$_2$ peak) from a maximal graded exercise test (GXT). While VO$_2$ peak is a valuable measure, heart rate recovery (HRRec) and endothelial function (EF) also describe cardiorespiratory health and fitness. The purpose of this study was to investigate whether differences exist in VO$_2$ peak, HRRec, and EF between healthy-weight (HW) and obese (OB) women and children and to determine if there were significant correlations among these variables. A total of 60 women and children participated in this study. Anthropometric, body composition, resting heart rate and blood pressure (BP) were measured. EF was evaluated to determine the reactive hyperemia index (RHI). Finally, each subject performed a graded exercise test (GXT) to determine VO$_2$ peak. Following the GXT, the subjects' recovery responses were monitored for 5 minutes. A factorial MANOVA was used to evaluate differences between obesity status and age in relative VO$_2$ peak and relative HRRec. The MANOVA resulted in a significant (p < 0.001) main effect for obesity status and age, but there was no interaction effect. HW individuals had a greater relative VO$_2$ peak compared to OB individuals. Children had a greater relative VO$_2$ peak and HRRec compared to adults. Absolute VO$_2$ peak and absolute HRRec were examined using univariate ANOVAs. Women had greater absolute VO$_2$ peak values compared to children (p<0.027) and children had greater absolute HRRec compared to women (p<0.001). Finally, an independent t-test was used to compare group means in RHI between HW and OB women. There were no differences in RHI between HW and OB women (p=0.128). For the total group, there were significant correlations between absolute VO$_2$ peak and absolute HRRec, relative HRRec, and RHI. Relative VO$_2$ peak was significantly correlated to RHI. Absolute HRRec was correlated with relative HRRec and RHI. Relative HRRec was correlated with RHI. Lack of significant differences in HRRec and EF across adiposity levels were likely due to the obese, but otherwise healthy population recruited for this study. Age affected the response to all variables included in this study.

Keywords: Obesity, children, cardiorespiratory fitness, heart rate recovery, endothelial function
CARDIORESPIRATORY RESPONSES IN HEALTHY-WEIGHT AND OBESE WOMEN AND CHILDREN

By

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________________________
July 18, 2013
Date
For my family

Rick, Gail, and Tim
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Chapter 1: Introduction

Evaluating cardiovascular health at rest and responses during and after exercise in adults and children is becoming increasingly important as the population becomes progressively more overweight and obese. This information may assist in early identification of individuals at risk of disease, provide guidance for more accurately designed exercise prescriptions, and help evaluate the effectiveness of treatment interventions. Currently, 33.8% of adults and 17% of children ages 2-19 years old are obese (35,82). Due to the high prevalence of childhood overweight and obesity, clinicians and investigators are faced with the challenge of understanding chronic pediatric pathologies including diseases such as hypertension, diabetes, and atherosclerosis, that were once limited to adult populations. These diseases are known to negatively impact cardiorespiratory health. Measuring peak oxygen consumption (VO$_2$ peak) is the most commonly non-invasive method used to assess cardiorespiratory fitness. Additional indicators of parasympathetic activity and peripheral arterial tone are heart rate recovery and endothelial function, which have been shown to be significantly related and associated with cardiorespiratory health and fitness (47).

There is a known relationship between heart rate recovery and all-cause mortality in adults; however fewer studies have examined the relationships between exercise capacity and heart rate recovery in adults and children (19,21,22). Cole and colleagues (21) found that there was a significant relationship between exercise capacity and heart rate recovery (HRRec) in adults. Lin et al. (63) examined HRRec in healthy children and adolescents 12-19 years old after a submaximal treadmill test. The authors reported that waist circumference and other metabolic risks, including systolic blood pressure, fasting glucose levels, triglyceride levels, and C-reactive protein levels were negatively related to HRRec in children.
Additionally, Hijmering et al. (45) reported a significant relationship between endothelial dysfunction and sympathetic activation of the nervous system utilizing flow-mediated vasodilation. Furthermore, there are limited studies using reactive hyperemia peripheral arterial tonometry (RH-PAT) in healthy children to estimate endothelial function (88,109). We are unaware of published reports using the RH-PAT to compare measures in healthy-weight versus obese prepubescent children. Brachial or radial ultrasonography are often used to determine measures such as arterial compliance and distensibility, because it is affordable and noninvasive (53). However these techniques can result in an undefined amount of error due to lack of standardization in procedures and significant variability among repeated tests (53). It has been suggested that the RH-PAT may be an advantageous method to noninvasively assess endothelial function because it provides both tester objectivity and reliability (39). Fei et al. (33) investigated the relationships among aortic pulse wave velocity, heart rate recovery, and VO₂ peak in healthy adults (mean age 48.4 ± 14.7 yrs). The authors report significant relationships between VO₂ peak and aortic pulse wave velocity (r = -0.51), and HRRec and aortic pulse wave velocity (r = -0.28) after logarithmic transformation. Fei et al. (33) suggested that HRRec should not be used as the sole indicator of cardiorespiratory health.

While there are many studies that examine variables including fitness, heart rate recovery, and endothelial function differences between healthy-weight and obese subjects separately, we are not aware of any studies that incorporate all three measures in both healthy-weight and obese individuals of two different maturational levels. The purpose of the present study was to compare differences in cardiorespiratory responses at rest, during a maximal graded exercise test
(GXT), and during recovery across adiposity and age levels. Therefore, the specific aims of the present study were to compare the group mean differences in VO$_2$ peak, HRRec, and endothelial function (via RH-PAT) measures in individuals with varying levels of adiposity (healthy-weight versus obese) and age (young children versus women), and to determine if significant correlations exist among these variables.
Chapter 2: Review of Literature

Introduction

Despite the reported adverse consequences of excess regional and total body fat, the incidence of obesity in the U.S. population continues to increase. Currently, the percentage of adults and children in the United States that are overweight and obese is reportedly 34% and 17%, respectively (35, 82). The 2012 Trust for America’s Health and Robert Wood Johnson Foundation (110) reported that Kentucky ranks 10th (30.4%) nationally in the incidence of obese adults and 3rd (21.0%) nationally in the incidence of obese children. Furthermore, the impact of obesity on some aspects of health and its relationship with total body fitness is still not fully understood in both adult and pediatric populations. Many researchers use peak oxygen consumption (VO$_2$ peak) measures as the criterion measure of cardiorespiratory fitness. However, there are other factors that should be considered when determining the cardiorespiratory health status of an individual. Heart rate recovery and endothelial function are other variables of interest to clinicians and researchers, when assessing cardiorespiratory fitness of an individual and understanding the development of cardiovascular disease.

Defining Healthy-Weight, Overweight, and Obesity

Definitions for healthy-weight, overweight, and obesity vary based on the criteria used to measure an individual. Most commonly, body mass index (BMI) is used to classify the weight status of an individual. Although sometimes used incorrectly as a measure of body composition, BMI is simply a ratio of body weight per unit of height and is calculated as the body weight in kilograms divided by the height in meters squared (kg/m$^2$). In adults, a BMI of 18.5 to 24.9 kg/m$^2$ is considered the healthy-weight range. However, a BMI of 25.0-29.9 kg/m$^2$ classifies an individual as overweight, and a BMI greater than 30.0 kg/m$^2$ is considered obese (17).
In children, classifications become slightly more complicated because a child’s height and weight are constantly changing due to growth and maturation. Therefore, when calculating a child’s BMI, researchers must also consider that child’s age and sex. The age and sex specific percentile cutoffs are 5th-84th percentiles for healthy-weight, the 85th-94th percentiles are considered overweight, and greater than or equal to the 95th percentile classifies a child as obese (18). Additionally, there have been changes in how those percentiles have been interpreted. Previous to 1994, the BMI categories for children were listed as healthy-weight, overweight, and obese. In 1994, children whose percentile between the 85th-94th percentile were considered “at risk for overweight,” but in 2007, the terminology was changed back to “overweight.” The children in the highest 5 percent considered “overweight” regarding the terminology from 1994, but now the top 5 percent are considered “obese” again. (83). Ogden et al. (83) briefly discuss the purpose of the change in terminology and cite the Institute of Medicine as stating that changing the labeling of the categories conveys the seriousness and urgency surrounding this issue.

While BMI allows researchers to classify individuals based on their weight, body fat tends to be more impactful on an individual’s health. Appropriate levels of body fat vary by age and sex. Males ages 20-39 years of age are considered to have an elevated health risk at a body fat percentage of 20-24%, whereas total body composition of 22-27% fat and 25-29% fat are considered high risk in 40-59 year olds and 60-70 year olds, respectively. In females, elevated health risk is associated with total body composition between 33-38% fat, 34-39% fat and 36-41% fat, in 20-39 year olds, 40-59 year olds, and 60-79 year olds, respectively (4).

In children, the values are less clearly delineated; however unhealthy percentages for males and females are relatively similar to adult percentages. Williams et al. (119) found associations of higher disease risks in boys with body fat percentages
greater than 25% and in girls with body fat percentages greater than 30% (in children ages 5-18 years). For the purposes of this literature review, both BMI and body fat will be discussed methods to define obesity status. Additionally, the interpretations of BMI percentiles in children as healthy-weight, overweight and obese, will be used describe children of different body sizes.

**Adverse Consequences of Obesity**

**Heart Rate**

The nervous system provides input for controlling heart rate. The nervous system is divided into the central and peripheral nervous systems. The central nervous system (CNS) is composed of the brain and spinal cord, and the peripheral nervous system (PNS) is includes the nerves outside of the brain and spinal cord. The peripheral nervous system is made up of two major divisions: the autonomic nervous system and the somatic nervous system. The autonomic nervous system is not under voluntary control and affects unconscious responses such as heart rate, respiration, and smooth muscle contraction. The autonomic system is further divided into two branches: the parasympathetic system and the sympathetic system. At rest, the parasympathetic nervous system is controlling heart rate. This system is responsible for the "rest and digest" events in the body. This control is often referred to as parasympathetic tone or vagal tone. The sympathetic division of the autonomic nervous system is responsible for the "fight or flight" responses in the body. (62,87).

**Impact of obesity on resting heart rate.** Normal resting heart rate for adults is typically between 60-80 beats per minute (bpm). A heart rate that is lower than 60 bpm is classified as bradycardia, whereas heart rate greater than 100 is called tachycardia (44). In healthy children, most resting values, including resting heart rate tend to be
greater compared to adults (31). Rowland (94) reported that greater resting heart rates were likely related to higher basal metabolic rate (BMR) in young children. Rowland (94) stated that there is must be an association between the rate that the sinus node depolarizes in children and BMR; however this relationship is not understood. Sorof et al. (106) found higher resting heart rates in obese children compared to non-obese children when age, sex, race and hypertension status were controlled (mean heart rate 84.2 bpm in the obese versus mean heart rate of 80.7 bpm in the non-obese). Their analysis found three levels of resting heart rates. The obese hypertensive children had the highest resting heart rates, normotensive obese children and hypertensive non-obese children had intermediate resting heart rates, and the normotensive non-obese children had the lowest resting heart rates. These authors speculated that obesity and hypertension had collaborative and independent effects on resting heart rate (p. 663) (106). In a study examining the physiological profiles of obese and non-obese sixth grade students (11.55 ± 0.39 yr and 11.49 ± 0.40 yr, respectively), the obese group was found to have significantly higher resting heart rates (84.67 ± 12.58 bpm versus 79.69 ± 11.32 bpm) (30). While the authors did not specifically discuss why resting heart rates were greater in the obese children, they reported higher systolic and diastolic blood pressure in the obese children (30). Perhaps, similar to Sorof et al. (106), the higher heart rates in the obese students were mediated by higher blood pressures in addition to their obesity. Similarly, in a study comparing exercise responses of obese adults to healthy-weight adults, the obese group had a higher mean heart rate during rest and unloaded free pedaling on a cycle ergometer compared to a non-obese group (93 ± 3 bpm versus 76 ± 2 bpm; 106 ± 3 bpm versus 86 ± 3 bpm, respectively) (98). The authors of this study report lower stroke volume in the obese adults, which would require a higher heart rate to maintain cardiac output (98).
**Exercise heart rate.** Upon the initiation of exercise, parasympathetic or vagal tone is slowly withdrawn to allow the heart rate to increase to meet the increased metabolic demands that are being placed on the body (114). Balady et al. (6) stated that heart rate increases in a linear fashion as work rate increases, however the degree to which heart rate increases is affected by several factors. These factors include “age, deconditioning, body position, type of exercise, and various states of health and therapy, including heart transplant” (p. 199) (6). Washington et al. (114) reported that additional influences of heart rate include sex and environmental conditions. As the work rate increases, the sympathetic activation contributes significantly to the increase and contractile force of the heart. This component of the autonomic system is responsibility for the “fight or flight” responses in the body. This sympathetic drive increases the rate and the strength of contraction of each beat (87).

Maximal heart rate is most often predicted by using the general equation, 220 minus the subject’s age (44). This equation is merely an estimate based on a regression equation, and assumes that everyone in a particular age group has a similar maximal heart rate. The variability of this estimated maximal heart rate is often reported to be approximately ± 12 bpm (6). Due to this variability, achievement of 85% of the maximal predicted heart rate has been used to determine a maximal effort during exercise testing (6). Additionally, failure of heart rate to increase despite an increase in workload is considered to be maximal effort (44). A relatively new population-specific equation for women has been reported for estimating peak heart rate. Gulati and colleagues (37) tested 5,437 women using a graded exercise test and found that there was a relationship between age and peak heart rate. The authors found that the equation, peak heart rate = 206 – 0.88(age) was more appropriate for predicting maximum heart rate in women (37). The rationale for developing this equation was that the currently
used equation (Peak HR = 220-age) is based on data for a predominantly male cohort. This equation tends to overestimate maximal heart rate for women. The authors found that when the male-referenced equation was used, many women were being labeled as “chronotropically incompetent.” All-cause mortality was used as an indicator of survival for 14 years after the study. Both equations were used with survivals to determine whether the two equations agreed or differed. The authors found that the new women-specific equation was a better predictor of mortality and identified less chronotropic incompetent women (37).

When adults and children are compared, children typically have a greater heart rate for a given workload, and achieve higher maximal rate rates. This is due to the smaller size of the heart in children and thus lower stroke volume (114). As children mature, heart rate decreases for a given submaximal load; this is a function of increased heart size and stroke volume (94). In addition, obesity status reportedly impacts submaximal and maximal heart rates. In both obese adults and children, submaximal heart rates tend to be higher than their normal weight counterparts (98,114). Salvadori et al. (98) suggested the higher heart rates in obese individuals during submaximal workloads are the result of lower stroke volume. Despite higher submaximal heart rates for a given workload, obese individuals tend to have a lower peak or maximal heart rate compared to healthy-weight individuals. Loftin et al. (68) reported significantly lower peak heart rates (192.3 ± 9.3 bpm) in an obese cohort of girls (n=43, 12.4 ± 2.7 years old) than the peak heart rate (203.4 ± 7.6 bpm) of normal weight girls (n=45, 12.6 ± 2.9 years old) during treadmill walking or running. The authors speculated that the type of treadmill exercise test (walking versus running) may be related to lower heart rates in the obese subjects. All obese subjects chose the walking exercise test protocol whereas the majority of the non-obese subjects chose the running protocol (68). Similarly, Salvadori
et al. (98) found lower peak heart rates in obese (156 ± 4 bpm) compared to non-obese (171 ± 3 bpm) men and women (ages 17-42 years) during cycling exercise. The authors attributed this higher peak HR in non-obese adults to statistically higher epinephrine levels when compared to the obese individuals (98).

**Heart rate recovery.** Following maximal exercise, the heart rate recovery has been reported to provide valuable information about an individual’s health and cardiorespiratory fitness. Heart rate recovery (HRRec) is defined as the one minute post exercise heart rate subtracted from the peak heart rate during exercise. Cole et al. (21) defined an abnormal one minute heart rate recovery as less than or equal to 12 beats per minute, while Watanabe et al. (113) found it to be less than or equal to 18 beats per minute. Both studies found abnormal HR recovery responses to be strongly related to increased mortality risk (21,113). Additionally, Cole et al. (21) reported that reduced exercise capacity was an independent factor in predicting abnormal heart rate recovery. Shelter et al. (101) reported that the rate at which the heart recovers back to the resting rate is related to high parasympathetic tone and health (p. 1980). Interestingly, Cheng et al. (19) found that while heart rate recovery was a strong predictor of death, cardiovascular fitness measured by time on treadmill during a maximal exercise test (modified Balke protocol) was a stronger predictor of cardiovascular disease deaths and all-cause mortality in men with diabetes. However, the exercise test used to measure fitness did not provide an actual value for oxygen consumption, only an estimate based on the duration of the test. The authors suggest that when predicting mortality risk in this population, factors such as cardiorespiratory fitness level should be used in addition to heart rate recovery (19). Baraldi et al. (7) examined differences in heart rate recovery after one minute of intense exercise in healthy weight adults and children. After exercising at different percentages of their VO₂ max, the time for heart rate recovery was
recorded as heart decreased back to baseline. Ten minutes of recovery were allotted to assure that heart rate would have time to return to the resting rate after each one minute bout of exercise. After one minute of exercise at 100% of VO$_2$ max and ten minutes of recovery, heart rate remained elevated above baseline for adults and children. In adults, heart rate was still 4.6% greater than the recorded baseline and in children it was 2.8% higher than baseline. After one minute of supramaximal exercise (125% VO$_2$ max), heart rate in adults and children remained elevated 7% and 2.6% greater than the baseline measures, respectively after ten minutes of recovery. The investigators found these recovery times to be significantly faster in children (7). This demonstrates that the higher intensity will result in a longer recovery time and that children can recover more quickly from intense exercise when compared to adults. The authors speculated that difference in the response between adults and children was related to the lower levels of circulating hydrogen ions, lactate, and catecholamines (p. 578) (7).

There are several possible explanations for this decreased recovery time in children compared to an adult. Falk et al. (31) reported three primary explanations for the recovery time discrepancies including size differences, maturation status, and metabolic-related factors. The investigators postulated that a smaller body size results in smaller muscle fibers and short circulation distances and times, which may result in a more rapid recovery (31). It was further proposed that maturational stage differences would be responsible for lower muscle mass and less nerve recruitment resulting in lower total power output relative to the child’s size. Finally, following heavy exercise, metabolic factors including a faster recovery of the acid base balance due to lower lactic acid production, and faster energy replacement may help to explain the differences observed in the response of children compared to adults (31). Rowland et al. (93) examined the impact of eliminating the role of skeletal muscle pump in passive recovery
from cycling exercise boys (12.0 ± 1.3 yr) and men (27.0 ± 3.7 yr) in two separate studies. The boys performed a maximal cycle exercise test and upon completion immediately dropped their legs down in a passive recovery position. Cardiac output was examined in the first 15 seconds. The mean decrease in cardiac output in the subjects was 15.8 ± 12.4%. The men cycled 60% VO$_2$ max (2 bouts of 4 minutes). After one bout, the men participated in active recovery, continuing to cycle at a lower intensity and after the other bout the men dropped their legs to the side in a passive recovery position. Recovery was examined in the first 20, 40, and 60 seconds following exercise. In the first 20 seconds of passive recovery, the men had a mean decrease in cardiac output of 10.9%. Rowland et al. (93) stated that these studies demonstrate that differences in heart rate recovery may be affected by maturational changes in venous return and potentially peripheral vasodilation between children and adults since there was little change in cardiac output following exercise during passive recovery. Furthermore, Ohuchi et al. (81) examined heart rate recovery and heart rate variability (as an index of cardiac autonomic activity) in children and young adults. The authors suggested that the faster heart rate recovery in children was related to greater parasympathetic activity compared to the young adults.

Singh et al. (103) tested fitness levels in healthy-weight, overweight, and obese boys and girls (5.2-18.8 years) to describe heart rate recovery in children after maximal exercise. Heart rate recovery was attenuated with increasing body mass in this population. Overweight and obese children had much slower heart rate recovery compared to their healthy-weight counterparts (103). The authors attributed the slower heart rate recovery in the obese children to reduced parasympathetic control and healthy lifestyle behaviors in the healthy-weight children (103). Furthermore, in a study comparing obese and non-obese middle school children to determine consequences of
obesity, Eagle et al. (30) found statistically greater heart rates after a one minute recovery from exercise in the obese group. In this study, heart rate recovery was used as an indicator of fitness level after a three minute step test. The slower recovery of the obese children indicated lower fitness levels when compared to their non-obese counterparts (30). These trends persist in the adult population as well. In a study examining 5 minute recovery time from maximal exercise in diabetic men, Cheng et al. (19) found that the quartile of men with the slowest heart rate recovery had the highest mean BMI. Similarly, Cole et al. (22) found that in participants with an abnormal two-minute heart rate recovery (≤ 42 beats per minute) the mean BMI was statistically greater (26.4 ± 4.1 kg/m² versus 25.6 ± 3.8 kg/m²) than the group with a normal heart rate recovery response. Additionally, once confounding variables were controlled for (including BMI), abnormal heart rate recovery was still a strong predictor of mortality. The authors did not directly comment on the statistical difference in BMI across normal and abnormal heart rate responses, but did report that the abnormal responders were less likely to exercise regularly based on behavioral data that was collected (22). Singh et al. (103) found attenuated heart rate recovery with increasing age. The authors did not provide an explanation for the attenuation of heart rates with increasing age; however the same inferences regarding lower levels of circulating catecholamines in younger children could be applied here. The smaller size of the younger children may also be related to the differences with increasing ages (31). Similarly, Buchheit et al. (15) reported faster heart rate recovery in children compared to adolescents and adults after bouts of cycling sprints. The authors stated that the findings may be related to the adolescents’ ability to produce more power and have greater anaerobic capacities compared to children due to their size and maturation status (15).
Hemodynamics

Hemodynamics are the forces associated with blood circulation (87). Changes in the blood vessel diameter, pressure, and flow occur during the rest to exercise transition. According to Christensen et al. (20), sympathetic activity results in blood flow redistribution to the working muscles from areas such as the splanchnic region, kidneys, and resting muscles. This increases the proportion of blood that the muscles are receiving, in addition to a greater absolute amount of blood due to increased cardiac output (87).

Cardiac output. Cardiac output (Q) is the total amount of blood that is circulating throughout the body measured in liters per minute (L/min). Q is the product of HR and stroke volume (SV). SV is the volume of blood ejected with every contraction of the heart, measured in milliliters (ml). At rest, Q is typically approximately 4.5 L/min to 5.5 L/min in adults. At maximal exertion, increases in HR and SV, can increase Q up to 20-30 L/min (58).

In a study examining the affects of different modes of exercise on hemodynamics, when the subjects performed a protocol involving one arm cranking showed no increase in stroke volume from rest, but during two leg cycling demonstrated an increase in both heart rate and stroke volume (11). It is important to note, that increases in SV only occur up to approximately 50% maximal oxygen consumption (VO₂ max, or the highest amount of oxygen that a person can consume and utilize during maximize exercise), exercising past that intensity is possible through increases in HR (58). However, in endurance trained individuals, the stroke volume may increase greater than 50% VO₂ max, resulting in a lower heart rate for any given submaximal workload in athletes (87). In obese adult populations, resting Q tends to be greater than
normal weight individuals. This increased Q is attributed to a higher SV, rather than increased HR (27). However, Salvadori et al. (98) reported a lower SV in obese compared to healthy-weight. The increased SV is related to increased blood volume and increased preload (the amount of blood in the ventricles at the end of the diastolic filling period) in obese individuals (52). However, additional studies have demonstrated higher resting HRs associated with obesity (30,106) which would result in an overall increase in Q when combined with the increased SV (102).

Resting Q in children is lower than adults as a result of smaller body size and their smaller hearts (114). Schuster et al. (99) reported a mean Q of 3.761 L/min in lean prepubertal boys. Katori et al. (49) evaluated Q in 151 subjects (4-78 yrs). The authors report an increase in Q into adolescence, with a progressive decline after age 15. This decline in relative Q associated with increasing size is in accordance with decreases in basal metabolic rate (BMR) (49,94). The reduction in the resting Q of adolescence is mediated through decreases in resting heart rate with growth (94). In boys with severe obesity, the mean resting Q was significantly higher at 5.358 L/min (99). In response to exercise, children have smaller increases in stroke volume due to their heart size (112, 114). This results in increases in Q during exercise which is primarily mediated by increasing heart rate in pediatric populations (114). According to Rowland (94), the rate at which cardiac output increases during exercise is similar to adults. At maximal intensities, prepubertal boys have shown increases in Q up to 12.8 L/min (112).

**Blood pressure.** Blood pressure is the force that is exerted on the walls of the arteries during the contraction (systole) and relaxation (diastole) phases of the cardiac cycle, and is product of Q and total peripheral resistance (TPR). Systolic blood pressure represents the force created during ventricular contraction of the heart. Diastolic blood pressure is the force remaining in the arterial system during the relaxation and
ventricular filling phase of the cardiac cycle. In adults, optimal resting blood pressure is reported to be a systolic value less than 120 millimeters of mercury (mm Hg) and a diastolic value less than 80 mm Hg (4). Following the initiation of exercise, systolic blood pressure should increase to meet the increased metabolic demands. Diastolic blood pressure values often remain constant or slightly decrease due to the reduction in total peripheral resistance (87). Schuster et al. (99) reported mean resting blood pressure values in healthy-weight boys (ages 10-12 years) of 102.6 mm Hg and 62.5 mm Hg for systolic and diastolic blood pressures, respectively. In severely obese boys, the mean value for systolic blood pressure was significantly higher (122.0 mm Hg) than the healthy-weight boys, whereas the diastolic values was not significantly different (70.7 mm Hg) (99). Similarly, Eagle et al. (30) reported resting blood pressure measures of healthy-weight middle school boys and girls (mean age 11.55 ± 0.39) of 108.02 ± 10.92 mm Hg (systolic) and 64.37 ± 13.16 mm Hg (diastolic). This was significantly lower than their obese counterparts (mean age 11.49 ± 0.40) who had mean values of 118.48 ± 10.80 mm Hg and 70.42 ± 7.99 mm Hg, for systolic and diastolic blood pressures, respectively. Williams et al. (119) reported that children (5-18 yrs) with excess body fat (>25% in males and >30% in females) were at a significantly greater risk for higher blood pressure values. Developing normative ranges for children and adolescents is difficult because blood pressure tends to be influenced by height and gender. According to the 4th report on the Diagnosis, Evaluation and Treatment of High Blood Pressure in Children and Adolescents (111), pediatric hypertension is defined as a systolic and/or diastolic blood pressure that is greater than the 95th percentile for age, height, and gender on three or more occasions. Prehypertension is defined as having a systolic or diastolic blood pressure greater than the 90th percentile (but less than the 95th percentile) or having a blood pressure greater than or equal to 120/80 mm Hg.
According to Washington et al., (114) systolic blood pressure increases greater than 220 mm Hg in adults during exercise is considered excessive, however a systolic pressure approaching 250 mm Hg is not regarded as dangerous in a child that has no history of cardiac issues. Washington et al. (114) reported that there is no known standard exercise response of diastolic blood pressure in children due to difficulty of the measurement; however similar to adults it would be considered abnormal to see a significant increase in diastolic blood pressure with increased intensity. Resting systolic blood pressure has been found to strongly related to maximal systolic values (8). Bassett et al. (8) examined 68 men with normal resting blood pressure and performed maximal cycle ergometer exercise tests. The subjects were categorized into 2 groups based on their maximal systolic blood pressure (< 220 mm Hg or > 220 mm Hg). The group of subjects with the higher maximal systolic blood pressure had higher resting values when compared to the lower maximal blood pressure group. While the authors did not speculate on the cause of these findings, they suggested that exercise blood pressures may be helpful in predictive future hypertension in normotensive adults (8). Salvadori et al. (98) compared cardiovascular responses in non-obese (mean BMI 22 kg/m²) and obese (mean BMI 40 kg/m²) adults during a cycling protocol to volitional exhaustion. The authors reported no differences in resting blood pressures (mean values 116/77 mm Hg in non-obese and 120/84 mm Hg in obese) or at the end of the exercise bout (mean values 153/90 mm Hg in non-obese and 155/95 in obese) (98). A study examining cardiovascular risk factors and vascular changes in children compared resting and exercise systolic blood pressures values in an obese exercise intervention group, an obese control group, and a lean control group (78). The resting blood pressures were significantly greater in the obese groups compared to the lean group (128 ± 15.7 mm Hg, 133 ± 14.1, and 113 ± 10.7 mm Hg). These groups (obese intervention, obese control, and lean control) were also compared during cycling
exercise at 2 W/kg of a modified Bruce cycling protocol. The obese groups had significantly higher systolic blood pressure values when compared to the lean group during exercise (161.6 ± 26.1 mm Hg, 166.9 ± 40.1 mm Hg, and 143 ± 12.7 mm Hg) (78).

**Oxygen Consumption (VO₂)**

**Resting oxygen consumption.** Indirect calorimetry uses measures of oxygen consumption as an estimate of energy expenditure. VO₂ is a weight-dependent variable where a larger individual will consume more oxygen than a smaller individual. VO₂ is typically measured in liters per minute (L/min), however is commonly expressed in milliliters per kilogram per minute (ml/kg/min) to reduce the impact of body weight. Springer et al. (104) found that resting VO₂ was higher in children (10.4 ml/kg/min) versus adults (6.4 ml/kg/min). Similarly, when children (ages 7-10) were compared to adolescents (ages 15-18), the younger group was found to have significantly higher (9.4 ml/kg/min versus 5.8 ml/kg/min) resting VO₂ (23). Neither of these studies discussed the fact that the resting VO₂ values for adults are significantly greater than the documented value of 3.5 ml/kg/min for resting VO₂ in adults. Both of these studies examined oxygen kinetics which requires a transition to exercise, therefore perhaps the higher VO₂ values were related to an anticipatory increase that sometimes occurs with exercise. Harrell et al (40) examined energy costs of certain physical activities of boys and girls (8-18 years old). Part of the rationale for performing VO₂ measures during activities in children is that the compendium of physical activities is based off of an adult cohort. The authors report mean and standard deviations of VO₂ in different developmental stages: 6.08 ± 1.38 ml/kg/min (Tanner Stage I); 6.01 ± 1.35 ml/kg/min (Tanner Stage II); 5.16 ± 1.39 ml/kg/min (Tanner Stage III); 4.25 ± 0.96 ml/kg/min (Tanner Stage IV); and 3.80 ± 0.74 ml/kg/min (Tanner Stage V). These values were compared to the published adult resting
VO₂ of 3.5 ml/kg/min. While neither Springer et al. (104) nor Cooper et al. (23) reported reasons for the difference in resting VO₂, Harrell et al. (40) stated that the difference is related to several factors. These authors reported decreases in resting VO₂ with increases in age and development (Tanner Stages). The authors stated the potential reasons for the differences are the increased energy expenditure as a result of growth and puberty due to increasing total mass, differences in body mass (specifically smaller muscle mass) resulting in a higher relative value for resting expenditure, and the increased proportion of metabolic active organs in children (40, 46). Hsu et al. (46) examined the differences in highly metabolic organ tissue masses (brain, liver, heart, and kidneys) between children (9.3 ± 1.7 yr) and young adults (26.0 ± 1.8 yr). As expected, the adults had greater masses for liver, heart, kidney, skeletal muscle, and total adipose tissue, however there were no differences regarding the brain mass between groups. In children, the brain and liver contributed a greater proportion to the fat-free tissue when compared to the adults. The authors speculated that the decrease in resting metabolic expenditure with age is associated with the decrease in the proportion of these metabolically active tissues. Furthermore, the authors proposed that there is an increased metabolic rate per unit mass of organs and tissues in children. Finally, Hsu et al. (46) suggested that measured resting VO₂ values may be higher than actual values due to the difficulty children have with complying with the instructions to remain in a still resting position.

**Oxygen kinetics.** When an individual transitions from a resting state into dynamic physical activity, the changes in VO₂ can be described by oxygen kinetics. The changes in the VO₂ at rest and during exercise have been explained in three phases. Whipp et al. (118) examined the rest-to-exercise transitions on an electronically braked cycle ergometer to more clearly understand the changes in oxygen kinetics. When VO₂
changes were examined, there was an “abrupt increase” in the VO\(_2\) consumed, the VCO\(_2\) produced, and ventilation was observed (23). This response was named Phase 1 and is often referred to as “cardiodynamic phase” (118). Measurements of Phase 1 vary in the literature. Cooper et al. (23) measured VO\(_2\) consumption twenty seconds after exercise commenced and expressed it as a percentage of the steady state VO\(_2\) consumption. Fawkner et al. (32) defined the end of Phase 1 to be fifteen seconds after the onset of exercise for all subjects.

Phase 2 has been defined as the exponential rise in VO\(_2\) consumption that occurs as a result anaerobic energy metabolism and ends with the achievement of Phase 3 (steady state) (118). Phase 2 is often described by a time constant reported in the literature. The time constant describes the change in VO\(_2\) consumption above baseline at 63\% of the final steady state value (23). This Phase 2 measurement is usually described by the following equation: \(\Delta VO_{2(20)} = \Delta VO_{2(ss)} \times (1 - e^{-t/T})\). The equation yields the change in VO\(_2\) consumption above the baseline at a particular time (t) based on the product of the change in VO\(_2\) consumption at steady-state (\(\Delta VO_{2(ss)}\)) and the time constant. Phase 3 consists of a steady state in O\(_2\) consumption (23).

Oxygen kinetics similarities and differences between children and adults have not been fully elucidated. Cooper et al. (23) found no significant correlations between Phase 1 and age or weight in younger (7-10 yr) and older (15-18 yr) children. Phase 1 was affected by work rate, with higher intensities resulting in higher VO\(_2\) consumption values for this phase. There were no correlations found between the time constant of VO\(_2\) consumption in Phase 2 with age, height, and weight (23). Similarly, Hebestreit et al. (42) found no significant differences when the oxygen kinetics of men versus boys were compared. However, Fawkner et al. (32) found greater work rates, VO\(_2\) consumption at steady state, and oxygen deficits in adults when compared to children. Men and women
had longer mean time constants and mean response times to reaching VO\textsubscript{2} steady state when compared to boys and girls, respectively. The authors concluded that children have more rapid responses to exercise transitions than adults (32).

Loftin et al. (69) compared VO\textsubscript{2} kinetics during constant load cycling in severely overweight girls and their healthy-weight counterparts. While the authors found no differences in phase I or phase II kinetics, the O\textsubscript{2} deficit was significantly greater in the severely overweight group. The authors attributed this to a higher contribution from anaerobic metabolic systems (69). Salvadego et al. (97) compared VO\textsubscript{2} kinetics between obese boys (16.5 ± 1.0 yr) and their healthy-weight counterparts (16.6 ± 1.1 yr) during constant load cycling exercise. The authors specifically examined the fundamental component and the slow component. The fundamental component describes the oxygen deficit, or the time from the start of exercise utilizes anaerobic energy until steady state is reached. The slow component represents the increase in VO\textsubscript{2} consumption that exceeds steady state with increasing duration or intensity. The boys exercised at 40%, 60%, and 80% of their predicted VO\textsubscript{2} max for 10 minutes and steady state VO\textsubscript{2} consumption values were compared. There were no differences in steady state VO\textsubscript{2} consumption values in absolute terms (L/min) or when scaled to fat-free mass (ml/kgFFM/min). The obese group had significantly lower steady state VO\textsubscript{2} consumption values at all intensity levels when presented in relative terms utilizing total body weight (ml/kg/min). At all intensity levels (40, 60, and 80% VO\textsubscript{2} max), the obese group had a slower fundamental component, indicating a greater oxygen deficit. Greater oxygen deficit is related to lower exercise tolerance and greater homeostatic disruption. At 60% of VO\textsubscript{2} max, a slow component was present in 9 out of 14 subjects of the obese group and was not present in any healthy-weight subjects. At 80% intensity, the slow component was present in all subjects, with the exception of 2 obese subjects who could
not maintain the specified intensity. However, there was no difference in the amplitude of the slow component between groups at 80% of VO\textsubscript{2} max. The earlier appearance of the slow component in the obese children may be related to lower exercise tolerance and earlier fatigue. Furthermore, there was a greater slope associated with the slow component demonstrated by the obese children compared to their healthy-weight counterparts that may result in earlier termination of exercise. The authors suggested that these findings indicate a reduced capacity of the skeletal muscle to perform aerobically (97).

**Maximal oxygen consumption.** Maximal oxygen consumption (VO\textsubscript{2} max) is used as an indirect measure of cardiorespiratory fitness status and is defined as plateau in oxygen consumption (or < 150 ml/min) despite an increase in workload. Many individuals do not reach VO\textsubscript{2} max, and there is an increased incidence of underachievement in pediatric populations (2,91). In adults, VO\textsubscript{2} peak is defined as the highest oxygen consumption value reached during maximal exercise. Supporting criteria to demonstrate that VO\textsubscript{2} peak has been achieved include subjective ratings of perceived exertion (17 on the Borg scale of perceived exercise), failure of heart rate to increase with increase in workload, venous lactate concentration greater than 8 millimols, or a respiratory exchange ratio (RER) greater than or equal to 1.15 (44). In children, VO\textsubscript{2} peak is considered the highest VO\textsubscript{2} during maximal exercise with a heart rate greater than 190 beats per minute and/or a RER greater than or equal to 0.99 (67).

When presented in absolute terms, adults typically have higher VO\textsubscript{2} max or peak values when compared to children, simply due to their larger amount of fat-free mass (the oxygen consuming tissue). This has been demonstrated in studies that show increasing absolute VO\textsubscript{2} max or peak values with maturation (3). Similarly, when comparing an obese group to a lean group, the obese group will have equivalent or
higher absolute values. Loftin et al. (67) found similar absolute values for VO₂ peak (L/min) when comparing obese girls (mean age 12.6 ± 3.0 yr; 1.77 L/min) to their non-obese counterparts (mean age 12.4 ± 2.7 yr; 1.96 L/min). Goran et al. (36) found that obese children (mean age 8.9 ± 1.2 yr) had a greater absolute VO₂ max (1.56 ± .40 L/min) compared to a lean group of children (mean age 8.6 ± 1.6 yr; VO₂ max 1.24 ± 0.27 L/min). The authors attributed the higher absolute VO₂ max in obese subjects to a greater amount of fat-free mass (36).

When VO₂ max is presented in terms relative to body mass, adults and children often have similar results. Welsman et al. (115) reported VO₂ peak values for three different maturation groups (prepubertal, circumpubertal, and adult) for males and females. In the male groups, there were no significant differences between VO₂ peak for the prepubertal (50 ± 4 ml/kg/min), circumpubertal (53 ± 4 ml/kg/min) and adults (53 ± 3 ml/kg/min). In the female groups, the circumpubertal group had a significantly higher VO₂ peak than the adults (47 ± 4 ml/kg/min versus 43 ± 3 ml/kg/min), however there were no differences between the prepubertal (45 ± 3 ml/kg/min) and the remaining groups. When expressed in relative terms, a much lower VO₂ max for obese groups was has been reported, demonstrating a lower performance value for this group due to the cost of moving the metabolically inactive fat mass. Loftin et al. (67) compared relative values for VO₂ peaks in prepubescent girls and found that obese girls (22.8 ± 7.3 ml/kg.min) had a significantly lower values compared to their healthy-weight counterparts (45.8 ± 7.2 ml/kg/min). This difference was attributed to the impact of excess body fat and Loftin et al. (67) concluded that VO₂ peak scaled to total body mass may demonstrate of the impact of excess adipose tissue rather than functional status. Similar results have been reported in adult populations. In an adult male population (~50 years old), a group of normal weight men had a significantly higher mean VO₂ max
(38.6 ± 1.2 ml/kg/min) compared to an obese group of men (27.9 ± 1.7 ml/kg/min) (112).

Welsman et al. (115) reported that the mean VO₂ max values for adult males (53.3 ml/kg/min) were significantly greater compared to adult females (43.3 ml/kg/min).

Welsman et al (115) reported that a greater VO₂ max may be due to greater increases in fat-free mass in males during puberty and a higher hemoglobin concentration (and thus greater oxygen carrying capacity) compared to females.

**Endothelial Function**

The endothelium is the thin lining of the lumen of the vascular system and dysfunction of this organ is associated with hypertension, atherosclerosis, and heart failure (24). Proper functioning of the endothelial lining includes “regulating vascular tone, growth, inflammatory response, coagulation, and thrombocyte adhesion” (57). The endothelium releases several factors, most notably, nitric oxide to regulate tone (29,86). Poredos (86) reported that many diseases produce oxidative stress in the body which results in the degradation of nitric oxide. This reduced level of nitric oxide can lead to the development of atherosclerosis. While the exact mechanisms are unknown, endothelial dysfunction does seem to play a role in the atherogenic process (86). Poredoes (86) lists several hypotheses related to the possible promotion of atherogenesis including: increased adherence of cells, increased permeability to macrophages and cholesterol which leads to accumulation, increased platelet adherence, and smooth muscle cell migration/proliferations (p. 276).

Atherosclerosis is the stiffening of the arteries in the body due to fatty deposits in the arterial walls that develop into plaques. Berenson and colleagues (9) performed autopsies on 204 individuals (aged 2-39 years) whose deaths were primarily the result of trauma. Ninety-three of these individuals had been a part of the Bogalusa Heart Study.
and the researchers had information regarding their cardiovascular risk factors prior to their deaths. The authors found an increased prevalence of fibrous plaques on both the aorta and the coronary arteries as age increased. Additionally, the lesions were positively associated with increasing body mass index, systolic and diastolic blood pressure, total cholesterol levels, and triglyceride levels (9). These lesions result in stiffening of the arteries which in turn causes decreased compliance and distensibility of the arteries.

**Methods of testing endothelial function.** Endothelial function is often measured by radial of brachial ultrasonography using flow-mediated dilation (FMD). Another newer method to measure endothelial functioning in the periphery is reactive hyperemia peripheral arterial tonometry.

**Flow-mediated dilation and ultrasonography.** Blood vessels have the ability to dilate and constrict, and they do this in response to physiological triggers present in the cardiovascular system. These triggers consist of substances released from the endothelial layer such as prostacyclin and nitric oxide. Nitric oxide can be released as a result other substances (adenine triphosphate, bradykinin, serotonin, substance P, and histamine) or as the result of shear stress (62). An increase in flow will result in dilation of the blood vessels to accommodate the additional blood flow and the stimulator for this dilation is nitric oxide in endothelium-dependent FMD (24). This technique is non-invasive and used in conjunction with radial or brachial ultrasonography.

To prepare subjects for this method of testing, the subjects need to be in a quiet, resting position in a thermo-neutral environment. The equipment includes a high-frequency vascular transducer, internal ECG, and vascular software for 2D imaging. The subject should be placed in a supine position so that the brachial artery can be easily imaged above the antecubital fossa. Additionally, veins and fascial planes are
used to maintain the same position while imaging. First, a baseline measure of the brachial artery should be acquired and then the FMD is caused by occluding blood flow to the lower arm by inflating a blood pressure cuff to a suprasystolic level (minimally 50 mm Hg above systolic blood pressure) for a given time period (typically 5 minutes). After the pressure in the cuff is released, the artery dilates due to the shear stress and an image of the brachial artery is recorded for 2 minutes after cuff deflation (24). There are some limitations to this method including the necessity of having a well-trained technician performing the measurements. According to Corretti et al. (24), the technician should have performed 100 supervised images to be named competent and perform at least 100 per year to maintain that competency. Since this method requires such specialized equipment and training, it can be “expensive and is highly operated dependent” (77). Kullo et al. (53) concurred, stating that there is a high level of variability between tests, a need for standardization of technique, and a need for the determination of FMD normative values. Bots et al. (14) determined from a meta-analysis of studies utilizing FMD, that it is difficult to set normative values because results from different populations (whether healthy or diseased) often overlap. Additionally, there is no standardization of the location of occlusion (above or below the antecubital space), and the length of time for occlusion varies (14). In a study examining FMD reproducibility measures over time, 40 healthy adults were tested 4 separate times with time intervals varying from 1-2 days, 1-2 weeks, and 2-4 months later. The mean coefficient of variation for FMD in all subjects was 1.8% and the majority (85%) of the studies was with 2.5% of the mean value for a given individual (105). West et al. (116) found a CV of 29.7% for FMD in patients with Type 2 diabetes. These authors reported that there is a wide range of FMD variability that has been published (CVs= 1-84%) and speculate that the causes for these differences may include technique, day-to-day variability, and measurement error (116). Pala et al. (85) explained that small variations
between observers may have created the large CVs because FMD is a percentage-ratio. Additionally, it is very difficult to measure the boundaries of the arteries in some patients which could cause error in the reading (85).

Woo et al. (121) and Kelly et al. (50) examined the impact of exercise on improving multiple factors, including endothelial function. Both of these studies both found lower FMD values in overweight and obese children and adolescents compared to published values in healthy-weight children. Woo et al. (121) found that an intervention of 6 weeks consisting of a diet-only, and a diet combined with an exercise regimen significantly improved FMD in overweight and obese children. The combined diet and exercise regimen had a greater effect on FMD that diet alone. In the diet only group, the mean percent dilation was 6.9 ± 2.0% at baseline and increased to 7.5 ± 1.9% after the 6 weeks of intervention. The combined diet and exercise group had a mean baseline dilation of 6.8 ± 2.0% which increased to 8.0 ± 1.8% after the 6 weeks intervention. Furthermore, children who continued to exercise for a year beyond the time of the baseline measures demonstrated mean increases of 8.6 ± 1.8 %. Kelly et al. (50) found that despite no changes in body composition or body weight, an improvement in endothelial function occurred with exercise training intervention in overweight and obese children (10.9 ± 0.7 yr). This 8-week training intervention included cycling exercise four times at week at 50-60% of VO₂ peak for 30 minutes and intensity, and duration was increased weekly so that the subjects would be able to cycle for 50 minutes at 70-80% VO₂ peak in the last two weeks. The variable that demonstrated the improvement in endothelial health was flow-mediated dilation area under the curve (FMD AUC). This variable reflects the brachial artery diameter changes over time. In the exercise training group, FMD AUC was 746 ± 66% per second and increased to 919 ± 94% per second.
In the control group, the FMD AUC was 731 ± 102% per second at baseline and 515 ± 73% per second after 8 weeks.

Exercise has demonstrated favorable results in endothelial function in obese adults as well. Obese adults engaged in strength training, moderate intensity aerobic exercise, and high intensity aerobic groups all showed improvements in endothelial function as measured by FMD. High intensity aerobic exercise was more effective in improving endothelial function when compared to the other groups, and there were no significant differences between strength training and moderate intensity groups (100). Similarly, a very low calorie diet stimulating weight loss in overweight and obese individuals improved FMD in both men and women by 60% (89). While many risk factors for atherosclerosis were reduced during the six week diet, the strongest correlate to endothelial function was fasting plasma glucose. The authors suggested several possible mechanisms that explain the inhibiting effect glucose exerts on endothelial function. Many of these theories involve the reduced availability of nitric oxide and oxidant stress. Raitakari and colleagues (89) cautioned that the beneficial effect of diet on endothelial function may only be attributed to overweight and obese individuals who improve their fasting plasma glucose associated with weight loss.

**Peripheral arterial tonometry.** Peripheral arterial tonometry produces pulse waveforms that can be examined for characteristics of vascular abnormalities and disease (52). Peripheral arterial tonometry utilizes an Endo-PAT 2000 device. The procedures for using this device include placing plethysmographic probes on the index fingers and monitoring digital pulse wave amplitude for 5 minutes. This procedure includes preoclusion baseline data. Following the baseline data collection, a blood pressure cuff is inflated on the non-dominant arm to suprasystolic values. This occlusion lasts for 5 minutes. When the pressure is released from the blood pressure cuff, the
pulse wave amplitude is monitored for at least 5 minutes. The result is a reactive hyperemia index (RHI) which is a ratio of the amplitude of the pulse waves one minute after cuff deflation divided by the amplitude of the signal 3.5 minutes before the cuff was inflated (12).

Many of the major cardiovascular risk factors are associated with endothelial function. Fitch et al. (34) found inverse relationships between the RHI and age, fasting glucose, triglycerides, waist-to-hip ratio, waist and neck circumference, and visceral abdominal tissue area in adults; however triglycerides were the only independent predictor of RHI. Obesity, waist circumferences, total cholesterol, triglycerides, and low density lipoproteins have also been found to be significantly related to PAT scores in adolescents (72). Additionally, higher levels of HbA1c are negatively correlated to PAT scores in adolescents (76).

Bonetti and colleagues (12) examined 94 individuals with and without coronary atherosclerosis. The subjects were identified as having coronary atherosclerosis after a coronary angiography. The threshold for endothelial dysfunction was determined to be an Endo-PAT score < 1.35 in middle-aged adults. The sensitivity and specificity of the EndoPAT to correctly identify patients with atherosclerosis were 80% and 85%, respectively (12). In a similar study, the Endo-PAT cutoff value for identifying patients with ischemic heart disease was < 1.82, with a sensitivity and specificity of 80% and 80%, respectively (73). According to the manufacturer’s website, a score less than 1.67 reflects endothelial dysfunction (48).

Currently, there is no standard value that has been accepted as the adverse cutoff point for endothelial dysfunction, however it is generally accepted that lower values indicate impairment of the endothelium. In a study examining coronary atherosclerosis in women, the control group without ischemic heart disease had a median Endo-PAT score of 2.15 compared to groups with obstructive coronary artery
disease and nonobstructive coronary artery disease that had mean Endo-PAT scores of 1.57 and 1.58, respectively (73). Shift workers have been compared to their non-shift working counterparts and were found to have lower Endo-PAT scores (1.73 ± 0.4 versus 1.94 ± 0.5) despite similar physical activity regimens, indicating endothelial dysfunction in the shift-workers (107).

Individuals with insulin resistance or diabetes are another population that has been studied extensively due to the impairments that high blood glucose causes to the endothelium. Haller and colleagues (39) examined adolescents (ages 14.6 ± 2.7) with type 1 diabetes. When compared to the control group, the diabetic adolescents had significantly lower Endo-PAT scores (1.63 ± 0.5 versus 1.95 ± 0.3). Another study found similar results in diabetic patients with 7 out of 15 diabetic adolescents having endothelial dysfunction (77). The mean score for those with endothelial dysfunction was 1.33 ± 0.16 compared to 2.0 ± 0.46 in the normal functioning group. Mahmud et al. (72) found obese adolescents with insulin resistance have lower Endo-PAT scores when compared to a healthy-weight group (1.51 ± 0.4 versus 2.06 ± 0.4).

Another study investigating the effects of disease on endothelial function and circulating biomarkers used the Endo-PAT to compare children whose mothers had preeclampsia during pregnancy to those children whose mothers had uncomplicated, healthy pregnancies. In the mothers, there were no differences in RHI between the preeclampsia group (2.13 ± 0.64) and the control group (2.18 ± 0.45). In the children (mean age 6 years), there were no differences in RHI between the preeclampsia group (1.21) and the control group (1.21) (56). While not statistically compared, the children from this study had reportedly lower RHI when compared to the adults from this study, speculatively indicating endothelial dysfunction. However, Radtke and colleagues (88) reported RHI values in prepubescent children and found that children in Tanner Stage I
had significantly lower RHI (median = 1.14) compared those children in Tanner stages II (Stage II-III, RHI median= 1.65) or higher (Stage IV-V, RHI median= 1.70). The authors attribute this to immature microvascular function and state that using the term “juvenile microvascular response” as a more appropriate description than describing it as endothelial dysfunction (88).

There are few studies that examine endothelial function and obesity. In a study that investigated adults with BMIs in healthy-weight, overweight, and obese ranges, the authors reported that healthy-weight adults had a significantly higher RHI compared to the obese group (2.36 versus 1.71, p= 0.0001) (38). Additionally, the overweight group had a significantly higher RHI when compared to the obese group (2.20 versus 1.71, p=0.009) with no differences between the healthy-weight and overweight groups. It is important to note that the obese group was older, had greater fasting blood glucose and insulin levels, higher blood pressures, and greater waist circumferences (38). Metzig et al. (75) examined the effects of an oral glucose tolerance test on post-prandial endothelial function and markers of inflammation in 34 obese children (ages 12.4 ± 2.6 yr). While there were no significant differences in endothelial function at 1 hour (RHI= 1.59 ± 0.5) or 2 hours (RHI=1.64 ± 0.5) post-prandial compared to baseline (RHI= 1.73 ± 0.5), there was a significant inverse relationship between glucose levels and RHI at 1 hour (r = -0.40). Currently, there are no known studies that examine endothelial function using the Endo-PAT 2000 comparing healthy-weight and obese children. Radtke and colleagues (88) stated that one limitation of their study examining children and pubertal changes in endothelial functioning was only examining healthy-weight children. The authors postulated that obesity could adversely affect the increase in RHI as children age. The authors based this hypothesis on the fact that BMI is often inversely correlated with RHI (72, 88).
Reliability of the Endo-PAT 2000. To determine the variability of Endo-PAT measures, Onkelinx and colleagues (84) examined the within day reliability (30 minutes between tests) and between day reliability in 18 men with coronary artery disease. For the within day measure, Endo-PAT or brachial arterial ultrasonography (BAUS) was used to determine RHI or percent change in artery size, respectively. For the within day measures, the BAUS demonstrated significantly less variability when compared to the Endo-pat measure (10% versus 18%). There was no difference in the between day variability across techniques (11% versus 11%) (84). The authors also report a significant correlation between FMD and PAT measurements ($r = 0.57$). Similarly, McCrea et al. (77) investigated the variability of Endo-PAT measures in overweight, disease-free adults by testing each subject separated by at least 1 week. The authors reported an interclass coefficient of 0.74 and a coefficient of variation of $12.2 \pm 2.2$. In another reliability study conducted in healthy, normal-weight adolescents (13-19 years old), there was no significant difference between the mean Endo-PAT score on the first visit ($1.91 \pm 0.57$) and on the second visit ($1.78 \pm 0.51$) conducted a maximum of 1 week apart (109). Overall, the Endo-PAT 2000 has demonstrated good reliability and the sensitivity detect differences to 0.01 of a RHI value, however is still not as reliable as the criterion method (BAUS) of non-invasively evaluating endothelial function.

Methods of estimating physical activity

Estimation of total daily energy expenditure (TDEE) can provide valuable insight for weight loss endeavors. TDEE includes resting metabolic rate (RMR), the thermic effect of food (TEF), and physical activity-related energy expenditure (PAEE) (61). The criterion measure for estimating physical activity is the use of doubly labeled water. This method requires the subjects to drink water that has nonradioactive isotopes of hydrogen ($^2$H) and oxygen ($^{18}$O). These isotopes evenly distribute throughout the body.
fluids and are eliminated through sweat, urine, pulmonary vapor, water and carbon dioxide. The elimination of these isotopes compared to a baseline measure allows the estimation of CO$_2$ production and thus O$_2$ consumption. The advantages of this method are that subjects can be measured under free living conditions over long periods of time, but the expense of the isotopes and analysis is a major disadvantage reducing the number of subjects that can be tested (74). Therefore, alternatives in estimating physical activity are necessary.

**Self-report and Physical Activity Surveys**

Self-reporting and physical activity surveys are often utilized in free-living conditions. Advantages of using self-report, physical activity records, or physical activity recalls are that they are easy and inexpensive to conduct when compared to other methods (61). Leenders et al. (61) utilized a seven day physical activity recall (PAR) which is a validated and standardized interview during which participants recount how much time they had spent doing activities of various intensities. The PAR was validated against heart rate monitors in 10-16 year old boys and girls. Validity and reliability of the PAR increased with age (96). When Leenders et al. (61) compared the PAR to the DLW method, there were no significant group mean differences between kilocalories (kcal) estimated by the two methods. However, when data from individual subjects were compared significant differences existed between the two methods. The authors conclude that the PAR may not be the best method for examining individual kcal counts and speculated these differences were the result of underreporting moderate intensity activities (61). This demonstrates a major disadvantage of self-reporting, due to inaccurate recall of all physical activities by the participants.
Ainsworth and colleagues (1) examined the reliability and validity of the Kaiser Physical Activity Survey (KPAS) which is focused on the physical activity of women. This survey is divided into sections to determine varying physical activity level, including: housework/caregiving, occupation, active living habits, and sports/exercise activities. The responses are categorical in nature utilizing a Likert-type scale with responses ranging from 1 ("never") to 5 ("always"). The survey also asks women to identify the frequency and intensity of the activities and then an index for each section is computed. Physical activity records, accelerometer counts, VO$_2$ peak, and body fat percentage were compared to the scores from the KPAS. The physical activity records were correlated to all KPAS indexes ($r \geq 0.35$, $p< 0.01$) except for the active living index. The accelerometer counts were related to housework/caregiving and occupational activities ($r \geq 0.35$, $p< 0.01$). Finally, sports/exercise and active living habits were significantly correlated with VO$_2$ peak ($r > 0.34$, $p< 0.01$) and body fat ($r = -0.41$, $p< 0.01$). The authors conclude that the KPAS is a valuable survey that identifies the habitual physical activities of women (1).

A popular physical activity recall for children and adolescents is the Previous Day Physical Activity Recall (PDPAR). This survey requires subjects to recall the activities from 3 p.m. to 11:30 p.m. the previous day. This time period was chosen due to the challenges that children face in remembering physical activity over longer periods. This time period is broken down in to 30 minutes intervals and children are instructed to use provided activity codes to describe their activities and intensities for the previous day. This recall has been validated in seventh through twelfth graders, but should be used with caution due to a lack of applicability in younger children (117). Advantages of the PDPAR are that specific time periods can be observed, and it can differentiate between intensity levels (sedentary time, moderate, vigorous). It has been suggested that the
recall should be administered at several different time periods to get a comprehensive view of physical activity (117). Weston et al. (117) found that the energy expenditure estimated from the PDPAR was significantly correlated with accelerometry \( r = 0.77 \) and pedometry \( r = 0.88 \) counts. In addition, energy expenditure rates determined from the PDPAR after school mode and intensity of activity estimates was significantly correlated with mean % heart rate reserve \( r = 0.53 \).

**Accelerometry**

Accelerometers are activity monitors that can record frequency, intensity, and duration of movement in one (uniaxial) or more planes (triaxial). These instruments measure accelerations and decelerations of movements and convert these data into counts. Accelerometers have internal clocks, therefore they can record times spent in moderate to vigorous activity (10). Accelerometers can be worn on various parts of the body (depending of the type of movement utilized), but can be much more expensive than pedometers. Leenders et al. (60) compared uniaxial and triaxial accelerometers to the PAR in 12 women. The uniaxial accelerometer counts were significantly correlated to the PAR \( r = 0.82, p<0.0001 \), but significantly underestimated physical activity by 46%. The triaxial accelerometer has a slightly stronger correlation with the PAR \( r = 0.89, p<0.0001 \), but still underestimated the physical activity by 25%. The authors speculated that differences between the two accelerometers relate to the differences in how they detect motion and compute energy expenditure. In addition, the authors stated that the purpose of the accelerometers is to detect motion during walking and running, but not to directly determine energy expenditure. Finally, the uniaxial accelerometer used in the study (CSA) was validated based on laboratory conditions which may result in underestimation of energy expenditure during free living conditions (60).
The advantages of using accelerometry to estimate physical activity include the ability to detect motion at moderate and vigorous intensities, the ability to detect motion in up to three planes, and storage of data over the course of several days (10). These monitors are able to provide an objective measure of physical activity; however the cost of accelerometers may reduce the number of subjects that can be tested. Finally, accelerometry cannot estimate energy expenditure from activities such as swimming, weight lifting, or activities of daily living that require carrying or transporting heavy objects (61).

**Pedometers**

Pedometers are activity monitors that are worn at the waistband at the midline of the thigh on the body (10). Pedometers typically have a lever arm that moves in response to the changes in the displacement of the hip. The movement of the lever arm results in the recording of the number steps taken. These activity monitors have been used for estimating daily step counts as well as a tool to promote physical activity. Tudor-Locke et al. (66) determined that adult participants needed to wear pedometers a minimum of three days to estimate physical activity.

In a study that examined the relationship among cardiorespiratory fitness, physical activity, and body fatness in children, Rowlands et al. (95) reported that the correlations between physical activity measured by pedometer and fitness were similar and not statistically different than the correlation between physical activity measured by accelerometer and fitness levels. The authors concluded that pedometers are a valid measure of physical activity monitoring in children. Additionally, this study found a significant relationship between low physical activity levels and decreased fitness and increased body fat levels (95).
Leenders et al. (60) found a significant correlation between pedometer step counts and the PAR ($r = 0.94, p < 0.0001$), however the pedometer underestimated the PAEE by 48%. The authors noted that pedometers do not record intensity of movement, and therefore may not provide an accurate representation of physical activity. However, the authors concluded that a high correlation between step counts and activity counts suggested that the majority of the accumulated physical activity could be measured through step counting. Additionally, Leenders and colleagues (60) reported several benefits of pedometry including low cost and the ability to give subjects immediate feedback regarding physical activity rather than having to calculate physical activity from accelerometer counts.

The New Lifestyles 1000 (NL-1000) activity monitor uses a piezo-electric accelerometry instead of a lever arm to determine step counts. The piezo-electric monitor registers steps by compressing a piezo-electric crystal when acceleration occurs. A voltage signal that is proportional to the movement is transmitted and these voltage oscillations are recorded as steps (25). A major benefit of utilizing this type of activity monitor is that it may not be affected by central obesity in the same way that traditional lever-armed monitors are. Crouter et al. (25) compared a spring arm pedometer to the piezo-electric NL-1000 during treadmill walking and free-living conditions in an overweight and obese population. The authors reported that the abdominal obesity tended to tilt the pedometers away from their vertical placement perpendicular to the ground resulting in an underestimation of step counts from the spring-levered pedometers. Crouter et al. (25) concluded that the NL-1000 is more accurate in abnormally obese populations, providing guidance when selecting an appropriate activity monitor for studies including overweight and obese individuals. Other benefits of the NL-1000 include the relatively inexpensive cost; an internal timing
mechanism which stores step data for up to seven days in one day epochs, and it provides measures of time spent in moderate to vigorous physical activity (MVPA). Ayabe et al. (5) assessed the NL-1000 for its ability to assess MVPA in healthy adults during treadmill walking. The authors reported that the NL-1000 was an accurate measure of MVPA during walking speeds of at least 85 m/min or a MET level of at least 3.4 METS. Additionally, Ayabe et al. (5) stated that age and BMI were not related to the accuracy of the recorded MVPA time.

Tudor-Locke et al. (64) proposed ranges of steps to classify activity levels for healthy adults. Less than 5,000 steps per day is considered “sedentary,” 5,000-7,499 steps per day is considered “low active,” 7,500-9,999 steps per day is considered “somewhat active”, and ≥10,000 is “active.” Those taking over 12,500 steps per day are considered “highly active.” For children, recommendations are slightly higher. Tudor-Locke and colleagues (65) also identified appropriate pedometer step count cut points for 6-12 year old boys and girls. The optimal step counts are 15,000 steps per day and 12,000 steps per day for boys and girls, respectively. Achieving less than these cut points results in an increased likelihood of a child being overweight or obese (65).

**Conclusion**

Obesity and maturational status affect physiological responses at rest, and during and after exercise. Children typically have higher resting physiological values when compared to adults, and also tend to recover more rapidly. Obese children tend to respond physiologically similar to overweight adults, having higher resting and submaximal values, but lower performance overall. With the increasing number of overweight and obese children in the United States, it is important to understand the
implications of excess body fat on health and fitness. Early interventions may be valuable in preventing long term consequences of overweight and obesity.


Chapter 3: Methods and Procedures

Subjects

The subjects for this study included 33 white, non-Hispanic children aged 7-11 years and 27 white, non-Hispanic young women between 25-45 years of age. The pediatric subject cohort included 20 healthy-weight (HW; BMI for age and sex between the 5th and the 84th percentile) children (13 boys and 7 girls) and 13 obese (OB; BMI for age and sex > 95th percentile) children (11 boys and 2 girls). The adult subject cohort included 15 healthy weight (HW; BMI between 18.5 and 24.9 kg/m²) women and 12 obese (OB; BMI >29.9 kg/m²) women. Subjects were recruited via physician and healthcare professional recommendations, local public school physical education teacher recommendations, and local advertisements. All subjects were apparently healthy with no present or history of cardiac issues, hypertension or taking medications to control hypertension, diabetes or taking medications to manage diabetes, were current non-smokers and did not currently have orthopedic limitations that would prohibit them from performing the graded exercise test. Prior to participation written consent was obtained for each of the women, and parental consent and verbal assent was obtained for each child subject in accordance with the policies and procedures of the University of Kentucky Office of Research Integrity. All women filled out a physical activity readiness questionnaire (PAR-Q) and a medical history form.

Anthropometric and body composition measures

All subjects completed anthropometric and body composition measures including standing height, body mass, three circumference site measures, and a total body dual-energy X-ray absorptiometry (DXA) scan during a single testing session. All subjects were measured in light-weight clothing containing no metal and without shoes. Standing height was determined to the nearest 0.1 cm using a wall-fixed stadiometer (SecaModel 216; Seca North American West, Ontario, CA) with the subjects’ hands positioned on the
hips during a maximal inhalation. Body mass was determined to the nearest 0.01 kg using a calibrated electronic scale (American scale, DIGI: DI-10, Rice Lake, WI). Three circumference measurements (waist, abdomen, and hip) were determined to the nearest 0.1 cm for each subject. These measurements were performed in triplicate by a member of the research team using a fiberglass anthropometric tape (Medco, Tonawanda, NY) and the standard procedures from the Anthropometric Standardization Reference Manual (70). In addition, body composition was measured using total body DXA scans performed using a Lunar DPX-IQ (Lunar Inc., Madison, WI) bone densitometer. The subjects were instructed to remove all objects such as jewelry or eyeglasses and wore light-weight clothing containing no metal during the scanning procedure. All scans were analyzed by a single trained investigator using the Lunar software version 4.3. DXA absolute and relative fat-free mass (FFM; kg), fat mass (kg), and mineral-free lean mass (kg) were determined for each subject.

**Resting Heart Rate and ECG**

Resting heart rate and ECG were measured prior to endothelial health and maximal graded exercise testing using a 12-lead electrocardiogram (Nihon Koden, 1550A and 1350A; Nihon Kohden American, Inc., Foothill Ranch, CA) recording. These resting measures were taken following 15 minutes of supine rest in a quiet thermoneutral room.

**Endothelial Health**

The Endo-PAT 2000 (Itamar Medical, Israel) was used to measure peripheral arterial pulse waves using finger probe plethysmography. Resting blood pressure was measured in the dominant arm and then the blood pressure cuff was moved to the non-dominant arm. Each subject sat in a relaxed position with arms extended in front of them during the measurement. Each subject was fitted with probes over the index finger of each hand. The probes were inflated followed by five minutes of baseline data
collection. Next, the blood pressure cuff on the non-dominant arm was inflated to and maintained at 200 mm Hg for the adults and to at least 40 mm Hg above systolic blood pressure for the children to ensure total brachial occlusion. This occlusion lasted five minutes while the data collection for the finger probes continued. After five minutes of occlusion, the blood pressure cuff was deflated and the reactive hyperemia was measured for five additional minutes. An Endo-PAT score or reactive hyperemia index (RHI) was calculated for each subject after testing. This score was determined as a ratio of the amplitude of the pulse waves one minute after cuff deflation divided by the amplitude of the signal 3.5 minutes before the cuff was inflated (12). Following the measurements, children participating in the study were asked to indicate their level of pain or discomfort using the Wong-Baker Visual Analog Scale (109,120). This standard procedure has been used previously for determining endothelial function in both adult and child populations (12,38,39,43,54,56,72,73,76,77,107,109).

**Graded Exercise Test**

The maximal graded exercise tests (Max GXT) took place at the University of Kentucky’s Pediatric Exercise Physiology Lab (UK PEP Lab) using an indirect calorimetry testing system with integrated electrocardiogram (ECG) (SensorMedics Vmax C Lite, Yorba, CA) and a treadmill ergometer (SensorMedics 2000, Yorba, CA). During the tests, oxygen consumption and cardiovascular responses were continuously measured and monitored. The Max GXTs were performed using progressive 2 minute workload stages. During the final minute of each stage, the respiratory exchange ratio (RER), blood pressure, and ratings of perceived exertion (RPE) were recorded. Heart rate was recorded in the last ten seconds of each stage. The protocol used was identical for both the children and adults. RPE were recorded in adults and children using the original (6-20) Borg Scale, and children were additionally ask to rate their
perceived exertion using the OMNI picture scale with the numerical classifications removed (13,90).

The treadmill Max GXT test began with the subject walking on the treadmill at a speed of 2.2 mph and at 0% grade (incline). With every stage, the treadmill speed increased by 0.4 mph. The treadmill grade or incline increased by 2% at stage 3 of the test and increased by 2% at the beginning of each successive stage. The test termination was based on volitional fatigue or the presence of any absolute or relative contraindications to continuing according to the ACSM guidelines (4). Verbal encouragement was given throughout the test. Upon completion of the Max GXT, a passive recovery (cool down) in a seated position occurred with heart rate and blood pressure measures taken and recorded every minute and continuous VO$_2$ consumption was measured for five consecutive minutes. Absolute heart rate recovery was defined as the heart rate (bpm) at one minute post-exercise subtracted from the maximal heart rate (bpm). Relative heart rate recovery was defined as absolute heart rate recovery divided by maximal heart rate and multiplied by 100. Absolute oxygen recovery was defined as the oxygen consumption (ml/kg/min) at one minute post-exercise subtracted from peak oxygen consumption (ml/kg/min). Relative oxygen recovery was defined as absolute oxygen recovery divided by peak oxygen consumption and multiplied by 100.

The equation, peak heart rate $= 206 - 0.88$ (age) was used as one criterion for determining if a peak VO$_2$ had been reach by each of the adult subjects (37). Additionally, either an RPE of 17 or greater and/or an RER of 1.1 or greater were used as criteria for achievement of peak VO$_2$ in the adults. For the children, a heart rate of greater than or equal to 190 bpm was used as an indicator of maximal effort during the Max GXT (67,92). Rowland (92) stated that due to the wide variability of maximal heart rate and RER in the pediatric population, it is important for the subject to demonstrate evidence of maximal effort.
Physical Activity Assessment

All subjects were asked to wear a New Lifestyles-1000 (NL; LeesTown, MO) medical grade activity monitor for three consecutive days including one weekend day and two weekdays, following the testing session in the laboratory. This monitor was chosen for its simplicity, durability, low cost, seven day memory capability, accuracy, reliability, and ability to assess time spent in MVPA (5, 25). Subjects were instructed to place the activity monitor on their waistband (or on a nylon belt provided) at the anterior midline of the thigh, on the right side of the body, according to manufacturer’s recommendations. Subjects placed the monitor on their waistband when they woke up in the morning and were instructed to take it off when they went to bed at night, unless they came in contact with water (e.g., bathing and swimming). To avoid reactivity, all activity monitors were sealed using a piece of tape after stride length, time of day, and intensity threshold were internally programmed. Stride length was determined by instructing each subject to take 10 steps down a corridor at their normal walking speed. The distance traveled over 10 steps was then measured using a tape measure and divided by the 10 steps taken. Stride length was calculated as the average of two 10-step trials. The researchers set the NL activity monitor MVPA threshold at activity setting #3 for the children and #4 for the adults, based on recommendations from the manufacturer. Outcome variables of interest were total steps (over the three days), total time spent in MVPA, average weekday steps and time spent in MVPA, and average weekend steps and time spent in MVPA.

Additionally, subjects were asked to describe their physical activity using a physical activity log and fill out an age-appropriate, validated activity questionnaires (1, 117). Each adult woman filled out a previously described parental background survey regarding general demographics and attitudes and beliefs towards physical activity (71).
Statistical Analyses

Primary variables (absolute and relative VO$_2$ peaks, absolute and relative HRRec, and reactive hyperemia index) were examined for univariate and multivariate outliers and normality. Variables were also examined for high correlations to avoid multicollinearity issues. If high correlations existed between a set of variables, one of the variables was removed from the model. Relative VO$_2$ peak remained in the model because it is our index of fitness and relative heart rate recovery remained in the model because it accounted for differences in maximal heart rate rates. Furthermore, reactive hyperemia index was removed from the model because it violated the assumption of equality of variance.

Therefore, relative VO$_2$ peak and relative HRRec were entered as dependent variables in the factorial multivariate analysis of variance (MANOVA). The independent factors were age (adult or child) and obesity status (healthy-weight or obese). Absolute VO$_2$ peak and absolute HRRec were analyzed individually utilizing a factorial univariate analysis of variance (ANOVA). Finally, RHI in women was analyzed (without analysis of children due to lack of variance) using an independent t-test. Furthermore, relationships among the primary variables of interest (VO$_2$ peak, HRRec, and RHI) were examined using Pearson product moment correlation analyses. All analyses were performed using IBM SPSS, version 20.0. The level of significance was set aprior at $p < 0.05$. 

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Chapter 4: Results

Subjects

This study included a total of 60 subjects (27 women ages 25-45 yrs, and 33 children ages 7-11 yrs). Demographic data for these subjects are shown in Tables 1-3. Thirty-five of the subjects were healthy-weight and 25 of the subjects were obese (Table 2). There were 15 healthy-weight (BMI >18.5 and < 25 kg/m²) women and 12 obese (BMI > 29.9 kg/m²) women, and 20 healthy-weight (BMI for age and sex > 5th percentile and < 85th percentile) children and 13 obese (BMI ≥ 95th percentile) children (Table 3).

Demographic Data

The group of women was older, taller, weighed more, and had greater BMIs than the group of children (Table 1). There were no differences between HW and OB groups in age and height, but the OB group weighed more and had greater BMIs (Table 2). Individual group comparisons demonstrated that the HW children were older than the OB children, and younger than the adult groups (Table 3). There were no differences in height between HW and OB children, but HW children were shorter than adult groups. The HW children weighed less than the OB children, the HW adults, and the OB adults, and had lower BMIs compared to the OB children, HW adults, and OB adults. The OB children were younger than both adults groups. The OB children were shorter than both adult groups. The OB children weighed less than both adult groups and had a lower BMI than the OB adults, but had a higher BMI than the HW adults. The HW women were older and taller than the OB adults, but older and taller than both children’s groups. The HW women weighed less than the OB women, but more than the HW children and the OB children. The HW women had a lower BMI than OB women and children, but higher BMI than the HW children. The OB women weighed more and had a higher BMI compared to all other groups.
Table 1. Demographic information for women and children

<table>
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<th>Adults, n=27</th>
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<tr>
<td>Age (yr)</td>
<td>9.8 ± 1.6</td>
<td>34.7 ± 6.5</td>
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<tr>
<td>Sex (% female)</td>
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<tr>
<td>Height (cm)</td>
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<tr>
<td>Weight (kg)</td>
<td>42.7 ± 14.4</td>
<td>79.9 ± 22.1</td>
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<td>BMI (kg/m²)</td>
<td>20.9 ± 5.8</td>
<td>28.8 ± 8.5</td>
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Table 2. Demographic information for healthy-weight and obese subjects

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<tr>
<td>Age (yr)</td>
<td>20.9 ± 13.1</td>
<td>21.1 ± 13.8</td>
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<tr>
<td>Sex (% female)</td>
<td>63</td>
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<tr>
<td>Height (cm)</td>
<td>153.6 ± 15.4</td>
<td>153.1 ± 15.9</td>
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<td>Weight (kg)</td>
<td>46.7 ± 15.2</td>
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<tr>
<td>BMI (kg/m²)</td>
<td>19.4 ± 3.0</td>
<td>31.9 ± 7.1</td>
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Table 3. Demographic information for healthy-weight and obese women and children

<table>
<thead>
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<th>Adults, n=27</th>
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<tbody>
<tr>
<td></td>
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<tr>
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<tr>
<td>BMI (kg/m²)</td>
<td>17.0 ± 1.6</td>
<td>26.9 ± 4.7</td>
</tr>
</tbody>
</table>
Primary Variables of Interest

Fitness and heart rate recovery.

There was a significant main effect of age (Wilks' lambda, $F(2, 55) = 39.305, p<0.001$) and a significant main effect for obesity status (Wilks' lambda, $F(2, 55) = 35.119, p<0.001$). There was not a significant interaction effect of age and obesity status (Wilks' lambda, $F(2, 55) = 0.681, p = 0.510$). When further examining the between-subjects effects, age had a significant impact on both relative VO$_2$ peak (Wilks' lambda, $F (1, 56) = 26.780, p < 0.001$) and relative HRRec (Wilks' lambda, $F (1, 56) = 32.764, p < 0.001$). Obesity status had a significant impact on relative VO$_2$ peak (Wilks' lambda, $F (1, 56) = 65.400, p < 0.001$).

Results from the univariate factorial ANOVA for absolute VO$_2$ peak demonstrated significant main effect for age ($F (1, 56) = 33.236, p < 0.001$), but no significant effect for obesity status ($F (1, 56) = 0.848, p=0.361$) and no interaction effect ($F (1, 56) = 0.027, p=0.869$). Similarly, the results from the univariate factorial ANOVA for absolute HRRec revealed a significant main effect for age ($F (1, 56) = 37.324, p < 0.001$), but no significant effect of obesity status ($F (1, 56) = 0.338, p = 0.563$) and no interaction effect ($F (1, 56) = 0.996, p = 0.323$). Finally, the results of the independent t-test used to determine whether a difference existed in RHI between healthy-weight and obese women found no significant differences ($t (24) = -1.577, p=0.128$).

The results of the MANOVA demonstrated that relative VO$_2$ peak was higher in children ($40.94 \pm 10.18$ ml/kg/min) than women ($31.22 \pm 8.50$ ml/kg/min) (Table 4). Additionally, relative VO$_2$ peak was significantly greater in healthy-weight groups ($42.56 \pm 8.80$ ml/kg/min) compared to obese groups ($28.18 \pm 6.29$ ml/kg/min) (Table 5). Overall, mean absolute VO$_2$ peak was greater in women ($2.36 \pm 0.44$ L/min) compared
to the children (1.67 ± 0.45 L/min) (Table 4), but there were no significant differences between healthy-weight (1.92 ± 0.56 L/min) and obese (2.07 ± 0.57 L/min) groups (Table 5). The maximal graded exercise test lasted longer for the children when compared to the adults (14.00 ± 2.59 and 13.28 ± 2.49 minutes, respectively). HW group had a longer mean test time compared to the OB group (15.21 ± 1.64 and 11.52 ± 1.98 minutes, respectively). HW children had a longer mean time on treadmill compared to OB children and OB women, but the time on treadmill was very similar between HW women and children (Table 6 & 7).

Age had a significant impact on relative heart rate recovery, where children had a greater relative heart rate recovery at one minute post exercise (25.67 ± 6.84%) compared to the women (15.79 ± 6.54%; Table 4). There were no significant differences in relative heart rate recovery between the HW (21.60 ± 8.11%) and the OB (20.70 ± 8.7%) groups (Table 5). Women had significantly slower absolute heart rate recovery (29.85 ± 12.91 bpm) compared to children (51.55 ± 14.31 bpm) (Table 4). There were no differences in absolute heart rate recovery between healthy-weight individuals (43.00 ± 16.98 bpm) and obese individuals (40.08 ± 18.20 bpm) (Table 5).

**Reactive hyperemia index.**

There were no significant differences between healthy-weight and obese women (p=0.128; Table 6). While children were not statistically analyzed due to the lack of a normal distribution, the children had a much lower RHI (1.13 ± 0.15) than the adults (2.40 ± 0.44; Table 4).
Other Physiological Measures

Resting heart rate.

Overall, women had a lower resting heart rate when compared to children (69.3 ± 11.3 and 77.2 ± 14.1 bpm; Table 4), and HW had a lower resting heart rate than the OB (71.3 ± 13.4 and 77.0 ± 12.9 bpm) subjects (Table 5). The HW children had lower resting heart rates (75.1 ± 14.2 bpm) when compared to OB children (80.5 ± 13.7 bpm; Table 7), were not different from the OB adults (73.2 ± 11.3 bpm), and were greater than HW adults (66.2 ± 10.6 bpm; Table 6). OB children had greater heart rates compared to healthy-weight adults and were greater than OB adults. HW women had lower heart rate compared to obese women (Table 6).

Blood pressure.

The resting systolic blood pressure was significantly greater in the women (105.8 ± 10.7 mm Hg) compared to children (117.1 ± 10.6 mm Hg; Table 4), but there was no difference in diastolic blood pressure for the women (67.6 ± 8.3 mmHg) versus the children (74.6 ± 6.0 mm Hg; Table 4). HW subjects had lower mean resting systolic blood pressure measures (106.7 ± 10.8 mm Hg) when compared to obese subjects (116.7 ± 11.2 mm Hg), but there were no differences in diastolic blood pressure between these groups (HW, 68.4 ± 7.5 mm Hg and OB, 74.1 ± 7.8 mm Hg; Table 5). HW children had a lower mean systolic blood pressure measure (102.1 ± 10.8 mm Hg) when compared to OB children (111.4 ± 8.0 mm Hg; Table 7), HW women (112.8 ± 7.6 mm Hg) and OB women (122.5 ± 11.5 mm Hg; Table 6). OB children had a mean systolic blood pressure that was lower than OB adults; however there were no differences between OB children and HW women for this variable. HW women had a lower mean
systolic blood pressure compared to OB women. There were no differences in diastolic values among subjects.

**Body composition.**

Women had greater amounts of fat mass (31.3 ± 17.1 kg), fat-free mass (46.6 ± 5.1 kg), and % fat (34.7 ± 12.2 %) compared to children (12.8 ± 10.2 kg, 28.6 ± 5.8 kg, 27.4 ± 12.9%; Table 4). The HW group had lower amounts of fat mass (11.0 ± 6.6 kg), fat-free mass (34.6 ± 9.7 kg), and a lower mean % fat (22.4 ± 7.4) compared to the OB group (35.3 ± 15.7 kg, 39.6 ± 11.1, 45.3 ± 7.0%; Table 5). HW children had a lower fat mass (6.4 ± 2.9 kg) compared to OB children (22.7 ± 9.4 kg; Table 7), HW women (17.2 ± 4.7 kg), and OB women (49.0 ± 7.1 kg). HW children had a lower fat-free mass (27.3 ± 5.4 kg) compared to OB children (30.6 ± 6.0 kg), HW women (44.4 ± 3.5 kg), and OB women (49.3 ± 5.5 kg). HW children had a lower % fat (18.5 ± 6.1%) compared to OB children (41.1 ± 6.9%), HW women (27.6 ± 5.6%), and OB women (49.8 ± 3.7%). OB children had lower fat mass than HW women and OB women. OB children had lower fat-free mass than HW women and OB women. OB children had a lower mean % fat than the OB women, but a greater % fat than the HW women. HW women had a lower amount of fat mass, fat-free mass, and % fat when compared to OB women (Table 6).

**Maximal heart rate.**

The group mean for maximal heart rate was greater in children (200.5 ± 8.8 bpm) compared to women (188.0 ± 8.8 bpm; Table 4) and the HW group had a greater maximal heart rate (197.6 ± 11.1 bpm) compared to OB groups (191.1 ± 91 bpm; Table 5). HW children had a greater mean maximal heart rate (202.7 ± 9.2 bpm) compared to OB children (197.1 ± 7.2 bpm), HW women (190.7 ± 10.1 bpm), and OB women (184.6 ±
5.9 bpm). HW and OB children (Table 7) and HW and OB women (Table 6) had similar maximal heart rate responses.

**Oxygen recovery.**

The group mean absolute and relative oxygen recovery was greater in children (21.7 ± 8.1 ml/kg/min; 54.1 ± 12.4%) compared to adult women (13.3 ± 5.3 ml/kg/min; 42.0 ± 12.0 %; Table 4). Absolute oxygen recovery was greater in HW (19.8 ± 8.8 ml/kg/min) compared to OB group (15.5 ± 6.7ml/kg/min), but relative oxygen recovery was lower in the HW group (46.2 ± 12.3%) compared to the OB group (52.4 ± 14.6 %; Table 5). HW children had a greater absolute oxygen recovery (23.7 ± 9.5 ml/kg/min) compared to the OB children (18.7 ± 4.5 ml/kg/min), HW women (13.5 ± 3.5 ml/kg/min), and OB women (11.7 ± 6.9 ml/kg/min). OB children had a greater absolute oxygen recovery compared to HW and OB women. HW women had a greater absolute oxygen recovery than the OB women (Table 6). HW children had a lower relative oxygen recovery (51.1 ± 13.2 %) than OB children (58.5 ± 10.1 ml/kg/min) and a greater oxygen recovery compared to HW women (39.4 ± 6.9 %) and OB women (45.2 ± 16.3 %). OB children had a greater relative oxygen consumption compared to all other groups. HW women had a lower relative oxygen consumption value compared to the OB women.

**Physical activity.**

In general, children took more steps and spent more time in MVPA compared to adult women (Table 8). The HW group was also more physically active than the obese group demonstrated by higher step counts and more time spent in MVPA (Table 9). HW children and HW women were more active compared to their obese counterparts (Tables 10-11).
<table>
<thead>
<tr>
<th></th>
<th>Children, n=33</th>
<th>Women, n=27</th>
</tr>
</thead>
<tbody>
<tr>
<td>Resting heart rate (bpm)</td>
<td>77.2 ± 14.1</td>
<td>69.3 ± 11.3</td>
</tr>
<tr>
<td>Systolic blood pressure (mm Hg)</td>
<td>105.8 ± 10.7</td>
<td>117.1 ± 10.6</td>
</tr>
<tr>
<td>Diastolic blood pressure (mm Hg)</td>
<td>67.6 ± 8.3</td>
<td>74.6 ± 6.0</td>
</tr>
<tr>
<td>Fat mass (kg)</td>
<td>12.8 ± 10.2</td>
<td>31.3 ± 17.1</td>
</tr>
<tr>
<td>Fat-free mass (kg)</td>
<td>28.6 ± 5.8</td>
<td>46.6 ± 5.1</td>
</tr>
<tr>
<td>Body fat (%)</td>
<td>27.4 ± 12.9</td>
<td>34.7 ± 12.2</td>
</tr>
<tr>
<td>Reactive hyperemia index</td>
<td>1.1 ± 0.2 (n=32)</td>
<td>2.2 ± 0.7 (n=26)</td>
</tr>
<tr>
<td>Absolute VO$_2$ peak (L/min)</td>
<td>1.7 ± 0.5</td>
<td>2.4 ± 0.4*</td>
</tr>
<tr>
<td>Relative VO$_2$ peak (ml/kg/min)</td>
<td>40.9 ± 10.2</td>
<td>31.2 ± 8.5*</td>
</tr>
<tr>
<td>Absolute O$_2$ Recovery (ml/kg/min)</td>
<td>21.7 ± 8.1 (n=32)</td>
<td>13.3 ± 5.3 (n=25)</td>
</tr>
<tr>
<td>Relative O$_2$ Recovery (%)</td>
<td>54.1 ± 12.4 (n=32)</td>
<td>42.0 ± 12.0 (n=25)</td>
</tr>
<tr>
<td>Maximal heart rate (bpm)</td>
<td>200.5 ± 8.8</td>
<td>188.0 ± 8.8</td>
</tr>
<tr>
<td>Absolute heart rate recovery (bpm)</td>
<td>51.5 ± 14.3</td>
<td>29.9 ± 12.9*</td>
</tr>
<tr>
<td>Relative heart rate recovery (%)</td>
<td>25.7 ± 6.8</td>
<td>15.8 ± 6.5*</td>
</tr>
<tr>
<td>Duration of exercise test (min)</td>
<td>14.0 ± 2.6</td>
<td>13.3 ± 2.5</td>
</tr>
</tbody>
</table>

*p < 0.05
Table 5. Physiological data for healthy-weight and obese subjects

<table>
<thead>
<tr>
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<th>HW, n=35</th>
<th>OB, n=25</th>
</tr>
</thead>
<tbody>
<tr>
<td>Resting heart rate (bpm)</td>
<td>71.3 ± 13.4</td>
<td>77.0 ± 12.9</td>
</tr>
<tr>
<td>Systolic blood pressure (mm Hg)</td>
<td>106.7 ± 10.8</td>
<td>116.7 ± 11.2</td>
</tr>
<tr>
<td>Diastolic blood pressure (mm Hg)</td>
<td>68.4 ± 7.5</td>
<td>74.1 ± 7.8</td>
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<tr>
<td>Fat mass (kg)</td>
<td>11.0 ± 6.6</td>
<td>35.3 ± 15.7</td>
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<tr>
<td>Fat-free mass (kg)</td>
<td>34.6 ± 9.7</td>
<td>39.6 ± 11.1</td>
</tr>
<tr>
<td>Body fat (%)</td>
<td>22.4 ± 7.4</td>
<td>45.3 ± 7.0</td>
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<tr>
<td>Reactive hyperemia index</td>
<td>1.7 ± 0.8 (n=33)</td>
<td>1.5 ± 0.6</td>
</tr>
<tr>
<td>Absolute VO$_2$ peak (L/min)</td>
<td>1.9 ± 0.5</td>
<td>2.1 ± 0.6</td>
</tr>
<tr>
<td>Relative VO$_2$ peak (ml/kg/min)</td>
<td>42.6 ± 8.8</td>
<td>28.2 ± 6.3*</td>
</tr>
<tr>
<td>Absolute O2 Recovery (ml/kg/min)</td>
<td>19.8 ± 8.8 (n=33)</td>
<td>15.5 ± 6.7 (n=24)</td>
</tr>
<tr>
<td>Relative O2 Recovery (%)</td>
<td>46.2 ± 12.3 (n=33)</td>
<td>52.4 ± 14.6 (n=24)</td>
</tr>
<tr>
<td>Maximal heart rate (bpm)</td>
<td>197.6 ± 11.1</td>
<td>191.1 ± 9.1</td>
</tr>
<tr>
<td>Absolute heart rate recovery (bpm)</td>
<td>43.0 ± 17.0</td>
<td>40.1 ± 18.2</td>
</tr>
<tr>
<td>Relative heart rate recovery (%)</td>
<td>21.6 ± 8.1</td>
<td>20.7 ± 8.7</td>
</tr>
<tr>
<td>Duration of exercise test (min)</td>
<td>15.2 ± 1.6</td>
<td>11.5 ± 2.0</td>
</tr>
</tbody>
</table>

*p < 0.05
Table 6. Physiological data for healthy-weight and obese women

<table>
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<tr>
<th></th>
<th>HW, n=15</th>
<th>OB, n=12</th>
</tr>
</thead>
<tbody>
<tr>
<td>Heart rate (bpm)</td>
<td>66.2 ± 10.6</td>
<td>73.2 ± 11.3</td>
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<tr>
<td>Systolic blood pressure (mm Hg)</td>
<td>112.8 ± 7.6</td>
<td>122.5 ± 11.5</td>
</tr>
<tr>
<td>Diastolic blood pressure (mm Hg)</td>
<td>72.5 ± 4.7</td>
<td>77.2 ± 6.7</td>
</tr>
<tr>
<td>Fat mass (kg)</td>
<td>17.2 ± 4.7</td>
<td>49.0 ± 7.1</td>
</tr>
<tr>
<td>Fat-free mass (kg)</td>
<td>44.4 ± 3.5</td>
<td>49.3 ± 5.5</td>
</tr>
<tr>
<td>Body fat (%)</td>
<td>27.6 ± 5.6</td>
<td>49.8 ± 3.7</td>
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<tr>
<td>Reactive hyperemia index</td>
<td>2.4 ± 0.8 (n=14)</td>
<td>2.0 ± 0.5</td>
</tr>
<tr>
<td>Absolute VO₂ peak (L/min)</td>
<td>2.3 ± 0.4</td>
<td>2.4 ± 0.4</td>
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<tr>
<td>Relative VO₂ peak (ml/kg/min)</td>
<td>36.9 ± 5.9</td>
<td>24.2 ± 5.3</td>
</tr>
<tr>
<td>Absolute O₂ recovery (ml/kg/min)</td>
<td>13.5 ± 3.5 (n=14)</td>
<td>11.7 ± 6.9 (n=11)</td>
</tr>
<tr>
<td>Relative O₂ recovery (%)</td>
<td>39.4 ± 6.9 (n=14)</td>
<td>45.2 ± 16.3 (n=11)</td>
</tr>
<tr>
<td>Maximal heart rate (bpm)</td>
<td>190.7 ± 10.0</td>
<td>184.6 ± 5.9</td>
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<tr>
<td>Absolute heart rate recovery (bpm)</td>
<td>32.4 ± 13.6</td>
<td>26.7 ± 11.8</td>
</tr>
<tr>
<td>Relative heart rate recovery (%)</td>
<td>16.9 ± 6.8</td>
<td>14.4 ± 6.1</td>
</tr>
<tr>
<td>Duration of exercise test</td>
<td>15.1 ± 1.5</td>
<td>11.1 ± 1.4</td>
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### Table 7. Physiological data for healthy-weight and obese children

<table>
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<tr>
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<th>HW, n= 20</th>
<th>OB, n= 13</th>
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<tr>
<td>Heart rate (bpm)</td>
<td>75.1 ± 14.2</td>
<td>80.5 ± 13.7</td>
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<tr>
<td>Systolic blood pressure (mm Hg)</td>
<td>102.1 ± 10.8</td>
<td>111.4 ± 8.0</td>
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<tr>
<td>Diastolic blood pressure (mm Hg)</td>
<td>65.3 ± 7.8</td>
<td>71.2 ± 7.9</td>
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<tr>
<td>Fat mass (kg)</td>
<td>6.4 ± 2.9</td>
<td>22.7 ± 9.4</td>
</tr>
<tr>
<td>Fat-free mass (kg)</td>
<td>27.3 ± 5.4</td>
<td>30.6 ± 6.0</td>
</tr>
<tr>
<td>Body fat (%)</td>
<td>18.5 ± 6.1</td>
<td>41.1 ± 6.9</td>
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<tr>
<td>Reactive hyperemia index</td>
<td>1.1 ± 0.2 (n=19)</td>
<td>1.1 ±0.1</td>
</tr>
<tr>
<td>Absolute VO\textsubscript{2} peak (L/min)</td>
<td>1.6 ± 0.5</td>
<td>1.7 ± 0.5</td>
</tr>
<tr>
<td>Relative VO\textsubscript{2} peak (ml/kg/min)</td>
<td>46.8 ± 8.2</td>
<td>31.9 ± 4.7</td>
</tr>
<tr>
<td>Absolute O\textsubscript{2} recovery (ml/kg/min)</td>
<td>23.7 ± 9.5 (n=19)</td>
<td>18.7 ± 4.5</td>
</tr>
<tr>
<td>Relative O\textsubscript{2} recovery (%)</td>
<td>51.1 ± 13.2 (n=19)</td>
<td>58.5 ± 10.1</td>
</tr>
<tr>
<td>Maximal heart rate (bpm)</td>
<td>202.7 ± 9.2</td>
<td>197.1 ± 7.2</td>
</tr>
<tr>
<td>Absolute heart rate recovery (bpm)</td>
<td>51.0 ± 15.0</td>
<td>52.4 ± 13.8</td>
</tr>
<tr>
<td>Relative heart rate recovery (%)</td>
<td>25.1 ± 7.3</td>
<td>26.5 ± 6.3</td>
</tr>
<tr>
<td>Duration of exercise test</td>
<td>15.3 ± 1.8</td>
<td>11.9 ± 2.3</td>
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### Table 8: Total steps and time spent in MVPA in children and adults

<table>
<thead>
<tr>
<th></th>
<th>Children, n=33</th>
<th>Adults, n=27</th>
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<tbody>
<tr>
<td>Average total steps</td>
<td>10483.7 ± 3606.6 (n=30)</td>
<td>7114.1 ± 3628.9 (n=26)</td>
</tr>
<tr>
<td>(steps/day)</td>
<td></td>
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<tr>
<td>Average weekday steps</td>
<td>10703.43 ± 3542.1 (n=30)</td>
<td>6935.0 ± 3553.2 (n=26)</td>
</tr>
<tr>
<td>(steps/day)</td>
<td></td>
<td></td>
</tr>
<tr>
<td>Average weekend steps</td>
<td>9926.0 ± 5697.7 (n=29)</td>
<td>7286.0 ± 5782.2 (n=26)</td>
</tr>
<tr>
<td>(steps/day)</td>
<td></td>
<td></td>
</tr>
<tr>
<td>Average time spent in MVPA</td>
<td>45.8 ± 22.8 (n=30)</td>
<td>21.9 ± 17.9 (n=26)</td>
</tr>
<tr>
<td>(min/day)</td>
<td></td>
<td></td>
</tr>
<tr>
<td>Average time spent in MVPA on weekday (min/day)</td>
<td>46.8 ± 22.6 (n=30)</td>
<td>22.1 ± 19.0 (n=26)</td>
</tr>
<tr>
<td>Average time spent in MVPA on weekend (min/day)</td>
<td>43.5 ± 33.0 (n=29)</td>
<td>21.6 ± 27.1 (n=26)</td>
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</tbody>
</table>
Table 9: Total steps and time spent in MVPA in healthy-weight and obese

<table>
<thead>
<tr>
<th></th>
<th>HW, n=35</th>
<th>OB, n=25</th>
</tr>
</thead>
<tbody>
<tr>
<td>Average total steps (steps/day)</td>
<td>10030.6 ± 3967.1 (n=34)</td>
<td>7201.8 ± 3368.8 (n=22)</td>
</tr>
<tr>
<td>Average weekday steps (steps/day)</td>
<td>9750.1 ± 3762.5 (n=34)</td>
<td>7723.2 ± 4112.9 (n=22)</td>
</tr>
<tr>
<td>Average weekend steps (steps/day)</td>
<td>10357.2 ± 6618.5 (n=33)</td>
<td>6159.1 ± 3132.0 (n=22)</td>
</tr>
<tr>
<td>Average time spent in MVPA (min/day)</td>
<td>40.2 ± 25.2 (n=34)</td>
<td>26.2 ± 18.9 (n=22)</td>
</tr>
<tr>
<td>Average time spent in MVPA on weekday (min/day)</td>
<td>38.6 ± 24.8 (n=34)</td>
<td>30.2 ± 23.1 (n=22)</td>
</tr>
<tr>
<td>Average time spent in MVPA on weekend (min/day)</td>
<td>43.1 ± 36.5 (n=33)</td>
<td>18.2 ± 15.1 (n=22)</td>
</tr>
</tbody>
</table>

Table 10. Total steps and time spent in MVPA in children

<table>
<thead>
<tr>
<th></th>
<th>HW, n=20</th>
<th>OB, n=13</th>
</tr>
</thead>
<tbody>
<tr>
<td>Average total steps (steps/day)</td>
<td>11125.5 ± 4102.4 (n=19)</td>
<td>9375.1 ± 2300.8 (n=11)</td>
</tr>
<tr>
<td>Average weekday steps (steps/day)</td>
<td>10942.4 ± 3695.8 (n=19)</td>
<td>10290.6 ± 3391.4 (n=11)</td>
</tr>
<tr>
<td>Average weekend steps (steps/day)</td>
<td>11381.6 ± 6591.6 (n=18)</td>
<td>7544.0 ± 2642.1 (n=11)</td>
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<tr>
<td>Average time spent in MVPA (min/day)</td>
<td>50.1 ± 25.8 (n=19)</td>
<td>38.4 ± 14.9 (n=11)</td>
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<tr>
<td>Average time spent in MVPA on weekday (min/day)</td>
<td>48.2 ± 24.5 (n=19)</td>
<td>44.2 ± 19.9 (n=11)</td>
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<tr>
<td>Average time spent in MVPA on weekend (min/day)</td>
<td>53.8 ± 37.3 (n=18)</td>
<td>26.8 ± 13.6 (n=11)</td>
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Table 11. Total steps and time spent in MVPA in adults

<table>
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<tr>
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<th>HW, n=15</th>
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<tbody>
<tr>
<td>Average total steps (steps/day)</td>
<td>8643.6 ± 3430.9</td>
<td>5028.5 ± 2584.6 (n=11)</td>
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<tr>
<td>Average weekday steps (steps/day)</td>
<td>8239.9 ± 3381.3</td>
<td>5155.7 ± 3085.4 (n=11)</td>
</tr>
<tr>
<td>Average weekend steps (steps/day)</td>
<td>9128.0 ± 6663.2</td>
<td>4774.2 ± 3065.8 (n=11)</td>
</tr>
<tr>
<td>Average time spent in MVPA (min/day)</td>
<td>27.7 ± 18.5</td>
<td>14.0 ± 14.3 (n=11)</td>
</tr>
<tr>
<td>Average time spent in MVPA on weekday (min/day)</td>
<td>26.4 ± 19.8</td>
<td>16.2 ± 17.0 (n=11)</td>
</tr>
<tr>
<td>Average time spent in MVPA on weekend (min/day)</td>
<td>30.3 ± 32.1</td>
<td>9.7 ± 11.5 (n=11)</td>
</tr>
</tbody>
</table>
Relationships among variables of interest

Table 12. Correlations among primary variables in the total group, n=60 (RHI, n=58)

<table>
<thead>
<tr>
<th></th>
<th>AbsVO₂</th>
<th>RelVO₂</th>
<th>AbsHRRec</th>
<th>RelHRRec</th>
<th>RHI</th>
</tr>
</thead>
<tbody>
<tr>
<td>AbsVO₂</td>
<td>1</td>
<td>-0.031</td>
<td>-0.497**</td>
<td>-0.511**</td>
<td>0.443**</td>
</tr>
<tr>
<td>RelVO₂</td>
<td>-0.031</td>
<td>1</td>
<td>0.241</td>
<td>0.163</td>
<td>-0.269*</td>
</tr>
<tr>
<td>AbsHRRec</td>
<td>-0.497**</td>
<td>0.214</td>
<td>1</td>
<td>0.992**</td>
<td>-0.459**</td>
</tr>
<tr>
<td>RelHRRec</td>
<td>-0.511**</td>
<td>0.163</td>
<td>0.992**</td>
<td>1</td>
<td>-0.436**</td>
</tr>
<tr>
<td>RHI</td>
<td>0.443**</td>
<td>-0.269*</td>
<td>-0.459**</td>
<td>-0.436**</td>
<td>1</td>
</tr>
</tbody>
</table>

*p<0.05  
**p<0.01

**Total group.**

When all subjects were analyzed together there were significant correlations between relative VO₂ peak and RHI (r = -0.269; Figure 1). Absolute VO₂ peak had significant negative correlations with absolute heart rate recovery (r = -0.497; Figure 2) and relative heart rate recovery (r = -0.511; Figure 3) and was positively correlated with RHI (r = 0.443; Figure 4). Absolute and relative heart rate recovery values were significantly correlated (r = 0.992; Figure 5). Additionally, RHI was inversely related with absolute heart rate recovery (r = -0.459; Figure 6) and relative heart rate recovery (r = -0.436; Figure 7). Other interesting significant correlations existed in this group. Absolute VO₂ peak was correlated with BMI (n = 60, r = 0.459, p < 0.01); waist circumference (n = 59, r = 0.446, p < 0.01); resting heart rate (n = 60, r = -0.483, p < 0.01); absolute oxygen recovery (n = 57, r = -0.332, p < 0.05); and relative oxygen recovery (n = 57, r = -0.399, p < 0.01). Relative VO₂ peak was also correlated with BMI (n = 60, r = -0.767, p < 0.01); waist circumference (n = 59, r = -0.767, p < 0.01); maximal heart rate (n = 60, r = 0.516, p < 0.01); absolute oxygen recovery (n = 57, r = 0.368, p < 0.01); time on treadmill (n = 60, r = 0.776, p < 0.01); average total steps (n = 56, r = 0.579, p < 0.01) and average time spent in MVPA (n = 56, r = 0.579, p < 0.01). Absolute heart rate recovery was
Figure 1. Correlation between Relative VO$_2$ Peak and RHI

Figure 2. Correlation between Absolute VO$_2$ Peak and Absolute Heart Rate Recovery
Figure 3. Correlation between Absolute VO\textsubscript{2} Peak and Relative Heart Rate Recovery

Figure 4. Correlation between Absolute VO\textsubscript{2} Peak and RHI
Figure 5. Correlation between Absolute Heart Rate Recovery and Relative Heart Rate Recovery

Figure 6. Correlation between Absolute Heart Rate Recovery and RHI
Figure 7. Correlation between Relative Heart Rate Recovery and RHI
correlated to BMI (n = 60, r = -0.347, p < 0.01); waist circumference (n = 59, r = -0.317, p < 0.05); maximal heart rate (n = 60, r = 0.579, p < 0.01); average total steps (n = 56, r = 0.319, p < 0.05); and average time spent in MVPA (n = 56, r = -0.366, p < 0.01).

Relative heart rate recovery was significantly correlated to BMI (n = 60, r = 0.318, p < 0.05); waist circumference (n = 59, r = -0.292, p < 0.05); maximal heart rate (n = 60, r = 0.482, p < 0.01); average total steps (n = 56, r = 0.299, p < 0.05); and average time spent in MVPA (n = 56, r = 0.349, p < 0.01). RHI was correlated to BMI (n = 58, r = 0.272, p < 0.05), resting heart rate (n = 58, r = -0.352, p < 0.01), maximal heart rate (n = 58, r = -0.445, p < 0.01); absolute oxygen recovery (n = 55, r = -0.360, p < 0.01); relative oxygen recovery (n = 55, r = -0.309, p < 0.05); and average time spent in MVPA (n = 54, r = -0.329, p < 0.05).

**Table 13. Correlations among primary variables in all women, n=27 (RHI, n=26)**

<table>
<thead>
<tr>
<th></th>
<th>AbsVO₂</th>
<th>RelVO₂</th>
<th>AbsHRRRec</th>
<th>RelHRRRec</th>
<th>RHI</th>
</tr>
</thead>
<tbody>
<tr>
<td>AbsVO₂</td>
<td>1</td>
<td>0.435*</td>
<td>-0.159</td>
<td>-0.201</td>
<td>-0.018</td>
</tr>
<tr>
<td>RelVO₂</td>
<td>0.435*</td>
<td>1</td>
<td>0.048</td>
<td>0.002</td>
<td>0.213</td>
</tr>
<tr>
<td>AbsHRRRec</td>
<td>-0.159</td>
<td>0.048</td>
<td>1</td>
<td>0.994**</td>
<td>-0.036</td>
</tr>
<tr>
<td>RelHRRRec</td>
<td>-0.201</td>
<td>0.002</td>
<td>0.994**</td>
<td>1</td>
<td>-0.042</td>
</tr>
<tr>
<td>RHI</td>
<td>-0.018</td>
<td>0.213</td>
<td>-0.036</td>
<td>-0.042</td>
<td>1</td>
</tr>
</tbody>
</table>

*p<0.05
**p<0.01

**All women.**

In all women, the significant correlations among the primary variables were between relative VO₂ peak and absolute VO₂ peak (r = 0.435) and absolute and relative heart rate recovery (r= 0.994). Other valuable correlations were between relative VO₂ peak and BMI (n = 27, r = -0.779, p < 0.01), waist circumference (n = 27, r = -0.794, p < 0.01); resting heart rate (n = 27, r = -0.612, p < 0.01); maximal heart rate (n = 27, r = 0.400, p < 0.05); time on treadmill (n = 27, r = 0.925, p < 0.01); average total steps (n = 26, r = 0.511, p < 0.01); and average time spent in MVPA (n = 26, r = 0.487, p < 0.05).
Absolute heart rate recovery was significantly correlated to maximal heart rate (n = 27, r = 0.395, p < 0.05).

**Table 14. Relationships among primary variables in all children, n=33 (RHI, n=32)**

<table>
<thead>
<tr>
<th></th>
<th>AbsVO₂</th>
<th>RelVO₂</th>
<th>AbsHRRec</th>
<th>RelHRRec</th>
<th>RHI</th>
</tr>
</thead>
<tbody>
<tr>
<td>AbsVO₂</td>
<td>1</td>
<td>0.311</td>
<td>-0.202</td>
<td>-0.247</td>
<td>0.040</td>
</tr>
<tr>
<td>RelVO₂</td>
<td>0.311</td>
<td>1</td>
<td>-0.203</td>
<td>-0.263</td>
<td>0.009</td>
</tr>
<tr>
<td>AbsHRRec</td>
<td>-0.202</td>
<td>-0.0203</td>
<td>1</td>
<td>0.985**</td>
<td>0.160</td>
</tr>
<tr>
<td>RelHRRec</td>
<td>-0.247</td>
<td>-0.263</td>
<td>0.985**</td>
<td>1</td>
<td>0.193</td>
</tr>
<tr>
<td>RHI</td>
<td>0.040</td>
<td>0.009</td>
<td>0.160</td>
<td>0.193</td>
<td>1</td>
</tr>
</tbody>
</table>

*p<0.05

**All children.**

In all children, the only significant correlation among the primary variables of interest was between absolute and relative heart rate recovery (r = 0.985). Other correlations of interest existed between absolute VO₂ peak and waist circumference (n = 33, r = 0.392, p < 0.05); and resting heart rate (n = 33, r = -0.440, p < 0.05). Relative VO₂ peak was significantly correlated to BMI (n = 33, r = -0.676, p < 0.01); waist circumference (n = 33, r = -0.674, p < 0.01); relative oxygen recovery (n = 32, r = -0.461, p < 0.01); time on treadmill (n = 33, r = 0.738, p < 0.01); average total steps (n = 30, r = 0.451, p < 0.05), and average time spent in MVPA (n = 30,r = 0.465, p ≤ 0.01). RHI was not significantly correlated to any variables.
Table 15. Correlations among primary variables in all healthy-weight, n=35 (RHI, n=33)

<table>
<thead>
<tr>
<th></th>
<th>AbsVO₂</th>
<th>RelVO₂</th>
<th>AbsHRRec</th>
<th>RelHRRec</th>
<th>RHI</th>
</tr>
</thead>
<tbody>
<tr>
<td>AbsVO₂</td>
<td>1</td>
<td>0.159</td>
<td>-0.507**</td>
<td>-0.498**</td>
<td>-0.457**</td>
</tr>
<tr>
<td>RelVO₂</td>
<td>0.159</td>
<td>1</td>
<td>0.187</td>
<td>0.133</td>
<td>-0.446**</td>
</tr>
<tr>
<td>AbsHRRec</td>
<td>-0.507**</td>
<td>0.187</td>
<td>1</td>
<td>0.991*</td>
<td>-0.436*</td>
</tr>
<tr>
<td>RelHRRec</td>
<td>-0.498**</td>
<td>0.133</td>
<td>0.991*</td>
<td>1</td>
<td>-0.396*</td>
</tr>
<tr>
<td>RHI</td>
<td>0.457**</td>
<td>-0.446**</td>
<td>-0.436*</td>
<td>-0.396*</td>
<td>1</td>
</tr>
</tbody>
</table>

*p<0.05
**p<0.01

All healthy-weight subjects.

There were several significant correlations among variables of interest in all HW subjects. Absolute VO₂ peak was significantly correlated to absolute heart rate recovery (r = -0.507), relative heart rate recovery (r = -0.498), and RHI (r = 0.457). Relative VO₂ peak was significantly correlated to RHI (r = -0.446). Absolute heart rate recovery was significantly correlated to relative heart rate recovery (r = 0.991) and RHI (r = -0.436). Finally, relative heart rate recovery was significantly correlated to RHI (r = -0.396). Other interesting correlations existed between absolute VO₂ peak and BMI (n = 35, r = 0.707, p < 0.01); waist circumference (n = 35, r = 0.598, p < 0.01), and resting heart rate (n = 35, r = -0.511, p < 0.01). Relative VO₂ peak was significantly correlated to BMI (n = 35, r = -0.486, p < 0.01); waist circumference (n = 35, r = -0.501, p < 0.01); maximal heart rate (n = 35, r = 0.422, p < 0.05); time on treadmill (n = 35, r = 0.591, p < 0.01); average total steps (n = 34, r = 0.491, p < 0.01); and average time spent in MVPA (n = 34, r = 0.608, p < 0.01). Absolute heart rate recovery was significantly correlated with BMI (n = 35, r = -0.489, p < 0.01); waist circumference (n = 35, r = -0.459, p < 0.01); maximal heart rate (n = 35, r = 0.485, p < 0.01); and average time spent in MVPA (n = 34, r = 0.373, p < 0.05). Relative heart rate recovery was significantly correlated to BMI (n = 35, r = -
0.454, p < 0.01); waist circumference (n = 35, r = -0.432, p < 0.05); and maximal heart rate (n = 35, r = 0.367, p < 0.05). RHI was significantly correlated to BMI (n = 33, r = 0.686, p < 0.01); waist circumference (n = 35, r = 0.545, p < 0.01), resting heart rate (n = 35, r = -0.382, p < 0.05), maximal heart rate (n = 35, r = -0.467, p < 0.01), absolute oxygen recovery (n = 31, r = -0.486, p < 0.01), and relative oxygen recovery (n = 31, r = -0.457, p < 0.05).

Table 16. Correlations among primary variables in all obese, n=25

<table>
<thead>
<tr>
<th></th>
<th>AbsVO₂</th>
<th>RelVO₂</th>
<th>AbsHRRec</th>
<th>RelHRRec</th>
<th>RHI</th>
</tr>
</thead>
<tbody>
<tr>
<td>AbsVO₂</td>
<td>1</td>
<td>-0.086</td>
<td>-0.473*</td>
<td>-0.522**</td>
<td>0.497*</td>
</tr>
<tr>
<td>RelVO₂</td>
<td>-0.086</td>
<td>1</td>
<td>0.275</td>
<td>0.252</td>
<td>0.519**</td>
</tr>
<tr>
<td>AbsHRRec</td>
<td>-0.473*</td>
<td>0.275</td>
<td>1</td>
<td>0.995**</td>
<td>-0.557**</td>
</tr>
<tr>
<td>RelHRRec</td>
<td>-0.522**</td>
<td>0.252</td>
<td>0.995**</td>
<td>1</td>
<td>-0.552**</td>
</tr>
<tr>
<td>RHI</td>
<td>0.497*</td>
<td>-0.519*</td>
<td>-0.557**</td>
<td>-0.552**</td>
<td>1</td>
</tr>
</tbody>
</table>

*p<0.05  
**p<0.01

All obese subjects.

In all OB subjects, there were significant correlations between absolute VO₂ peak and absolute heart rate recovery (r = -0.473), relative heart rate recovery (r = -0.522), and RHI (r = 0.497). Relative VO₂ peak was significantly correlated to RHI (r = -0.519). Absolute heart rate recovery was significantly correlated with relative heart rate recovery (r = 0.995) and RHI (r = 0.557). Finally, relative heart rate recovery was significantly correlated to RHI (r = -0.552). Other valuable correlations existed among other variables. Absolute VO₂ peak was also correlated to BMI (n = 25, r = 0.581, p < 0.01); waist circumference (n = 24, r = 0.521, p < 0.01); resting heart rate (n = 25, r = -0.548, p < 0.01); absolute oxygen recovery (n = 24, r = -0.506, p < 0.05); relative oxygen recovery (n = 24, r = -0.574, p < 0.01); average total steps (n = 22, r = -0.507, p < 0.05), and average time spent in MVPA (n = 22, r = -0.488, p<0.05). Relative VO₂ peak was
significantly correlated to BMI (n = 25, r = -0.723, p < 0.01); waist circumference (n = 25, r = -0.764, p < 0.01); maximal heart rate (n = 25; r = 0.503, p < 0.05); time on treadmill (n = 25, r = 0.576, p < 0.01); average total steps (n = 22, r = 0.505, p < 0.05); and average time spent in MVPA (n = 22, r = 0.451, p < 0.05). Absolute heart rate recovery was correlated to BMI (n = 25, r = -0.483, p < 0.05); maximal heart rate (n = 25, r = 0.756, p < 0.01); and relative oxygen recovery (n = 24, r = 0.415, p < 0.05). Relative heart rate recovery was significantly correlated with BMI (n = 25, r = -0.486, p < 0.05), waist circumference (n = 24, r = -0.411, p < 0.05); maximal heart rate (n = 25, r = 0.699, p < 0.01); absolute oxygen recovery (n = 24, r = 0.413, p < 0.05); and relative oxygen recovery (n = 24, r = 0.437, p < 0.05). Finally, RHI was also correlated to BMI (n = 25, r = 0.675, p < 0.01); waist circumference (n = 24, r = 0.606, p < 0.01); maximal heart rate (n = 25, r = -0.583, p < 0.01), average total steps (n = 22, r = -0.589, p < 0.01); and average time spent in MVPA (n = 22, r = -0.611, p < 0.01)

Table 17. Correlations among primary variables in healthy-weight women, n=15, (RHI, n=14)

<table>
<thead>
<tr>
<th></th>
<th>AbsVO₂</th>
<th>RelVO₂</th>
<th>AbsHRRec</th>
<th>RelHRRec</th>
<th>RHI</th>
</tr>
</thead>
<tbody>
<tr>
<td>AbsVO₂</td>
<td>1</td>
<td>0.876**</td>
<td>-0.044</td>
<td>-0.092</td>
<td>0.048</td>
</tr>
<tr>
<td>RelVO₂</td>
<td>0.876**</td>
<td>1</td>
<td>-0.104</td>
<td>-0.141</td>
<td>-0.001</td>
</tr>
<tr>
<td>AbsHRRec</td>
<td>-0.044</td>
<td>-0.104</td>
<td>1</td>
<td>0.992**</td>
<td>-0.110</td>
</tr>
<tr>
<td>RelHRRec</td>
<td>-0.092</td>
<td>-0.141</td>
<td>0.992**</td>
<td>1</td>
<td>-0.103</td>
</tr>
<tr>
<td>RHI</td>
<td>0.048</td>
<td>-0.001</td>
<td>-0.110</td>
<td>-0.103</td>
<td>1</td>
</tr>
</tbody>
</table>

*p<0.05  
**p<0.01  

Healthy-weight women.

In HW women, the only significant correlations among the primary variables were between absolute and relative VO₂ peak (r = 0.876) and between absolute and relative heart recovery (r = 0.992). In addition, Absolute VO₂ peak was significantly correlated with absolute oxygen recovery (n = 14, r = 0.781, p < 0.01). Relative VO₂ peak was
significantly correlated to absolute oxygen recovery \( (n = 14, r = 0.721, p < 0.01) \) and time on treadmill \( (n = 15, r = 0.753, p < 0.01) \).

**Table 18. Correlations among primary variables in healthy-weight children, n=20 (RHI, n=19)**

<table>
<thead>
<tr>
<th></th>
<th>AbsVO₂</th>
<th>RelVO₂</th>
<th>AbsHRRec</th>
<th>RelHRRec</th>
<th>RHI</th>
</tr>
</thead>
<tbody>
<tr>
<td>AbsVO₂</td>
<td>1</td>
<td>0.714**</td>
<td>-0.141</td>
<td>-0.411</td>
<td>0.010</td>
</tr>
<tr>
<td>RelVO₂</td>
<td>0.714**</td>
<td>1</td>
<td>-0.260</td>
<td>-0.260</td>
<td>-0.068</td>
</tr>
<tr>
<td>AbsHRRec</td>
<td>-0.141</td>
<td>-0.219</td>
<td>1</td>
<td>0.986**</td>
<td>0.112</td>
</tr>
<tr>
<td>RelHRRec</td>
<td>-0.411</td>
<td>-0.260</td>
<td>0.986**</td>
<td>1</td>
<td>0.159</td>
</tr>
<tr>
<td>RHI</td>
<td>0.010</td>
<td>-0.068</td>
<td>0.112</td>
<td>0.159</td>
<td>1</td>
</tr>
</tbody>
</table>

*p<0.05
**p<0.01

**Healthy-weight children.**

In HW children, the only significant correlations among the primary variables were between absolute and relative VO₂ peak \( (r = 0.714) \) and between absolute and relative heart recovery \( (r = 0.986) \). Additionally, absolute VO₂ peak was significantly correlated to resting heart rate \( (n = 20, r = -0.458, p < 0.05) \); average total steps \( (n = 19, r = 0.539, p < 0.05) \) and average time spent in MVPA \( (n = 19, r = 0.532, p < 0.05) \). There were significant correlations between relative VO₂ peak and relative oxygen recovery \( (n = 19, r = -0.535, p < 0.05) \); time on treadmill \( (n = 20; r = 0.626, p < 0.01) \); average total steps \( (n = 19, r = 0.542, p < 0.05) \); and average time spent in MVPA \( (n = 19, r = 0.563, p < 0.05) \).
Table 19. Correlations among primary variables in obese women, n=12

<table>
<thead>
<tr>
<th></th>
<th>AbsVO₂</th>
<th>RelVO₂</th>
<th>AbsHRRec</th>
<th>RelHRRec</th>
<th>RHI</th>
</tr>
</thead>
<tbody>
<tr>
<td>AbsVO₂</td>
<td>1</td>
<td>0.808**</td>
<td>-0.264</td>
<td>-0.302</td>
<td>0.019</td>
</tr>
<tr>
<td>RelVO₂</td>
<td>0.808**</td>
<td>1</td>
<td>-0.333</td>
<td>-0.367</td>
<td>-0.090</td>
</tr>
<tr>
<td>AbsHRRec</td>
<td>-0.264</td>
<td>-0.333</td>
<td>1</td>
<td>0.998**</td>
<td>-0.107</td>
</tr>
<tr>
<td>RelHRRec</td>
<td>-0.302</td>
<td>-0.367</td>
<td>0.998**</td>
<td>1</td>
<td>-0.110</td>
</tr>
<tr>
<td>RHI</td>
<td>0.019</td>
<td>-0.090</td>
<td>-0.107</td>
<td>-0.110</td>
<td>1</td>
</tr>
</tbody>
</table>

*p<0.05
**p<0.01

Obese women.

In OB women, the only significant correlations among the primary variables were between absolute and relative VO₂ peak (r = 0.808) and between absolute and relative heart recovery (r = 0.998). Additionally, absolute VO₂ peak was related to time on treadmill (n = 12, r = 0.671, p < 0.05). Relative VO₂ peak was significantly correlated to BMI (n = 12, r = -0.636, p < 0.05), waist circumference (n = 12, r = -0.818, p < 0.01), resting heart rate (n = 12, r = -0.792, p < 0.01) and time on treadmill (n = 12, r = 0.908, p < 0.01). RHI was significantly correlated with relative oxygen recovery (n = 11, r = 0.641, p < 0.05).

Table 20. Correlations among primary variables in obese children, n=13

<table>
<thead>
<tr>
<th></th>
<th>AbsVO₂</th>
<th>RelVO₂</th>
<th>AbsHRRec</th>
<th>RelHRRec</th>
<th>RHI</th>
</tr>
</thead>
<tbody>
<tr>
<td>AbsVO₂</td>
<td>1</td>
<td>0.230</td>
<td>0.130</td>
<td>0.002</td>
<td>0.131</td>
</tr>
<tr>
<td>RelVO₂</td>
<td>0.230</td>
<td>1</td>
<td>-0.330</td>
<td>-0.342</td>
<td>-0.132</td>
</tr>
<tr>
<td>AbsHRRec</td>
<td>0.130</td>
<td>-0.330</td>
<td>1</td>
<td>0.988**</td>
<td>0.303</td>
</tr>
<tr>
<td>RelHRRec</td>
<td>0.002</td>
<td>-0.342</td>
<td>0.988**</td>
<td>1</td>
<td>0.358</td>
</tr>
<tr>
<td>RHI</td>
<td>0.131</td>
<td>-0.132</td>
<td>0.330</td>
<td>0.358</td>
<td>1</td>
</tr>
</tbody>
</table>

*p<0.05
**p<0.01
Obese children.

In OB children, the only significant correlation was between absolute heart rate recovery and relative heart rate recovery ($r = 0.988$). Additionally, absolute VO$_2$ peak was significantly correlated BMI ($n = 13$, $r = 0.597$, $p < 0.05$); waist circumference ($n = 13$, $r = 0.755$, $p < 0.01$); maximal heart rate ($n = 13$, $r = 0.742$, $p < 0.01$). Relative VO$_2$ peak and absolute oxygen recovery were significantly correlated ($n = 13$, $r = 0.706$, $p < 0.01$).

Absolute heart rate recovery was significantly correlated to maximal heart rate ($n = 13$, $r = 0.561$, $p < 0.05$). Finally, RHI was significantly correlated to time on treadmill ($n = 13$, $r = 0.767$, $p < 0.01$).
Chapter 5: Discussion

There were no significant differences in absolute VO$_2$ peak (L/min) between healthy-weight and obese groups. This result is similar to previous findings comparing healthy-weight and obese populations of both children and adults (36,67). This is likely because obese individuals have equal or greater amounts of fat-free mass which is the oxygen-consuming tissue. Goran et al. (36) examined aerobic capacity before and after weight loss in 31 overweight women. After the weight loss, absolute VO$_2$ peak decreased from $2.16 \pm 0.27$ L/min to $2.08 \pm 0.33$ L/min. Goran et al. (36) also reported absolute VO$_2$ peak results comparing lean and obese children. Lean children had a lower absolute VO$_2$ peak ($1.24 \pm 0.27$ L/min) compared to obese children ($1.56 \pm 0.40$ L/min). The obese children in this study had a significantly higher fat-free mass ($26.4 \pm 5.3$ kg) compared to the lean children ($21.4 \pm 3.9$ kg). While fat-free mass was not compared statistically in the present study, the healthy-weight group had a lower mean fat-free mass ($34.6 \pm 9.7$ kg) compared to the obese group ($39.6 \pm 11.1$ kg). Perhaps, the small sample size combined with the large range of values for fat-free mass is the reason that absolute VO$_2$ peak was not significantly different across adiposity groups.

The adults had a significantly greater absolute VO$_2$ peak (L/min) compared to the children and this is due in part to the higher body mass ($79.9 \pm 22.1$ kg) and subsequently higher fat-free mass ($46.6 \pm 5.1$ kg) in the adults compared to the children ($42.7 \pm 14.4$ kg and $28.6 \pm 5.8$ kg) (3).

As expected, there were significant differences in relative VO$_2$ peak (ml/kg/min) between healthy-weight and obese groups. This finding has been consistently reported (36,67,112). Loftin et al. (67) reported that obese girls had significantly lower relative VO$_2$ peaks ($22.8 \pm 7.3$ ml/kg/min) compared to normal-weight girls ($45.8 \pm 7.2$ ml/kg/min). Similarly, Goran et al., (36) found significantly lower relative VO$_2$ peaks in obese children ($32.0 \pm 4.1$ ml/kg/min) compared to lean children ($44.2 \pm 3.2$ ml/kg/min).
Lower peak oxygen consumption measures are typically reported in obese individuals because they are impeded as a result of carrying excess body fat during weight-bearing activities.

In this study, there were also significant differences across age groups in relative VO2 peak. Typically, similar values between adults and children are reported in the literature (7,115). Welsman et al., (115) examined the effects of maturation on VO2 peak in untrained subjects. The authors reported similar values for relative VO2 peak across prepubertal (50 ± 4 ml/kg/min), circumpubertal (53 ± 4 ml/kg/min) and adult (53 ± 3 ml/kg/min) groups in males. In female groups, the circumpubertal group had a significantly higher relative VO2 peak (47 ± 4 ml/kg/min) compared to prepubertal (45 ± 3 ml/kg/min) and adult females (43 ± 3 ml/kg/min). We did not control for level of fitness and had a wide range of fitness levels in the women and children regardless of adiposity level. The mean age for the women included in the study by Welsman et al. (115) was younger (21.7 ± 2.8 yr) compared to the women in the present study (34.7 ± 6.5 yr), which may account for the lower reported VO2 peak values in the adults in the present study.

In this study, there were no differences in absolute or relative heart rate recovery between adiposity groups despite significant differences in fitness levels. In adult populations, heart rate recovery has been used as a clinical indicator of morbidity and mortality risks (19,21,113). Cole et al. (21) investigated the predictive power of an abnormal heart rate recovery. During the follow up period of six years, 56% of the subjects that died had an abnormal HRRec. Subjects that had an abnormal HRRec were significantly older and had a significantly greater resting heart rate. Furthermore, a greater percentage of subjects with abnormal HRRec were hypertensive, diabetic, and/or were smokers (21). Similarly, Watanabe et al. (113) found that even when
controlling for confounding variables (age, sex, blood pressure, resting heart rate, medications, etc.) abnormal HRRec was still a strong independent predictor of death.

Additionally, studies have cited differences in heart rate recovery in healthy-weight and obese populations (19,21,30,103). Eagle et al. (30) reported a greater heart rate during recovery from a 3 minute step test (indicating a slower HRRec) in obese children (117.94 ± 19.76 bpm) compared to non-obese children (100.84 ± 15.94 bpm). In adults, a lower exercise capacity has been associated with attenuated HRRec (21). These authors reported that exercise capacity in men with an abnormal HRRec was lower (7.9 MET) than men with normal HRRec (9.8 MET). Similarly, women with abnormal HRRec had a lower exercise capacity (6.0 MET) compared to women with a normal HRRec (7.4 MET). Cheng et al. (19) also reported lower exercise tolerance with attenuated HRRec. The men in this study were divided into quartiles based on their HRRec during the 5 minutes following maximal exercise (<55 bpm, 55-66 bpm, 67-75 bpm, and >75 bpm). Subjects in the lowest quartile had a significantly lower exercise capacity (8.60 ± 1.7 MET) compared to the highest quartile (11.5 ± 1.9 MET). Despite lower fitness levels of the obese cohort in the present study, the mean absolute and relative heart rate recovery for obese women and obese children was not significantly different compared to healthy-weight women and healthy-weight children. These findings are in contrast to previous reports demonstrating that low fitness levels and poor body composition negatively impact heart rate recovery in adults and children (19,21,22,30,103).

Presence of the metabolic syndrome may be related to deleterious effects on cardiorespiratory fitness in obese individuals (26,51,108). The criteria for the metabolic syndrome include central abdominal obesity, impaired fasting glucose, hypertension, hypertriglyceridemia, and reduced HDL-cholesterol levels (79). Deniz et al. (26) investigated heart rate recovery in obese men (24 ± 3 yrs) with and without the
metabolic syndrome. The authors reported that men with the metabolic syndrome had slower heart rate recovery values following a maximal GXT compared to the obese group without metabolic syndrome. All subjects in the study were considered sedentary, therefore the authors contributed the slower recovery to disturbances associated with the metabolic syndrome, such as increased sympathetic activity, and not cardiovascular fitness level or obesity status. However, it should be noted that the men in the metabolic syndrome group had a significantly greater BMI compared to the control group (38.6 ± 3.68 kg/m² vs. 32.22 ± 2.99 kg/m²), exercised for a shorter period of time (6.23 ± 1.53 minutes vs. 7.20 ± 1.26 minutes) and had a lower exercise capacity (9.15 ± 1.73 METs vs. 10.45 ± 2.85 METs). This suggested that the obesity in the metabolic syndrome group was more severe and they had a lower fitness level (26). Similarly, Sung et al. (108) reported a slower heart rate recovery (10.3 ± 11.6 bpm) in adults with the metabolic syndrome compared to a control group (13.6 ± 9.7 bpm). These authors also reported that heart rate recovery became increasingly slower as the number of metabolic syndrome criteria increased. Additionally, Kizilbash et al. (51) reported that heart rate recovery was slowest in individuals who had 2 or more criteria for the metabolic syndrome. These authors stated that the reduction in heart rate recovery is the result of the metabolic syndrome and does not occur prior to accumulation of the components of the metabolic syndrome (51).

The obese subjects in the present study were screened for other health risks. No subjects with known diabetes or hypertension (or taking antihypertensive medications) were included in the study. However, blood samples were not collected from these subjects so it is impossible to know whether any of these subjects had increased fasting glucose levels, hyperinsulinemia, or dyslipidemia. No subjects reported taking medications to control cholesterol. Therefore, it seems as though our obese cohort was obese, yet otherwise healthy. Perhaps heart rate recovery was not negatively affected
in the obese subjects as a result of little metabolic disturbance in this cohort. If such a risk threshold exists, further research in the obese population is necessary to determine the cutoff point for this variable.

There were significant differences between the heart rate recovery values of the adults and children. Following a maximal graded exercise test, children recovered significantly faster than adults in both absolute and relative terms. This is in agreement with previously published reports (7,15). Baraldi et al. (7) examined HRRec in adults and children at varying levels of cycling exercise. As expected, as the intensity increased, both adults and children spent more time recovering however, children returned to their baseline heart rate values faster than adults. Buchheit et al., (15) also reported more rapid HRRec in children (50 ± 1 bpm) when compared to adolescents (37 ± 1 bpm) and adults (39 ± 1 bpm) following 10 bouts of cycle sprint exercise. The authors suggested that the faster recovery may be related in part to the lower power output produced by children and the lower levels of lactate and acidosis (15). Similarly Falk et al. (31) and Rowland (94) have suggested that faster recovery in children is the result of lower levels of lactate production due to lower anaerobic thresholds, shorter circulation times, and smaller amounts of fast twitch fibers.

Endothelial function was not significantly different across adiposity groups in adults or children. This result was unexpected, yet may be in part due to the fact that obese but otherwise healthy adults and children were recruited for this study. Diabetes and impaired fasting glucose are major contributors to endothelial dysfunction in both adults and children (39,72,76,89). While the differences between the healthy-weight and obese women were not statistically different, it may be considered practically significant. According to the manufacturer's website, a reactive hyperemia index between 1.68 and 2 is not associated with increased health risks, however without continuous healthy
lifestyle behaviors endothelial dysfunction is still a possibility. A reactive hyperemia score greater than 2 suggests that endothelial function is optimal (48).

The difference between women and children in reactive hyperemia scores was expected. Prepubescent children are known to have lower reactive hyperemia scores due to immature vasculature (88). The overall mean value for RHI in children in the present study is similar to what is reported in the literature for children of this age (88). Radtke et al. (88) listed the lack of inclusion of obese children in their study to be a limitation of their study. While obese children were included in the present study, lack of variance among all the children prevented statistical analysis. The lack of variance suggests that the immature vasculature that younger children have may provide a certain level of protection from the deleterious effects on endothelial function seen in adulthood. Mahmud et al. (72) examined endothelial function in obese adolescents (13.4 ± 1.7 yr) and their non-obese counterparts (14.0 ± 1.4 yr). The authors reported a significantly lower RHI in the obese group (1.51 ± 0.4) compared to the non-obese adolescents (2.06 ± 0.4). Mahmud et al., (72) also found an increased in RHI with increasing age similar to other published reports (86). In the study by Mahmud et al. (70), all children were at least Tanner Stage 2, whereas in the current study, we attempted to only include children that were prepubescent. However, a limitation of the current study is that we were unable to determine Tanner Staging for all children in the study. Finally, it is important to note, that the Endo-PAT 2000 is not the gold standard of noninvasively evaluating endothelial function in adults and children. Many studies utilize brachial artery ultrasonography as noninvasive measure of endothelial function (50,89,100,121). Use of the Endo-PAT 2000 has been limited and more research is needed to further establish this technique. However, Nohria et al. (80) demonstrated the validity of the Endo-PAT 2000 and the authors reported that this operator-independent method of evaluating nitric oxide availability may be a simple way to test endothelial
function. While the Endo-PAT 2000 may be unable to differentiate impairment of RHI in prepubescent children, Woo et al. (1212) was able to determine that obese children (Tanner Stage 2 or less) demonstrated a reduced vasodilation response utilizing FMD. This suggests that the Endo-PAT 2000 may not be appropriate for use in prepubescent children.

Absolute heart rate recovery was not significantly correlated with relative VO$_2$ peak (ml/kg/min) in the total group, all women, all children, all healthy-weight, all obese, healthy-weight women, healthy-weight children, obese women, or obese children. This is similar to the findings of Campos et al. (16) who reported no significant correlations between relative VO$_2$ peak (ml/kg/min) and 1 minute heart rate recovery ($n = 14$, $r = 0.160$, $p = 0.69$) in professional cyclists. While Sung et al. (108) reported that the correlation between heart rate recovery and VO$_2$ peak was weak; the authors speculated that this was due to the small range of VO$_2$ peak values in their sample of men and women. These results are somewhat unexpected because high fitness levels have been associated with greater parasympathetic control. This is important because a decrease in parasympathetic tone is associated with slower heart rate recovery (47).

In the present study, absolute and relative heart rate recovery values were significantly correlated with absolute VO$_2$ peak in the total group. These negative correlations demonstrated that as body weight increased, absolute and relative heart rate recovery decreased. This result is expected because adults had a greater body mass and subsequently greater fat-free mass (the oxygen consuming tissue) and slower HRRec. In the total group, absolute VO$_2$ peak was also significantly correlated to RHI. As absolute VO$_2$ peak increased, RHI increased as well, which was expected since the adults tended to have greater absolute VO$_2$ peaks and greater RHI compared to children. Furthermore, absolute and relative heart rate recovery were both significantly correlated to BMI and waist circumference (only significantly correlated to relative heart
rate recovery in obese) in the total group, the healthy-weight group, and the obese group in the present study, which is similar to previous reports (28, 63, 103). RHI was significantly correlated to absolute and relative heart rate recovery. These results are similar to the findings of Huang et al. (47) where it was reported that heart rate recovery was an independent predictor of endothelial function (measured by FMD).

There are several limitations of the current study. First of all, we did not control for certain aspects of testing, including time of day, fasting, fitness level of our subjects, or menstrual cycles of women. While we did not control for time of day for testing, a large majority of the subjects were tested in the afternoon and early evening. Many studies investigate endothelial function under fasting conditions (38,43,76,77,109). Kuvin et al. (55) used the Endo-PAT 2000 in an ambulatory setting to examine the effectiveness of this tool under non-fasting conditions. Kuvin et al. (55) reported that even without controlling for a fasted state the Endo-PAT 2000 still proved to be a useful tool as it was still able to detect differences in between individuals with and without coronary artery disease. Furthermore, it has been reported that there is no acute endothelial dysfunction in obese children as a result of an oral glucose challenge (75). Previous studies have demonstrated that aerobic capacity and endothelial function may vary based on phase of the menstrual cycle. Lebrun et al. (59) examined the effect of menstrual cycle phase on athletic performance in trained athletes (VO₂ max > 50 ml/kg/min). The authors found a lower absolute (p < 0.04) and relative (p < 0.06) VO₂ max during the luteal phase of the menstrual cycle compared to the follicular phase. Hashimoto et al. (41) investigated variation in FMD during different phases of the menstrual cycle. The authors reported an increase in percent FMD during the follicular and luteal phase compared to the menstrual phase. Hashimoto et al. (41) concluded that changes in estradiol concentration are associated with variations in endothelial function during the menstrual cycle. As previously stated, we did not include Tanner
Staging in our study, which would have provided a more complete description of the maturational status our pediatric population. Finally, we had a small sample size relative to the number of variables we were interested in examining. While our study was designed to provide adequate power to detected differences in cardiorespiratory fitness, perhaps a larger sample size would have result in differences RHI or obesity-related differences in heart rate recovery.

In summary, there were significant obesity- and age-related differences in fitness levels; however heart rate recovery was only impacted by age. There were no obesity-related differences in RHI in the group of women included in the present study. VO₂ peak, heart rate recovery, and RHI were significantly related and perhaps can be used to provide a complete profile of cardiorespiratory health and fitness. The primary finding of this study was that obesity without associated comorbidities does not affect indicators of cardiorespiratory health (HRRec and endothelial function). Future studies should continue to examine the impact of obesity on heart rate recovery and endothelial function in obese, but otherwise healthy adults and children. Additionally, examining VO₂ peak normalized to fat-free mass rather than total body weight may provide additional insights to this cohort. Loftin et al. (67) and Goran et al. (36) suggested that obese children may not have a lower VO₂ peak when normalized to their fat-free mass rather than their total body weight. Furthermore, incorporating FMD as an additional measure of endothelial function may further strengthen the results of future studies since FMD examines a separate vascular bed. This may provide a better interpretation of endothelial health in prepubescent children.

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References


74. McArdle WD, Katch FI, Katch VL. Exercise Physiology. 7$^{th}$ ed. Philadelphia: Lippincott Williams & Wilkins, 2010.


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