FAT OXIDATION AT REST AND DURING EXERCISE IN POSTMENOPAUSAL MIDDLE EASTERN AND WHITE WOMEN

FAIZA ABDULLAH

A THESIS SUBMITTED TO
THE FACULTY OF GRADUATE STUDIES
IN PARTIAL FULFILLMENT OF THE REQUIREMENTS
FOR THE DEGREE OF
MASTERS OF SCIENCE

July 2013

ABSTRACT

Background: The declining levels of estrogen and progesterone during the menopausal transition predispose women to an increase in overall body mass and altered adiposity distribution that may place them at risk for developing cardiometabolic disease. Estrogen, progesterone and testosterone levels have been linked to changes in lipid metabolism and since these hormone levels change during the menopausal transition they may contribute to the unfavorable alteration in body composition. Differences in fat oxidation rates (FOR) have been observed between ethnic groups and the hormonal modifications that occur after menopause have been speculated to influence these differences. Only select ethnic groups have been studied, and exploring additional groups will allow further understanding of the menopausal transition and FOR.

Purpose: The purpose of this study was to compare resting FOR, FOR during incremental exercise and $FOR_{max,}$ hormone levels, anthropometric characteristics, physical activity (PA) levels and cardiometabolic risk factors between Middle Eastern (ME) and W postmenopausal women.

Results: Significant differences between groups were observed are as follows; the ME women were younger ($p= 0.004$), observed their first period at an earlier age ($p= 0.023$) and had their last menstrual cycle early in life ($p= 0.031$). Time since first and last cycle was shorter in the ME group ($p= 0.007$). ME had a higher body mass ($p= 0.027$), a higher body mass index ($p= 0.32$) and a larger waist circumference ($p= 0.005$). W women had a higher peak aerobic capacity ($p=0.003$) and higher self-reported levels of PA ($p=0.017$).

Discussion: The ME women did not exhibit lower FOR at rest, during incremental exercise or at maximum exercise and ME women did not have a lower level of estrogen or have more cardiometabolic risk factors than the W women. This study sheds light on FOR within a new ethnic group of postmenopausal women not yet investigated. As well, data on FOR, FOR_{max} and the exercise intensity expressed as a percentage of VO_2 peak, at which maximal fat oxidation occurs using a treadmill protocol and hormonal measurements for ME and W postmenopausal

women. A treadmill protocol was created and used which provided whole-body FOR at multiple sub-maximal PA intensities. PA levels and anthropometrics were also obtained in this study providing detailed data on the health and fitness characteristics of the study populations.

TABLE OF CONTENTS PAGE #

ACKNOWLEDGEMENTS

During my time in graduate school and while completing this thesis I have a few people I would like to thank. First and foremost I would like to thank my supervisor Veronica Jamnik, if it wasn't for her tough love and encouragement this wouldn't have been possible. Next, I would like to thank my lab mates and colleagues for their help and support throughout this process, my family for their love and comfort during the stressful times and my supervising committee, Dr. Riddell and Dr. Gledhill, for their motivational support during the final stage of this process. Lastly, Allah, I would like to thank Allah for blessing me and continuing to shower down His love.

LIST OF TABLES PAGE #

LIST OF FIGURES PAGE #

LIST OF APPENDICES

ABBREVIATIONS

BMI – Body mass Index

BP –Blood pressure

CPAFLA – Canadian Physical Activity, Fitness and Lifestyle Approach

EE – Energy Expenditure

FAI – Free androgen index

FATmax – Exercise Intensity expressed as a percentage of VO_2 peak, at which maximal fat oxidation occurs

FFM – Fat free mass

FOR – Fat oxidation rate

FOR_{max} – Maximal fat oxidation rate

FSH – Follicular stimulating hormone

HDL – High-density lipoprotein

HPAPQ – Healthy Physical Activity Participation Questionnaire

HR – Heart rate

HRQOL - Health-related quality of life

HSL – Hormone sensitive lipase

LDL – Low-density lipoprotein

LH – Luteinizing hormone

LPL – Lipoprotein lipase

NEAT - Non-exercise activity thermogenesis

NIH - National Institute of Health

PA – Physical Activity

PAR-Q+ - Physical Activity Readiness Questionnaire for Everyone

RER – Respiratory exchange ratio

RPE – Rate of perceived exertion

SD – Standard Deviation

VCO_2 – Volume of Carbon dioxide produced

VE – Minute ventilation

VO_2 – Volume of Oxygen consumption

VO_2 max – Maximal aerobic power

VO_2 peak – Peak aerobic power

WC – Waist Circumference

WHO – World Health Organization

X– Mean

1.0 INTRODUCTION

The menopausal transition has been speculated to predispose women to comorbidities that are obesity-related thereby increasing the risk of cardiometabolic diseases (Carr, 2003; Sowers et al. 1995; Barrett-Connor 1993; Price et al. 1998). Changes in estrogen, progesterone, testosterone and other associated hormone levels during the menopause transition have been linked to increasing the risk of cardiometabolic diseases (Glendy et al 1937, Barrett-Connor et al. 1991, Brezinika and Padoms, 1994, Carr, 2003). Increased abdominal adiposity post menopause is the most prominent physiological change observed (Price et al. 1998), a feature that is speculated to promote increased cardiometabolic risk in aging women. Estrogen, progesterone and testosterone levels have been reported to influence substrate metabolism and more specifically fat metabolism at both rest and during exercise (Zarins et al.2010). As these hormone levels change during the menopausal transition they may be contributing to the increase in overall body mass and visceral adiposity distribution observed post menopause. Fat oxidation rates (FOR) at rest and during exercise have been noted to decrease though the menopausal transition and therefore the decrease in estrogen has been associated to decreases in FOR at rest and during physical activity (PA) (Santa-Clara et al. 2006; Hickner et al. 2001). In addition, differences in hormones levels (Setaiwan et al. 2006, Randolph et al. 2004) and FOR (Hall et al. 2010) have been observed among White, African American, European and South Asian ethnic groups. Select ethnic groups have been studied, but there are gaps in the literature. Although over 70% of postmenopausal Iranian women are reported to be abdominally obese there have been no studies of their hormonal and FOR characteristics during exercise. Therefore, in the present study, FOR at rest and during exercise in Middle Eastern (ME) and White (W) postmenopausal women will be examined.

2.0 LITERATURE REVIEW

2.1 MENOPAUSAL TRANSITION

The reproductive system of a female undergoes significant stages of change or transitional periods or phases during the course of life, which has been speculated to influence her physiology and risk for disease development (Glendy et al. 1937, Barrett-Connor et al. 1991, Brezinika and Padmos, 1994, Carr, 2003). A stage of change or transitional period that occurs late in life is called the menopausal transition (Burger et al. 2002, Nelson et al. 2005, Sherman, 2005, Burger et al. 2007), which is broken down into early and late perimenopause, menopause, plus early and late postmenopausal stages. The normal reproductive period before the onset of the menopausal transition is referred to as premenopause. The overall transitional period is a result of ovarian failure and a decrease in estrogen, progesterone and testosterone levels, which takes the woman from a state of having menses to the absence of menses (Burger et al. 2002, Sherman, 2005, Burger et al. 2007). Each time point of the menopausal transition has its own characteristics.

Perimenopause is the first stage in this transitional period which is characterized by the start of irregular and shortened menstrual cycles, increases in follicular stimulating hormone (FSH) and lutenizing hormone (LH) levels and the decline of reproductive abilities (Soules et al. 2001, Nelson et al. 2005, Sherman, 2005, Burger et al. 2007, Elavsky and McAuley 2007), these changes continue into the late perimenopause period where these changes continue and menstrual irregularities are greater. The time between the final menstrual cycle and up to 12 months post the final cycle is still part of the perimenopause stage. Menopause is a point in the transition that refers to the absence of cycles for 12 consecutive months (Soules et al. 2001). Menopause can only be confirmed and defined after 12 months of amenorrhea following the final menstrual period which reflects a near complete reduction of ovarian hormone secretion (Soules et al.

2001). Menopause is a natural occurrence but it can also be surgically induced through the removal of both ovaries (Sherman, 2005). While unique to each woman, the mean age at which the menopausal transition begins is 47.5 years and its duration is approximately 5 years (Gosden, 1985, Nelson et al. 2005). The postmenopausal stage begins after the woman has been in the state of amenorrhea for the preceding 12 months or more (Soules et al. 2001, Carr, 2003, Sherman, 2005) until her death. An illustration of the menopausal transition is depicted in Figure 1.

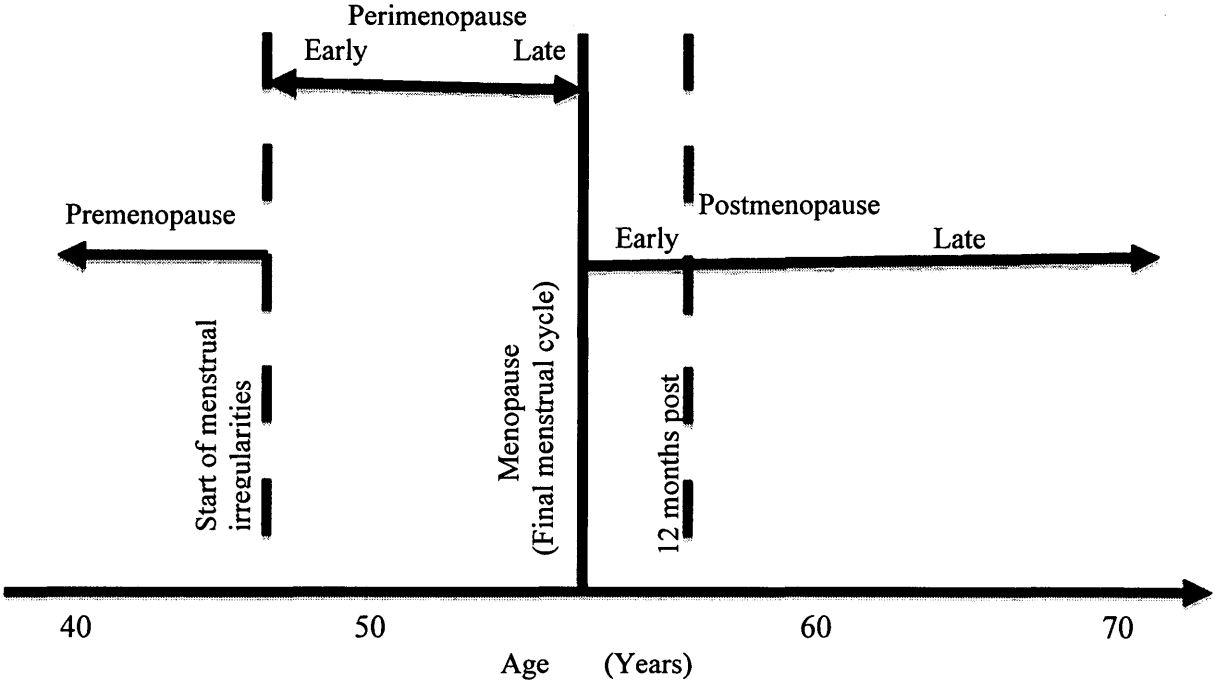


Figure 1. Illustration of the menopausal transition.

The symptoms of the menopausal transition include; hot flashes, night sweats, vaginal dryness, sleep disturbance, mood symptoms, cognitive disturbances, somatic and urinary complaints, abnormal uterine bleeding, sexual dysfunction, and perceived reduced quality of life (Nelson et al. 2005; Sherman, 2005). John Leake (1777), a researcher from the late 18th century commented on these symptoms as being 'chronic conditions' that only affect the female sex. With little knowledge about the menopausal transition and its associated physiology, women were

unaware of the changes that were occurring and were unable to understand the process of menopause. The physiology of menopausal transition and the associated hormonal changes have only been noted in literature since the 1920's (Soules et al 1982). In the nineteenth century women did not live to reach the age of menopause, but modern day women spend a third of their life in the postmenopausal state (Soules et al. 1982). The symptoms of the menopausal transition can vary from woman to woman, from the specific symptoms they experience and the severity of the symptoms. The physiological changes during the menopausal transition has only been well described in recent years, with research now focusing on the health related implications of the hormonal changes during the event.

2.2 Menopause Transition and Physical Activity:

Most investigations focus on the quality of life and the vasomotor symptoms associated with menopause and overlook other physiological symptoms. Alterations in PA participation and visceral fat accumulation, which also occur during the menopausal transition, may impact the overall health of a woman by increasing the prevalence of chronic conditions such as cardiovascular disease, hypertension, type 2 diabetes, osteoporosis (Sherman, 2005) and metabolic syndrome (Mayes and Watson 2004; Carr, 2003).

The symptoms of menopause can vary from woman to woman from the specific symptoms they experience and the severity of the symptoms. Exercise intervention studies have shown the increases in bodily movements can decrease the severity of the menopausal symptoms and improve over-all well being (Villaverde-Gutiérrezet et al. 2006; Elavsky and McAuley, 2007) summary provided in Table 1. Exercise is a sub-category of PA which is bodily movement resulting in energy expenditure (EE) varying in intensity (Caspersen et al 1985, Bouchard and Shephard, 1994). Exercise is planned, structured and has the objective of improving physical fitness components which require a threshold intensity to induce chronic adaptations (Caspersen

et al 1985, Bouchard and Shephard, 1994). PA can vary in intensity, duration and frequency and it includes; activities of daily living, incidental PA, non-exercise activity thermogenesis (NEAT) (Levine, 2008, Trembaly et al. 2010) occupational-related activities (Bouchard and Shephard, 1994, Sheppard, 1995) as well as active transport (Salli et al. 2004). Accumulating these differing forms of PA can benefit overall health (Sheppard, 1995, Warburton et al. 2007). By performing frequent and short episodes of PA one can accumulate and progress to meet the minimum global recommendations of PA, which are 150 minutes of moderate-to vigorous intensity PA over the course of the week (Haskell et al. 2007, Warburton et al. 2007, Tremblay et al. 2011, WHO, 2010).

Table 1. Summary of physical activity intervention studies on menopausal symptoms.

Authors	Characteristics	PA Intervention	Measurements	Findings
Villaverde-Guitierrezet et. al (2010)	-Sedentary, healthy postmenopausal women -Natural menopause ->70% had BMI>25	-12 month intervention -Control group and PA group -PA was at 50-80% of max HR 2x/week	-Self-reported HRQOL -Menopausal symptomatology	-PA group had improved symptomatology and HRQOL -Control had worsened symptomatology and HRQOL
Elavsky and McAuley (2007)	-Low PA or sedentary White women ->80% of women were transitioning through menopause or postmenopausal -Natural menopause, no HRT	-4 month intervention -Control, Yoga and walking groups -Yoga, 2x/week for 90-mins focused on strength, flexibility, balance and meditation -Walking, 3x/week for 1-hr was at 50-75% of HHR	-Perceived QOL, satisfaction of life, depression, menopausal symptoms, body fat (DEXA), VO_2 peak, PA	-Improvements in menopausal symptoms were observed across all groups, greatest improvement in the walking group

BMI – Body mass index, DEXA – Dual-energy x-ray absorptiometry, HHR – Heart rate reserve, HRT – Hormone replacement therapy, HRQOL – Health-related quality of life, QOL – Quality of life.

Several researchers have examined the effects of PA and its influence on menopausal symptoms. Villaverde-Gutiérrezet et al. (2006) conducted an exercise intervention study with sedentary postmenopausal women; the women were randomly assigned to either the experimental

or the control group. The majority of the study participants were overweight or obese, body mass index (BMI) of 25-30 and 30< respectively, but asymptomatic of disease. The exercise sessions incorporated aerobic conditioning and muscular strength training while maintaining the intensity of the exercises between 50-85% of age-predicted maximum heart rate (HR max). It also included range of motion movements for the major joints (stretching) and relaxation techniques. Initially the exercise duration was 30 minutes and progressed to 60 minutes by the end of the intervention. The PA sessions were led by a physiotherapist and lighting, temperature and music were used to set a relaxing environment for the participants. The self-reported health-related quality of life (HRQOL) measurement tool was employed to examine self-reported physical, psychological and social dimensions of functioning and well-being, social support, sexual decline and problems in the preceding week. Given the high adherence of the participants at 90%, the authors reported that a 12 month exercise program consisting of two sessions a week can improve the menopausal symptoms and HRQOL of menopausal women.

Another investigation by Elavsky and McAuley (2007) evaluated the effect of moderate aerobic exercise and Hatha style yoga for menopausal symptoms. Participants in the study were grouped in one of the following three groups; aerobic (walking), yoga or control (no intervention) group. Baseline measurements of menopausal symptoms, PA levels via the Aerobics Center Longitudinal Study Physical Activity Survey, maximal aerobic power measured through direct gas analysis, body composition via dual energy X-ray absorptiometry, depression, quality of life and satisfaction levels. The variables were all measured again following the 4 month intervention. The walking intervention consisted of 60 minute sessions three times a week, working at 50% of heart rate reserve (HRR) progressing to 75% of HHR by the end of the four months. Concurrently, the yoga group met twice a week and each 90 minute session focused on developing musculoskeletal fitness, flexibility and balance, as well as concentration and meditation. Both intervention groups accumulated 180 minutes of structured PA per week and

were encouraged to incorporate PA outside of the sessions and in their daily lives. The authors reported that walking had the greatest effect on attenuating menopausal symptoms, while yoga had a lesser overall effect but benefitted the participants more in the sexual domain.

2.3 Menopausal Transition and Disease Risk

A common physiological change during the menopausal transition is a gain in centralized adiposity also known as abdominal obesity, visceral obesity and central obesity. Alterations in the body's fat distribution from the more traditional gynoid (lower body fat storage) to android (upper body storage) is a notable feature (Price et al. 1998). However, it is unclear if fat deposition in the intra-abdominal area is a consequence of menopause alone or due to the lifestyle habits associated with aging.

It has been reported that fasting insulin levels are elevated and the risk of impaired glucose tolerance is greater when transitioning through menopause (Soler et al. 1989). Since estrogen has been shown to have significant effects on carbohydrate and lipid metabolism (Zarins et al. 2010), with the lower levels of estrogen during the menopausal transition, carbohydrates are more readily metabolized while lipids are stored and spared, contributing to the decrease in fat oxidation and greater fat accumulation (Zamboni et al. 1992). The menopausal transition has been speculated to predispose women to comorbidities that are obesity-related thereby increasing the risk of cardiometabolic diseases (Carr, 2003; Sowers et al. 1995; Barrett-Connor 1993; Price et al. 1998). This relationship was established after adjusting for age, BMI, household income and physical inactivity (Park et al. 2003). Cross-sectional studies have concluded that decreased basal lipolysis and increased lipoprotein lipase (LPL) activity in adipocytes from pre to post menopause may be a contributing factor predisposing women to gain fat mass (Ferrara et al. 2001). LPL is an enzyme responsible for hydrolysis of the triacylglycerol found in the blood and its uptake and storage of free fatty-acids (FFA) into adipocytes and other metabolically active

tissue (Pedersen et al. 2004, Mead et al. 2002). LPL found in adipocytes results in the storage of FFA and LPL found in muscle results in the oxidation of FFA (Ellis et al. 1994).

Adipose tissue contains two forms of estrogen receptors; estrogen receptor-α and the recently discovered estrogen receptor-β (Pedersen et al. 2004), which allows estrogen to bind to the adipocyte and influence processes within. This implies that estrogen plays a role in the regulation of the LPL gene expression (Mead et al. 2002). The study by Pedersen et al. (2004) demonstrated that estrogen attenuates the lipolytic response in subcutaneous adipocytes through up-regulation of the number of antilipolytic 2A-adrenergic receptor. This gene expression regulation differs between lower versus upper body, and this difference has been observed to change from pre to post menopause (Price et al. 1998). As a result, the lack of estrogen in postmenopausal women augments fat accumulation and suppresses LPL gene expression in specific areas of the body, contributing to the android fat distribution seen during the postmenopausal term. Estrogen now is known to influence substrate metabolism and more specifically FOR, however, the menopausal process also involves alterations in many other sex hormones such as testosterone, progesterone, etc. which will be discussed in further detail.

2.4 Fat Oxidation Rate

Fat oxidation refers to the metabolic process in which FFAs are metabolized to synthesize energy for use at rest or during PA. The plasma triglycerides provide a minimum of 50% of the FFA source and the remainder is provided by hydrolyzed intramuscular triglycerides (Stefanick and Wood, 1994). The proportion of fat to carbohydrate oxidation differs at varying PA intensities (Christensen and Hansen, 1939). FFAs are the primary source of energy at rest and during prolonged sessions of light to moderate intensity PA (below 50% of maximum aerobic power) (Stefanick and Wood, 1994), and as the intensity of the PA increases, carbohydrate contribution increases and concurrently, fat oxidation decreases (Jeukendrup and Achten, 2001).

Substrate oxidation can be estimated by the use of the respiratory exchange ratio (RER), which ranges from 0.7 to 1.0 in value, with 100% fat oxidation occurring at 0.7 and 100% carbohydrate oxidation occurring at 1.0 of the range (Lusk, 1928).

The standard method for measuring substrate metabolism is human is through indirect calorimetry. This is performed though measuring oxygen consumption (VO_2) and carbon dioxide (VCO_2) production that provides information on the substrate type and rate of utilization in an organism (Lusk, 1976). By measuring VO_2 and VCO_2, RER absolute FOR can be calculated. Indirect calorimetry is non-invasive compared to other methods such as the use of radioisotopes, blood extractions and muscle biopsies and therefore more widely used. Different protocols have been designed to measure FOR during exercise, most of them being conducted on a cycle ergometer, only calculating FOR at specific intensities while others have created protocols that are incremental and measure FOR at multiple sub-maximal exercise intensities. When measuring FOR in an incremental to maximum effort protocol it is important to have reasonably short work periods (2- 4 minutes) with gradually increasing exercise intensities, allowing both for steady state gas exchange to occur but also for more data collection and therefore more FOR calculations across a wide range of intensities before the participants becomes exhausted. The FOR at each workload and its associated exercise intensity expressed as a percentage of maximal or peak oxygen consumption (VO_2 max or VO_2 peak) is fitted to a polynomial curve, FOR along the y-axis and % VO_2 max or VO_2 peak along the x-axis. This curve will provide information on the FOR throughout the exercise, FOR_{max} and the % VO2 max or VO_2 peak that elicited the FOR_{max}, referred to as FATmax. Knowledge of the FOR_{max} and FATmax or the range of intensities one can oxidize the most FFA have importance in both health-related fitness and performance-related fitness (Jeukendrup and Achten, 2001).

Researchers have examined FOR at rest and during varying sub-maximal intensities. They have also investigated the differences between sexes, age groups and physical fitness levels.

The modality that an individual uses to exercise also influences substrate utilization for the same relative intensity in both fed and fasted states (Achten et al. 2003). Researchers examined the FOR using the treadmill and compared the FOR using the cycle ergometer (Achten et al. 2003). They concluded that fat utilization is higher on the treadmill over a wide range of the same relative intensities. Also, FOR_{max} attained was greater on the treadmill but occurred at the same FATmax as on the cycle. A rational for the higher FOR at the different intensities on the treadmill is due to the greater muscle mass recruitment during treadmill activities and therefore a greater release of catecholamines. Catecholamines are activators of lipid mobilization and thus promote fat oxidation during exercise; and the release of catecholamines are relative to the muscle mass recruitment (Davies et al. 1974), explaining the greater FOR on the treadmill than for cycling exercise.

Another study, in which the two modalities were compared, reported that the intensity that elicited FOR_{max} was higher while running than cycling (Chenevière et al. 2010). Maximum fat oxidation occurred at 57.2% of VO_2 max on the treadmill and at 44.2% of VO_2 max on the cycle ergometer (Chenevière et al. 2010). Participants had to work at a higher intensity on the treadmill in order to reach their FOR_{max}. The researcher also concluded that FOR only differed between the two modalities when the intensity of the exercise exceeded 70% of VO_2 max (Chenevière et al. 2010) and attributed this difference to the greater muscle recruitment during treadmill running versus cycling. The different outcomes from these two studies could be attributed to the different testing protocols that were used on the treadmill, while Achten et al. (2003) used a walking protocol, Chenevière et al. (2010) used a running protocol. Overall, both studies concluded that FOR are greater on the treadmill and fat utilization is higher across all workloads on the treadmill in comparison to the cycle ergometer.

Other variables known to alter fat oxidation are sex, age and maturation, and aerobic fitness level. Researchers have shown that women utilize more FFA in comparison to men at any

given sub-maximal intensity (D'eon and Braun, 2002). This discrepancy is attributed to the higher levels of estrogen and progesterone which are innate to women (D'eon and Braun, 2002). Both men and women undergo decreased efficiency of fat utilization at rest as they age, regardless of the increase in fat mass (Rising et al. 1996). Furthermore, during exercise older individuals oxidize less fat at the same absolute and relative intensities in comparison to young adults (Sial et al. 1996). This change in lipid oxidation with age has been related to the decrease in skeletal muscle metabolism and not to FFA availability (Sial et al. 1996). Endurance training improved lipid oxidative capacities in older adults (Johnson et al. 2010, Zarins et al. 2010) similar to levels observed in untrained young adults.

As a result of endurance training skeletal muscle mitochondrial content increases, which in turn results in a greater muscle respiratory capacity (Sial et al. 1998). Efficiency in fat utilization through aerobic training has also been documented in young adults (Friedlander et al. 1998, Sidossis et al. 1998). Similar adaptations occur as in the older adults. Strength training also has its effects on improving fat utilization during rest and over a 24-hour period (Treuth et al. 1995). Factors such as sex, age and fitness level or exercise training have been documented to alter the rate at which FFA are utilized at rest and at varying sub-maximal exercise intensities. Table 2. summarizes the FOR studies that were discussed above.

Table 2. Summary of studies in which the different factors that influence fat oxidation rate were examined.

Authors	Participant Characteristics	PA Intervention	Findings
Achten et al. (2003)	-12 healthy, moderately trained men -Mean age 21 yrs -All individuals went through both protocols	-Cycle, starting stage at 95W, increase by 35W every 3-mins until exhaustion -Treadmill, 7.5km•h^{-1} at 1% incline and incline was increased by 2% every 3-mins until and RER of 1.0 was attained	-FOR is higher on the treadmill versus cycle over a wide range of the same relative intensities -FOR_{max} attained was greater on the treadmill but occurred at the same relative intensity as on the cycle -FOR_{max} was attained at 59.2% of VO_2 max on the treadmill and at 62.1% of VO_2 max on the cycle ergometer
Chenevière et al. (2010)	-13 healthy and moderately trained men and women -Mean age 29 yrs -All individuals went through both protocols	-Cycle, began at 40W and increased by 20W every 3-mins until RER of 1.0 was attained -Treadmill, began at 3km•h^{-1} at 1% incline, speed was increased by 1km•h^{-1} every 3-mins until a RER of 1.0 was attained	- FOR_{max} and FOR from 70-85% of VO_2 max were higher for running than cycling -FOR_{max} was attained at 57.2% of max VO_2 on the treadmill and at 44.2% of max VO_2 on the cycle ergometer
Sial et al. (1996)	-6 elderly, mean age 73 yrs and 6 young mean age 26 yrs, matched for gender and lean body mass	-Cycle ergomter was used to measure substrate utilization at 56% of VO_2 max, younger group was measures at the same relative and absolute intensities	-Mean FOR was 25-35% lower in the elderly group than in the young adults at both the same absolute and relative intensities -FOR is decreased and carbohydrate oxidation is increased during moderate intensity exercise in elderly men and women
Zarins et al. (2010)	-10 healthy, nonsmoking, postmenopausal women -Mean age 55 yrs	-1-hr of supervised moderate-intensity aerobic exercise 5 days/wk for 12 weeks (4 sessions on the cycle and 1 treadmill) -Training was at 50-65% of peak HR	-There was a decreased carbohydrate and increased lipid oxidation rates at the same absolute, but not relative exercise intensity
Friedlander et al. (1998)	-8 healthy, nonsmoking, sedentary female between the ages of 18 – 35 yr	-5 days a week for 1-hr each day on the cycle ergometer. -Aerobic training intensity was gradually increased from 50% to 75% of VO_2 peak	-FOR increases after endurance training at the same relative (117%) or absolute (58%) intensity
Treuth et al. (1995)	-13 healthy women -Mean age 67 yrs	-Whole body strength training sessions occurred 3days/wk for 1-hr -2 sets of 12 reps for each exercise	-9.1% increase in resting energy expenditure -93%increase in fat oxidation and 84% decrease in carbohydrate oxidation

2.5 Fat Oxidation Rate and Ethnicity:

Until recently, the FOR in different ethnic groups had received minimal attention in the literature. Hall et al. (2010) studied the FOR of South Asian men, an ethnic population that is at an elevated risk for developing abdominal obesity and metabolic syndrome. Hall compared the South Asian men with BMI-matched European men. Participants were not matched for physical fitness levels, but habitual PA levels were measured through the self-report using International Physical Activity Questionnaire, and daily food intake was also measured through a food diary. PA and food intake were compared between the two groups and no significant differences were observed. The authors concluded that the South Asian men had a reduced capacity for fat oxidation during sub-maximal exercise in comparison to the European men, after correcting for age, BMI and fat mass. The study findings suggest that there may be ethnic differences in FOR despite not controlling for physical fitness level.

Ethnic differences have also been detected and documented in females. Ethnic disparities in resting metabolic rate (RMR) and FOR at rest and during exercise (Santa-Clara et al. 2006; Hickner et al. 2001) have also been documented between both premenopausal and postmenopausal African-American and White women. These studies collectively suggest that the lower RMR and FOR at rest and during exercise are linked to the weight gain observed with age not necessarily due to the menopausal transition, in African-American females. Anthropometric variables and physical activity levels were not matched in these studies between the groups, although differences in RMR and FOR were detected between ethnic groups, all confounding variables were not controlled for.

Decreased FOR at rest and during PA may have important clinical implications and can influence one's ability to manage weight. Furthermore, significant increases in abdominal obesity and overall weight gain have been observed in certain ethnic populations as a function of age. In a population-based study, Janghorbani et al (2007) looked at overweight and obesity rates

in Iranian adults. They found that 36% of the women between ages 55-64 were overweight and 31.1% were obese class 1 using the World Health Organization (WHO) BMI cut-off points; underweight <18.5, normal weight 18.5-24.9, overweight 25.0-29.9, obese class I 30.0-34.9, obese class II 35.0-39.9 and obese class III >40.0 (WHO, 2000). In the same age group 73.3% were categorized as being abdominally obese, having a waist circumference (WC) of 88 cm or greater. The Iranian community is a group in which the majority of women above 55 years of age are abdominally obese, overweight, or obese therefore possessing physiological characteristics that will increase their risk for developing chronic metabolic and cardiovascular conditions (Carr, 2003).

2.6 Menopausal transition, Hormonal Changes and Their Suspected Impact on Fat Oxidation

As mentioned earlier, perimenopause is the term used when menstrual irregularities first begin and this phase of the menstrual transition is broken up into early and late perimenopause. The time frame for each of these periods is individualized to each woman. Menopause is referred to the last menstrual cycle the woman has, this is not established until 12 months post when they fulfill the criteria. The time from the last menstrual cycle and onward is referred to as post menopause, where no further cycles occur. During each of these phases of menopause FSH, estrogen, progesterone and testosterone levels shift which are discussed in detail later in the current paper.

The underlying physiological process of the menopausal transition is linked to the hormonal alterations. As mentioned before the hormonal changes that occur are that FSH levels increase and estrogen, progesterone and testosterone levels drop and these collective changes are speculated to influence disease risk as women age (Glendy et al. 1937, Barrett-Connor et al. 1991, Brezinika and Padmos, 1994, Carr, 2003). Differences in hormone levels have been observed

within ethnic groups when researchers examined FOR in White and African-American women and discovered that hormonal variations among these women may be used to explain the diverse FOR premenopause. More specifically estrogen and genetic disparities have been stipulated as the contributing factor in determining the differences between these groups (Santa-Clara et al. 2006; Hickner et al. 2001). In the postmenopausal period, estrogen discrepancies among ethnicities persist (Setaiwan et al. 2006) and are assumed to continue to have an influence on FOR. Randolph et al. (2004) demonstrated that estrogen and FSH levels varied by ethnicity when measured longitudinally over the course of the menopausal transition, indicative of ethnic specific variations in the pituitary-ovarian relationship.

Estrogen and FSH levels also differed between age independent of menopausal status and the influence of BMI varied by menopausal status (Randolph et al.2004). In the premenopausal and early perimenopausal phases BMI had a negative effect on estrogen in comparison with the transition from late perimenopause to post menopause where BMI had a positive effect on estrogen levels (Randolph et al.2004). The number of children and the age of first birth have not been associated with significant differences in hormone levels, whereas late age at menarche was modestly associated with decreased estrogen concentrations post menopause (Setaiwan et al. 2006). Different variables have shown to effect hormone levels premenopause as well as through the menopause transition, which indirectly influence FOR.

Alterations in the hormonal milieus of women continue to impact their health in the later years of life. In brief, in a normal healthy female, estrogen concentrations drastically decrease through the menopausal transition and FSH levels increase in response. The biological measure of menopause occurs when FSH levels exceed 50 IU/ml (Burger et al. 1995) in addition to having no menstrual cycles in the preceding 12 months. These modifications influence the concentrations of other metabolism altering sex hormones such as progesterone, testosterone and luteinizing hormone (LH); therefore making way to alterations in substrate metabolism by

favoring fat storage and influencing woman's overall physiology. When estrogen levels decrease dramatically at the onset of the menopausal transition, FSH increase while LH levels stay relatively stable, these are the two hormones that assist in follicular maturation and the production plus release of estrogen (Soules and Bremner, 1982). The elevated FSH levels are required to mature the follicles in the ovaries and prepare them for conception, this no longer occurs during the menopausal transition (Soules and Bremner, 1982). FSH levels will increase as estrogen levels decrease and inhibin, a regulator of FSH, will also increase in response to decreased levels of FSH (Soules and Bremner, 1982, Randolph et al. 2004). During the menopausal transition the main goal of FSH is to promote estrogen secretion and follicular maturation. The influential effect of increased FSH levels during and after ovarian failure and its relation to the FOR is unknown. These collective hormonal changes are the start of may other physiological changes that occur during the menopausal transition.

There are different forms of estrogen, each one having its dominance during different stages of life and the reproductive cycle. Estradiol is the prominent form and the most physiologically active form in the younger years where as estrone is the most prominent form of estrogen post menopause. Both forms of estrogen are produced from circulating steroids that are released by the ovaries and by the adrenal glands. Steroids converted in the ovaries produce estradiol and steroids converted in the adipocytes produce estrone (Kalyani et al. 2009) and the transcription of estrone increases with age (Price et al. 1998). Estrogen, regardless of its form, is still the main factor in determining women's ability to oxidize fat (D'eon et al 2002), but with reduced levels its role in fat oxidation is diminished. Estrogen receptors have been found on adipocytes which indicate that estrogen plays a role in lipid metabolism (Pedersen et al. 2004). In addition to estrogen receptors, progesterone and androgen receptors have also been found on adipocytes (Mayes and Watson, 2004) which lead to the idea that these hormones may also have an effect on lipid metabolism. Figure 2 summarizes how different levels of estrogen and

progesterone affect lipid synthesis and metabolism in muscle and adipose tissue using results from animal studies.

Progesterone is also a steroid hormone but its source of production and release is different than estrogen; progesterone is released from the corpus luteum, placenta and adrenal glands and it is a precursor for the production of testosterone and estrogen (D'eon and Braun, 2002). Studies observing the effects of progesterone on FOR in both human and animal studies have observed mixed results (D'eon and Braun, 2002). Human studies that observed different phases of the menstrual cycle when progesterone levels are elevated above estrogen levels (Toth et al. 1987, Hackney et al. 1994), it was difficult to ascertain the effect of progesterone independent of estrogen on FOR (D'eon and Braun, 2002). In animal studies looking at ovariectomized rats, researchers found progesterone behaved similarly to estrogen in suppressing carbohydrate utilization and promoting fat oxidation (Kendrick et al. 1987, Kalkhoff, 1982), whereas others investigators found that to have a opposing effect to estrogen, FOR was reduced (Hatta et al. 1988, Hansen et al. 1996, Campbell and Febbraio, 2001). Once again, Figure 2 briefly summarizes how estrogen and progesterone influences fat metabolism using an animal model. Links between progesterone and testosterone (androgen) levels with lipid metabolism have not been established in postmenopausal women. The concurrent decrease in progesterone and testosterone levels decrease along with estrogen may have an impact on FOR.

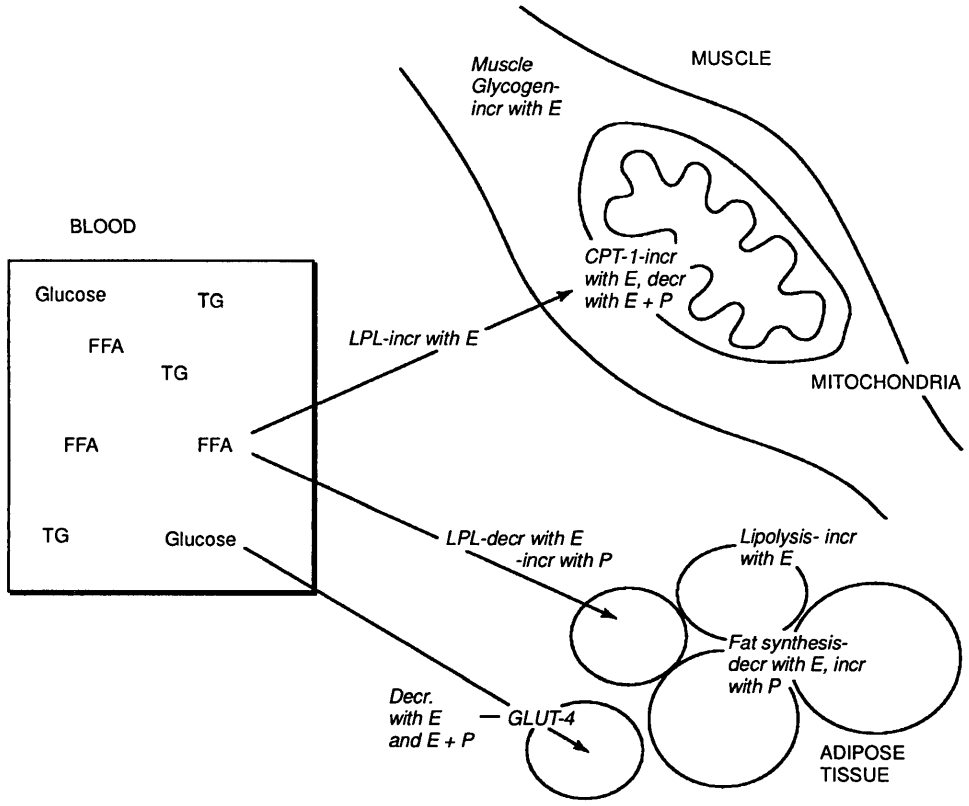


Figure 2. 'The effects of estrogen and progesterone on pathways of carbohydrate and fat metabolism. Mechanisms shown are derived from an integrated view of data collected in animal models.' Modified from D'eon et al. (2002). *CPT-1 caratine palmityltransferase 1, TG triglycerides, incr increase, decr decrease, E estrogen, P progesterone.*

Low amounts of ovarian estrogen result in greater amounts of inhibin and the up regulation of FSH (Soules and Bremner, 1982, Randolph et al. 2004). FSH and LH are hormones that are responsible for the stimulation of the ovaries to release sex hormones (estrogen, progesterone, testosterone) and mature the follicles. The influential effect of FSH and LH on sex hormone levels after ovarian failure is unclear and its relation to the FOR is unknown. Sex-hormone binding globulin (SHBG) levels, a protein responsible for the transport of estrogen and testosterone, also drop concurrently with sex hormones levels. There is a difference in the affinity of SHBG for the different sex hormones; it has a greater affinity for testosterone than for estrogen and therefore a greater binding and transport capacity for testosterone and reduced capacity for estrogen (Janssen et al. 2008). This selective property of SHBG influences the

transport of estrogen by favoring testosterone transport and therefore serves as a marker of estrogen bioavailability (Janssen et al. 2008) which also is an indirect measure of androgenicity (Kalyani et al. 2009), elevated testosterone levels. Therefore, the effects of testosterone on the physiological process within women during the postmenopausal term are enhanced.

Associations have been made in a longitudinal cohort study, between elevated levels of testosterone and suppressed levels of SHBG with overall obesity and abdominal obesity in women independent of age, PA and other chronic health conditions (Sutton-Tyrrell et al. 2010). In a cross-sectional study, elevated testosterone and low levels of SHBG were found to impair fasting glucose in postmenopausal women (Kalyani et al. 2009). In men, testosterone plays an important role in substrate metabolism (Saad and Gooren, 2011) and low levels have shown to be negatively associated with blood pressure, fasting plasma glucose, triglycerides, body mass index and abdominal obesity, as well as demonstrating positive correlations with HDL-cholesterol (Marin et al. 1996). Sutton-Tyrrell et al. (2010) suggest that high levels of androgens impair insulin action in skeletal muscle and adipose tissue, contributing to reduced whole-body insulin sensitivity; a known physiological characteristic of menopause. With the emerging evidence of androgen receptors being located on adipocytes the effect of testosterone on FOR in women is not well understood and with the increased dominance of testosterone post menopause it is crucial to understand its influences. Free testosterone can also be measured in women when SHBG and testosterone levels are known and this value is known as a free androgen index (FAI). This is a ratio of total testosterone to the concentration or the binding capacity of SHBG (Vankrieken, 1997). Strong relationships were established between obesity and high free FAI and low SHBG in postmenopausal women (Sutton-Tyrrell et al. 2010). Elevated androgen levels are now considered as a predictor of obesity in women transitioning through menopause (Sutton-Tyrrell et al. 2010).

Insulin is a metabolic stabilizing hormone that influences substrate utilization by controlling the storage and breakdown of glucose (Saltiel and Kahn, 2001). It is known that insulin greatly inhibits the breakdown of fat in adipocytes through reducing the activity of hormone sensitive lipase (HSL) and increases lipid synthesis (Saltiel and Kahn, 2001, Bjorntorp, 1996). Insulin resistance trails the development of abdominal obesity (Janssen et al. 2008, Kohrt et al. 1993) and in the case of insulin resistance there is an excess of insulin. As mentioned before SHBG levels affect estrogen and testosterone activity and now it is being postulated that insulin may inhibit SHBG secretion (Svendsen et al. 1993). Janssen et al. (2008) have inferred strong associations between incident metabolic syndrome to higher FAI and lower SHBG levels. Moreover, insulin does not only control lipid metabolism directly but it influences estrogen and testosterone bioavailability through the management of SHBG secretion, which then can effect FOR.

Cortisol is a hormone that fluctuates throughout the day in response to life's everyday stressors. Like insulin, cortisol promotes lipid synthesis at the adipocyte, inhibits adipose tissue metabolism and affects fat distribution (Bjorntorp, 1996, Bjorntrop, 1997). Researchers suggest that cortisol levels increase in response to abdominal obesity (Bjorntorp, 1996) and high cortisol levels have been associated with a greater waist to hip ratio and abdominal obesity in postmenopausal women (Marin et la. 1992). Paradoxically, cortisol is also linked to increases in adipose tissue lipase enzyme expression, which would be expected to increase the capacity for lipolysis (Peckett et al. 2011). Table 3 summarizes all the hormonal changes that take place throughout the menopausal transition.

Table 3. Summary of the function and production of hormones and the changes as a result of menopausal transition.

Hormone	Function/Production	Effect of menopausal	Influence on FOR
Estradiol	-Produced in adipocyte and adrenal glands	↑	↑ lipid metabolism -More active form
Estrone	-Produced in ovaries	↓	↑ lipid metabolism -Less active form
Progesterone	-Precursor to estrogen and testosterone production -Produced in the corpus luteum, placenta and adrenal glands	↓	↑↓ unclear
Testosterone	-Produced in the ovaries	↓	Unknown, high FAI linked to obesity
Follicular stimulating hormone	-Released from the pituitary -Responsible to the stimulation of ovaries to release sex hormones	↑	Unknown
Luteinizing hormone	-Released from the pituitary -Responsible to the stimulation of ovaries to release sex hormones	↔	Unknown
Sex-hormone binding globulin	-Transports estrogen and testosterone -Has a greater binding and transport capacity for testosterone than for estrogen	Slight ↓	Unknown, low levels linked to obesity
Insulin	-Released by the pancreas -Influences substrate utilization by controlling the storage and breakdown of glucose	↑ as result of insulin insensitivity	↑ lipid synthesis and inhibits lipid metabolism
Cortisol	-Released by the adrenal glands	↑ with an ↑ in central fat	↑↓ unclear

3.0 RATIONALE, PURPOSE AND HYPOTHESIS

Although fat oxidation and cardiometabolic disease risk have been studied in various ethnic groups, ME women are a group for whom no information has been reported on either FOR or the effects of the menopausal transition on disease risk. The purpose of this study was to compare resting plus exercise FOR, FOR_{max}, FATmax, hormone levels, anthropometric characteristics, PA levels and cardiometabolic risk factors between postmenopausal ME and W women. It was hypothesized that the ME women would exhibit lower FOR at rest and at different sub-maximal exercise intensities and have a lower FOR_{max} and FATmax as well as, lower levels of estrogen and posses higher cardiometabolic risk factors compared to the W women.

4.0 METHODS

4.1 PARTICIPANTS AND RECRUITMENT

Participants were recruited through friends, fellow students, advertisement posted throughout York University, and networking through cultural community centers. Once the recruitment process was completed the participants were grouped based on their ethnic origin ME or W, which was determined by their birthplace. Menopausal status was based on the number of menstrual cycles the participant reported having in the past 12 months in the pre-screen and confirmed by their FSH level, >30 mIU/mL with the blood draw. Participants were healthy females, undiagnosed of any major illness, not on metabolic altering medications or hormone-replacement therapy, and had not undergone surgically induced-menopause. In order to attain power of 0.05 for a two-tailed alpha with an effect size of 0.8, 12 participants were recruited for each group.

4.2 PRE-SCREENING

Before the initiation of the exercise protocols, the participants were taken through an extensive pre-screening procedure. Pre-exercise screening took place on the first experimental day. They completed the Physical Activity Readiness Questionnaire for Everyone + (PAR-Q+) (Warburton et al. 2011) (Appendix A) and an Informed Consent document (Appendix B). The PAR-Q+ was used to clear the participants for exercise and to confirm the absence of any metabolic conditions that may hinder the outcome of the research. The participants had no contraindications or restrictions to exercise participation based on their responses on the PAR-Q+. Participants that were not taking medications for diabetes, cholesterol or hormone replacement therapy were accepted into the study. In addition to explaining the risks involved in taking part in the research, the Informed Consent document outlined the purpose, importance of the study and how it will add to the existing body of research and improve clinical practices in

weight management and disease prevention. Pre-exercise heart rate (HR) and blood pressure (BP) were measured using the BpTRU, electronic blood pressure monitoring device, to ensure participants were within an acceptable range before the initiation of the exercise protocols; BP <160/90 mmHg and free of comorbidities (Thomas et al. 2011). The participants had their blood pressures and heart rates measured on both the right and left arms while seated in a relaxed position, feet flat on the ground and legs uncrossed. Six measurements were taken consecutively and averaged. The average values were obtained and used in the analysis.

4.3 PARTICIPANT CHARACTERISTICS

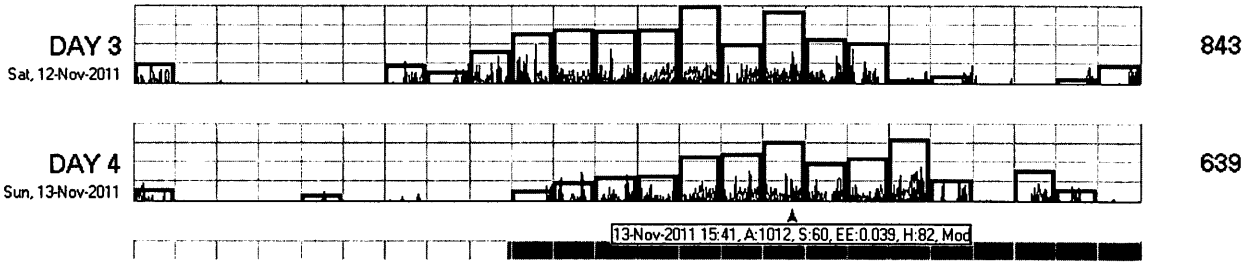
4.3.1 Anthropometric Characteristics

Standardized protocols were used to measure height, weight and waist circumference (WC). Height was measured without shoes using a stadiometer (Fitness Precision, *Toronto Ontario*). Body mass was measured using a digital scale (Seca Alpha, *Germany*) with no shoes and light clothing. WC was measured using the National Institute of Health (NIH) protocol; the measuring tape was placed on the skin at the level of the iliac crest. Body fat percentage was determined through bioelectrical impedance analysis instrument (Tanita Scale, model TBF-612, *Arlington Heights, III*). Skinfold measurements were taken using Harpenden fat calipers at standard sites; bicep, tricep, subscapula, iliac crest and medial calf, as per the Canadian Physical Activity, Fitness and Lifestyle Approach (Gledhill and Jamnik 2003). Markings were made at the skinfold sites as well as one centimeter below, first for where the fold was made and the second for the placement of the jaws of the fat caliper. BMI was calculated using body mass in kilograms divided by height in meters^2. BMI, WC along with skinfold sum was used to rate the participants' Composite Body Composition Health Risk Score (Gledhill and Jamnik, 2003).

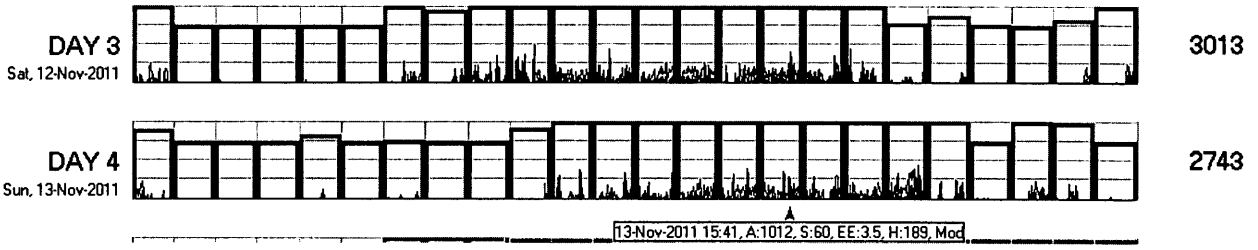
4.3.2. Physical Activity

Participants' PA levels were characterized through the use of omnidirectional Actical (Respironics Inc, 2008) accelerometers and the self-reported Healthy Physical Activity Participation Questionnaire (HPAPQ) (Gledhill and Jamnik, 2003). Both objective and subjective forms of PA assessment tools were used to reduce associated limitations and biases. The accelerometers allowed for 15-second epoch analysis of intensity and volume of PA, step counts and energy expenditure (EE) (Riddoch et al. 2007), while the questionnaire provided information on the frequency and intensity of the activity as well as perceived fitness (Gledhill and Jamnik, 2003). PA levels with the accelerometers were tracked over a 3-day period consisting of two weekdays and one weekend. The accelerometer was worn on a waist belt located over the iliac crest. A minimum of 600 minutes of data per day was collected for adequate analysis, 10 waking hours out of a 24-hour day (Riddoch et al. 2007). As illustrated in Figure 3. the accelerometers' software program provided detailed graphs for each participant where the datum was obtained. The graphs provided activity counts, step counts for 15-second epoch periods of time and hourly averages of EE. Activity counts are a measure of PA that considers both the intensity and frequency of the activity. The three-day averages for each of the measurements were calculated and used in the analysis in addition to the raw HPAQ scores.

Figure 3. Actical accelerometer graph example.
A) Visual of 2 day collection, black line graph illustrate activity count, blue line graph illustrates step counts, green bar graph illustsrates hourly EE without including resting metabolic rate calculated through body surace area equation.



B) Same as above but hourly EE includes computer calculated resting metabolic rate.



4.4 Blood and Urine Profile

Blood samples were drawn from each participant at the beginning of the second session while in a fasted state to characterize the blood lipid, sex hormone and glucose levels. The blood extractions were performed by a certified phlebotomist. When the blood was obtained from the participants, the vials rested for 30 min before being spun in a centrifuge for 15 min. After the samples were spun the plasma was transferred to storage vials to be sent out to an external laboratory (Canadian Life Labs) for analysis.

Lipid Panel. HDL-cholesterol, LDL-cholesterol, triglycerides, total cholesterol, were measured and the HDL-cholesterol to total cholesterol ratio was calculated to ensure normal blood lipid levels for each participant.

Hormone Panel. As part of the blood draw, the following hormones were analyzed; estradiol, estrone, progesterone, testosterone, SHBG, androgen index which was calculated using the values from the testosterone and SHBG, FSH, LH, and insulin.

Urine Sample. Urine samples were obtained to measure cortisol levels. The urine samples were taken on the same morning as the blood draws, while in a fasted state. The sample was collected in sterile urine cups and transferred to vials to be sent to an external laboratory (Canadian Life Labs) along with the blood samples for analysis.

4.5 Exercise Protocol

Open circuit spirometry, a form of indirect calorimetry was used during the graded exercise test and the fat oxidation protocol. The details of the open circuit spirometry involved, the participant inhaling air from the atmosphere and expiring air through a mouth-piece attached to a two-way valve (Ewald Koegal Co, S*an Antonio Texas*), connected to a hose and then to a 120L Tissot gasometer (Warren E Collins LTD. *Braintree, Massachusetts*), while wearing a nose plug. The fractional concentration of expired oxygen (F_EO_2) and carbon dioxide (F_ECO_2), and volume of air expired, or minute ventilation (VE), were measured in the final 30 seconds of each 2 min workload and from that VO_2 and VCO_2 was calculated. Oxygen and carbon dioxide fractional concentrations of the expired air were analyzed through rapid response gas analyzers (Applied Electrochemistry, Model S-3A and CD-3S, *Sunnyvale, California).* The graded exercise test was performed on the first day the participants arrived in the laboratory. The tests were conducted by qualified exercise professional (Warburton et al. 2011), the first exercise day was used to familiarize the participant to walking on a treadmill and to take them through an incremental to maximum effort treadmill loading sequence for the determination of VO_2 max or VO_2 peak.

Participants wore electronic heart rate monitors (Polar Electro, *Kemple Findland*) during

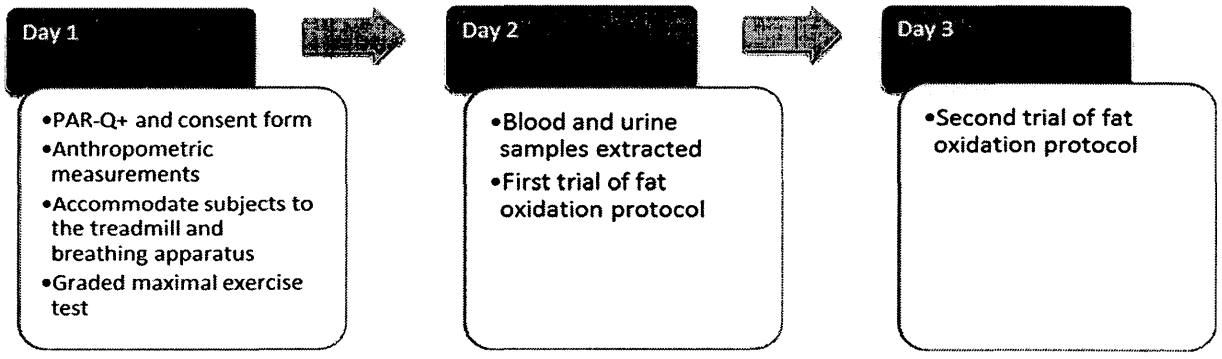
the incremental to maximal test, the protocol for this test had 2-min work stages that increased in intensity at every stage, the last 30-seonds of the 2-min stages were when the gas collection took place, HR and the rating of perceived exertion (RPE) were also obtained. The participants were instructed to remain on the treadmill until they reached volitional fatigue at which point they received a 2-minute low-intensity active recovery. Following the recovery period, the participants continued the test for another stage, and then were given another 2-minute break after completing the workload. This discontinuous portion of the graded exercise test was used to ensure that the attainment of VO_2 max or VO_2 peak. The test was terminated when the participant was either no longer able to continue or when the criteria for attaining VO_2 max was reached. The attainment of VO_2 max was determined by applying the following criteria; a plateau in VO_2 with increasing workloads where VO_2 does not increase more than 1.5 mL of $O_2 \cdot kg^{-1} \cdot min^{-1}$, a respiratory exchange ratio (RER) value greater than 1.15, and no increase in heart rate with an increase in workload (Howley et al. 1995; Heyward, 2006). The term VO_2 peak is being used in this study instead of VO_2 max because most participants did not exhibit the criteria for attaining VO_2 max, the VO_2 values that were obtained represented a peak.

On the second experimental day, the study participants underwent the fat oxidation protocol following an 8-10 hour overnight fast. The fat oxidation protocol was a modified version of Achten, Venables and Jeukendrup treadmill protocol (2003). The workloads were the same duration in length and the increase in intensity at each new stage was consistent, but the current protocol was walking in nature, rather than running. At the start of the protocol, electronic heart rate monitors were strapped onto the participants around their chest on the skin in order to measure heart rate at different workloads in addition to RPE. Resting VO_2 and fat oxidation measurements were obtained with the participant seated in a chair. Expired air was collected at rest for 5-6 minutes, a nose plug was used to prevent any air from leaking out from the nose and to ensure that all the expired air was collected in the Tissot tanks. Expired gas was

collected and analyzed; VE, VO_2 and VCO_2 were calculated, similar to the graded exercise test and then used in the formula to calculate FOR. The protocol began at 1.5-1.8 mph and 1% incline; depending on the comfort and height of the participant. Speed increased by 0.2 mph every 3 minutes until the participant was at a brisk walking pace, 3.4-3.6 mph. Once the maximum walking speed was attained, the incline increased by 2% every 3 minutes thereafter, until an RER of 1.0 or greater was obtained. The last 60-30 (in the earlier workloads a longer gas sample was obtained) seconds of the workload were used to collect the expired gases for the calculation of FOR. At the end of each workload both HR and the participants' RPE score were obtained and were used to gage the participants' work intensity. The third day was used to perform a second trial of the fat oxidation protocol, the same starting speed and loading sequence were used, duplication of the first trial was attempted. The FOR_{max} values of the two trials were used in the analysis.

Since walking is the more common form of movement and transportation in daily human life, the use of a treadmill/walking protocol was used in this study to assess FOR over a range of sub-maximal intensities. Walking is a movement familiar to all people and is a representative of daily movements in comparison to cycling. The use of a walking protocol makes this study unique in that walking is a weight-bearing activity which incorporates greater muscle recruitment resulting in a greater demand for energy. While, most studies have employed a cycle ergometer protocol as their method for measuring FOR. The study schematic is summarized in Figure 4.

Figure 4. Participant 3-day data collection schema.



4.6 Calculation of Fat Oxidation Rate

Frayn's (1983) equation for the calculation of FOR was employed to measure substrate oxidation at each workload using indirect calorimetry. Urinary nitrogen excretion rate was assumed to be negligible for the purpose of the calculations. The equation is as follows:

$$\text{Fat } (g \cdot min^{-1}) = 1.67 * VO_2 \ (L \cdot min^{-1}) - 1.67 * VCO_2 \ (L \cdot min^{-1})$$

FOR was expressed relative to fat-free mass (FFM) and by body mass for each participant at each workload. The FOR relative to FFM were used to create the FOR curves for each participant and the analysis of resting and FOR_{max}.

4.7 Ethics Approval

Ethics approval was obtained from the Human Participants Review Sub-Committees of York University (Toronto, Ont.) before the initiation of the study. All the women provided written Informed Consent to their voluntary participation in the study.

4.8 Statistical Analysis

The study participant characteristics were expressed as means and standard deviations (X±SD). An independent t-test was used to determine differences between the ME and the W for the anthropometrics, menstrual characteristics, resting BP and HR, PA levels, blood lipid panel, blood hormone panel, glucose, resting VO_2, and VO_2 peak. A paired t-test was performed on the two trials of the fat oxidation trials to determine any differences between the two occasions. Regression analysis was performed on the all the hormones and other measured variables against WC and FOR for each group. Statistical analysis was conducted using a standard statistical software program, SPPS 20.0 (2012).

5.0 RESULTS

A total of 23 participants were recruited for this study, 12 in the W and 11 in the ME group. All were cleared during the pre-screening to participate in the study. Anthropometric, menstrual and age measures are shown in Table 4. Significant differences between the groups where observed at age of study participation the ME were younger (p= 0.004), ME observed their first period at an earlier age (p= 0.023) and had their last menstrual cycle early in life (p= 0.031). Menstrual age, time since first and last cycle was shorter in the ME group (p= 0.007), ME had a higher body mass (p= 0.027), a higher BMI (p= 0.32) and a larger WC (p= 0.005).

Table 4. Age, menstrual characteristics and body composition of the White and Middle Eastern study participants with t-test and p-values for differences between groups.

Characteristics	White (n=12; X±SD)	Middle Eastern (n=11; X±SD)	p-value
Age (yrs)	57.1± 3.3	52.6 ± 2.8	0.004*
Age of First Cycle (yrs)	12.2 ± 8.2	13.3 ± 1.1	0.023*
Age of Last Cycle (yrs)	51.1 ± 2.6	46.5 ± 5.7	0.031*
Menstrual Age (yrs)	39.0 ± 2.2	32.2 ± 5.6	0.007*
Height (cm)	161.5 ± 4.6	162.2 ± 5.9	0.738
Body mass (kg)	71.0 ± 12.1	84.0 ± 16.7	0.027*
BMI	27.0 ± 3.8	31.8 ± 5.9	0.032*
NIH Waist Circumference (cm)	90. 9 ± 11.0	106.1 ± 11.2	0.005*
Sum of 5 Skinfolds (mm)	107.5 ± 35.6	136.1 ± 49.3	0.146
Tanita Foot Scale Body Fat (%)	37.0 ± 5.4	41.7 ± 6.4	0.085

**significant difference between groups*

Table 5 contains a summary of the cardiometabolic measures for both groups. Differences between the groups were only detected in the relative VO_2 peak measurement (p= 0.003) which was higher in the W group. Accelerometer and self- reported measurements; step counts, EE, activity counts and scores on the HPAPQ are summarized in Table 6. The objective measurements from the Actical accelerometers did not show significant differences between the

groups. The self-reported HPAPQ demonstrated that the W group had a higher perception of their fitness and PA level (p= 0.017).

Table 5. Cardiometabolic characteristics for the White and Middle Eastern study participants with t-test and p-values for differences between groups

Characteristics	White (n=12; X±SD)	Middle Eastern (n=11; X±SD)	p-value
Resting Blood Pressure Left Arm (mmHg)	109/72 ± 12/11	114/72 ± 13/9	0.427 / 0.854
Resting Blood Pressure Right Arm (mmHg)	113/72 ± 16/9	113/74 ± 14/10	0.901 / 0.747
Resting Heart Rate (bpm)	67 ± 7.2	72 ± 12	0.199
Resting VO_2 Relative ($mLO_2 \cdot min^{-1} \cdot kg$ of body mass)	3.63 ± 1.05	3.90 ± 0.83	0.527
Resting VO_2 Absolute ($LO_2 \cdot min^{-1}$)	257.0 ± 0.085	0.309 ± 0.047	0.097
VO_2 peak Relative ($mLO_2 \cdot min^{-1} \cdot kg$ of body mass)	28.16 ± 5.09	21.45 ± 3.79	0.003*
VO_2 peak Absolute ($LO_2 \cdot min^{-1}$)	1978.0 ± 0.344	1.776 ± 0.343	0.195
Peak Heart Rate (bpm)	164 ± 9	158 ± 10	0.131

**significant difference between groups.*

Table 6. Physical activity measurement for the White and Middle Eastern study participants with t-test and p-values for differences between groups .

Characteristics	White (n=12; X±SD)	Middle Eastern (n=11; X±SD)	p-value
Step Count (steps)	10,205 ± 5 387	7,823 ± 3 304	0.220
Total Energy Count (kcal)	725.5 ± 571.4)	762.0 ± 578.0	0.880
Activity Count (intensity)	172 135 ± 101 216	130 038 ± 68 103	0.260
HPAPQ	9.3 ± 2.1	6.2 ± 3.5	0.017*

**significant difference between groups.*

The FOR was calculated using the VO_2 and VCO_2 measured at each work load. The calculated FOR and the corresponding % of VO_2 peak were used to create the curves on the GraphPad PRISIM 5 computer software. Following standard procedures, measured resting values were not included in the creation of the curves, because the calculated resting FOR were negative values (Chui, 2011). A negative FOR is not biological possible, this has occurred as a result of

hyperventilation during the resting gas collection. FOR and $\%VO_2$ peak for each workload were used to graph the curves, FOR was graphed along the y-axis and % of VO_2 peak along the x-axis. Participants' FOR_{max} values and FATmax for both trials of the protocol are shown in Table 7. A second trial of the fat oxidation protocol for one participant in the ME group was not obtained due to inability to complete the full protocol and therefore the final number of participants used in the analysis was 22. Figure 5. depicts the FOR_{max} and $\%VO_2$ peak for trials 1 and 2. A mean FOR_{max} of 7.89 ($g \bullet min^{-1} \bullet kg$ of fat free mass) was attained for trial 1 and 8.53($g \bullet min^{-1} \bullet kg$ of fat free mass) for trial 2 (not significantly different, NS). FATmax was attained at 46.2 % VO_2 peak for trial 1 and at 47.3 $\%VO_2$ peak for trial 2 (NS). The strength of the relationship between the two trials for FOR_{max} is $R^2=0.829$ ($p<0.00$) and $\%VO_2$ peak is $R^2=0.333$ ($p=0.13$). No differences were detected between trials, when comparing FOR_{max} of ME and W women. Since no differences were detected from one trial to the next, the 2 trials were then compiled and fitted to a single curve for each participant to allow for more data points to create a best-fit polynomial curve.

When the data were compiled from both trials, the curves were fitted to a third order polynomial. The more commonly used second order polynomial resulted in lower R^2 values for the ME women. The R^2 represents the fit of the curve to the actual data points. Most curves from the ME women had a R^2 value of less than 0.5, indicating that the points did not fit well to a second order polynomial curve. The R^2 0.5 is the accepted cut-off point that has been used in previous studies measuring FOR and fitting them to curves (Chui, 2011). When the data points were fit to a third order polynomial for the ME the R^2 values were significantly higher ($p=0.048$). There were no significant differences between the second and third order polynomials R^2 values for the W women ($p=0.683$). The associated tables and statistics are found in Appendix F.

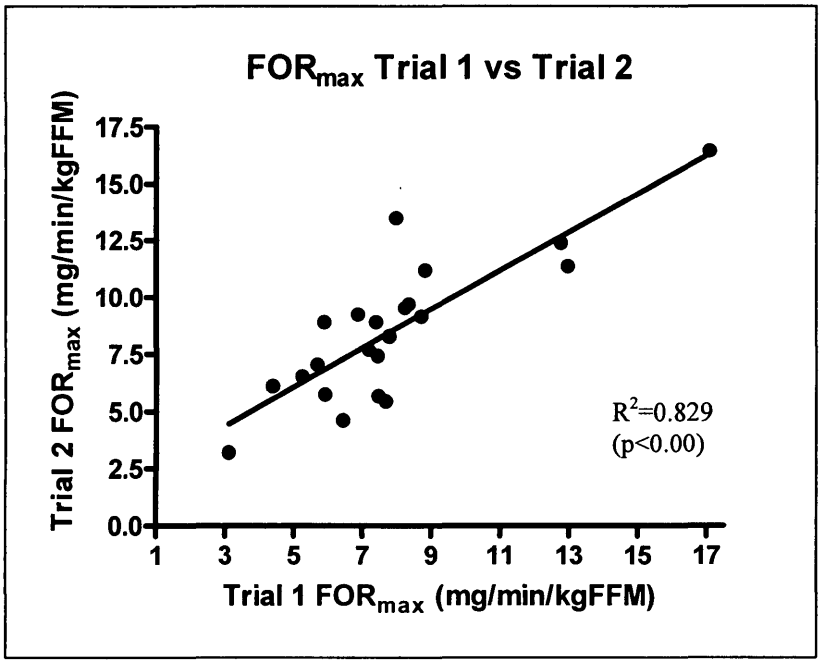
The FOR_{max} and FATmax as well as t-test scores for both groups are shown in Table 8. To maintain consistency with previous researchers who have studied FOR, only curves that had a R^2 value greater than 0.5 were included in the analysis (Chui, 2011). No significant differences

were detected between groups for FOR_{max} or FATmax. FOR_{max}, FATmax and exercise intensities that elicited FOR at 5%, 10%, and 20% below the FOR_{max} were determined for each participant's curve. These specific points were then averaged and plotted against the average FOR to provide a range of exercise intensities that elicit the highest FOR for each group. Curves were then compiled for each group and plotted to provide average: FOR_{max}, FOR at 5%, 10%, and 20% of FOR_{max} and the associated exercise intensities, as well as FATmax, along with the variance bars for both groups (Figure 6).

Table 7. Trials 1 and 2 of FOR_{max} and FATmax with paired t-test p-values for differences between the trials.

Characteristics	Trial 1 (n=22; X±SD)	Trial 2 (n=22; X±SD)	p-value
FOR_{max} ($g \bullet min^{-1} \bullet kg$ of fat free mass)	7.89 ± 3.04	8.53 ± 3.12	0.112
FATmax (% of VO_2 peak)	46.2 ± 11.6	47.3 ± 11.3	0.717

Figure 5. Reproducibility of FOR_{max} and FATmax from trial 1 to trial 2
A) FOR_{max}



B) FATmax

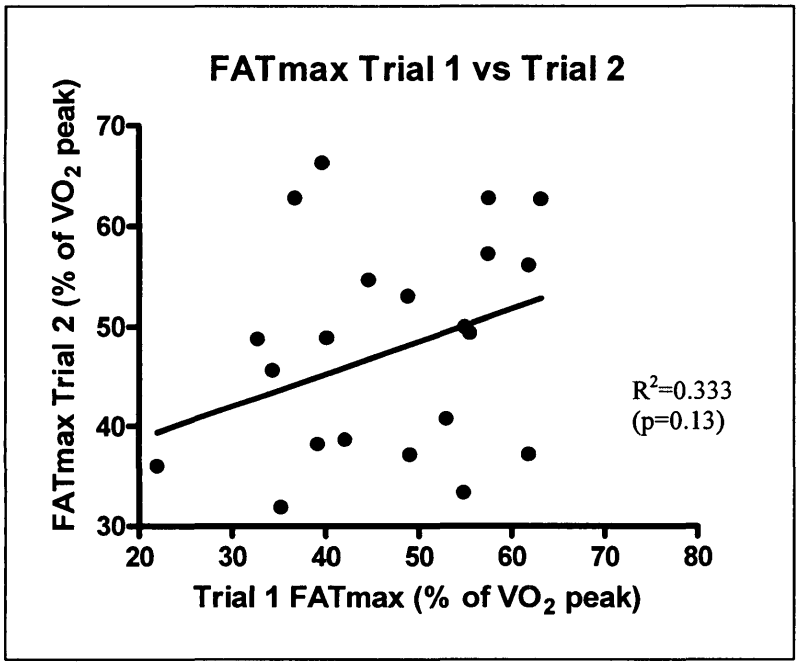
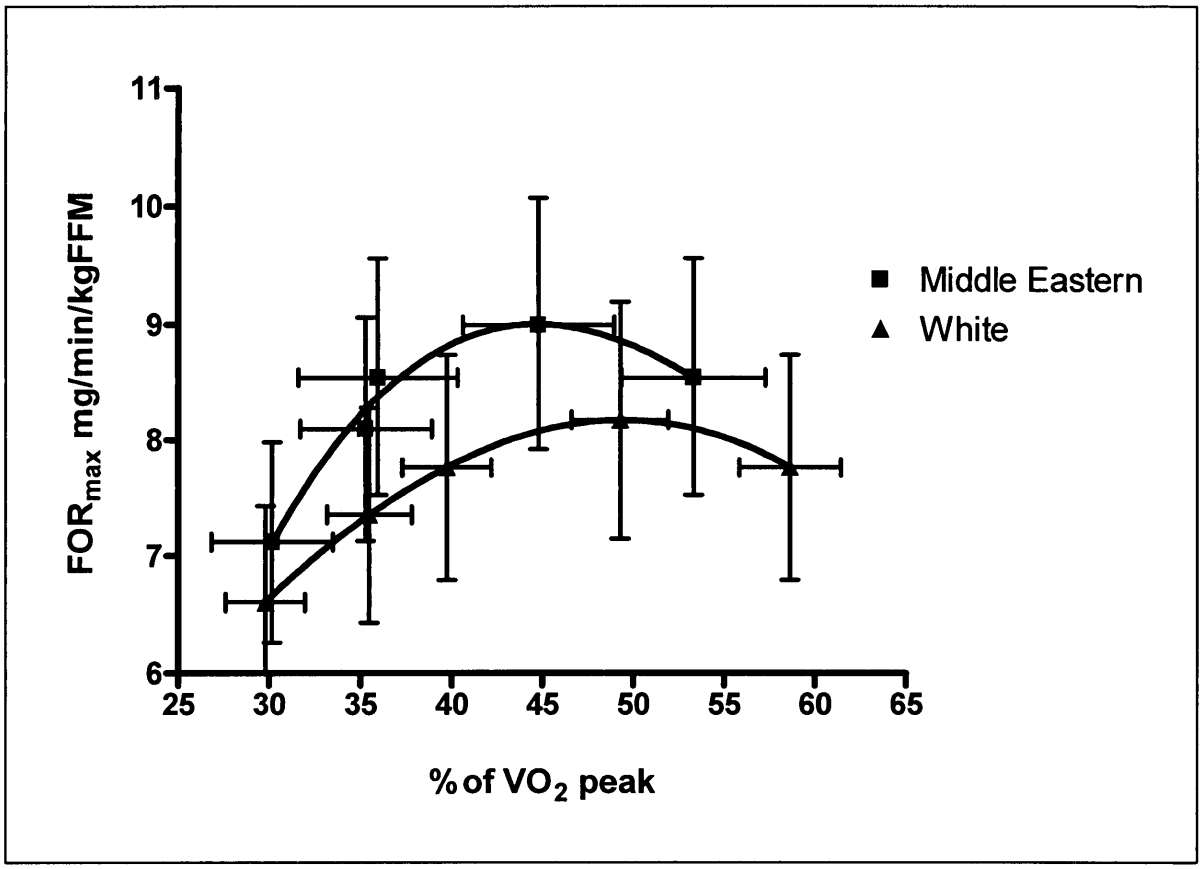


Table 8. Average fat oxidation characteristics for the White and Middle Eastern women and t-test p-values for differences between groups.

Characteristics	White (n=12; X±SD)	Middle Eastern (n=11; X±SD)	p-value
Average FOR_{max} ($g \bullet min^{-1} \bullet kg$ of fat free mass)	8.17±(3.24)	9.0 (3.22)	0.587
Average FATmax (% VO_2 peak)	49.3(8.41)	44.8 (12.56)	0.367

Figure 6. Fat oxidation rates for White and Middle Eastern participants at FOR_{max} and at +95%, -95%, -90%, -80% of FOR_{max} with its associated $\%VO_2$ peak.



The hormonal and blood profiles are shown in Table 9 along with select laboratory normative data (Canadian Life Labs) for postmenopausal women. A complete blood sample was not available for 1 participant in the ME group. For some variables there is a n=11 and the remainder have a n=10, this has been indicated in the Table. Significant differences were not detected between the two groups, indicating that there are no hormonal variations between these two ethnicities. Some of the laboratory tests had low sensitivity, therefore accurate values were not provided for estrone and progesterone, instead a range was provided. This inaccuracy of the hormone values may have masked the ability to detect any significant disparity.

Table 9. Hormone, blood lipid results of the White and Middle Eastern participants with t-test and p-values for differences between groups, as well as laboratory normative values for postmenopausal women.

Characteristics	White (n = 12; X ± SD)	Middle Eastern (n = as indicated; X ± SD)	p-value	Laboratory normative values for postmenopausal women
Estradiol (pmol/L)	84.93 ± 31.38	71.27 ± 4.55 (n=11)	0.163	< 120 pmol/L
Estrone (pmol/L)	138.25 ± 65.45	155.00 ± 52.74 (n=10)	0.522	52–379 pmol/L
Progesterone (nmol/L)	1.08 ± 0.29	1.36 ± 0.68 (n=11)	0.224	< 3 nmol/L
Testosterone (nmol/L)	1.33 ± 0.60	1.12 ± 0.63 (n=10)	0.421	N/A
Follicular Stimulating Hormone (IU/L)	85.0 ± 26.9	68.6 ± 28.9 (n=10)	0.184	> 35 IU/L
Luteinizing hormone (IU/L)	38.8 ± 12.3	33.4 ± 11.2 (n=10)	0.304	40–105 IU/L
Sex Hormone-binding Globulin (nmol/L)	45.04 ± 15.96	43.84 ± 21.91 (n=1)	0.883	18.0–144.0 nmol/L
Androgen Index	0.213 ± 0.131	0.146 ± 0.118 (n=10)	0.225	N/A
Cortisol (nmol/L)	715.58 ± 1436.90	190.09 ± 100.50 (n=11)	0.232	N/A
Insulin (pmol/L)	46.08 ± 20.81	64.90 ± 50.98 (n=11)	0.275	N/A
Glucose, fasting (nmol/L)	5.33 ± 0.49	5.39 ± 0.80 (n=10)	0.840	3.6 – 6.0 nmol/L
Total Cholesterol (nmol/L)	5.53 ± 1.32	5.08 ± 1.21 (n=10)	0.425	N/A
LDL (nmol/L)	3.42 ± 1.15	3.12 ± 0.78 (n=10)	0.502	< 2.0 nmol/L
HDL (nmol/L)	1.57 ± 0.35	1.28 ± 0.57 (n=10)	0.166	N/A
CH/LDL ratio	3.69 ± 1.16	3.98 ± 1.03 (n=10)	0.548	N/A
Triglyceride (nmol/L)	1.21 ± 0.61	1.33 ± 0.64 (n=10)	0.657	< 1.70 nmol/L

6.0 DISCUSSION

The purpose of this study was to compare FOR at rest and during incremental exercise, FOR_{max}, FATmax, hormone levels, anthropometric characteristics, PA levels and cardiometabolic risk factors between postmenopausal ME and W women. It was hypothesized that the ME women would exhibit lower FOR at rest and at different sub-maximal exercise intensities and therefore have a lower FOR_{max} and FATmax as well as, have lower levels of estrogen and posses higher cardiometabolic risk factors compared to the W women. In contrast to the hypothesis, no between group differences were observed between resting FOR, FOR during incremental exercise, FOR_{max}, FATmax, hormone levels, height, body fat%, skinfold measurements, PA levels, resting BP, resting HR, resting VO_2, exercise HR max, fasting blood glucose and fasting blood lipid profile. If anything, FOR tended to be higher in the ME group compared to the W group (see Figure 6). However, small differences were observed in BMI, body mass, WC, age at first and last menstrual cycle, age at study participation and VO_2 peak.

6.1 Anthropometrics and Physical Activity

The ME women were found to be significantly younger than the W women, although all participants in both groups were postmenopausal. The participants in the groups were essentially a convince sample of postmenopausal women which resulted in this unplanned difference. The ME women had their first menstrual cycle at a later age and experienced their last menstrual cycle at an earlier age compared to the W women. These findings coincide with the epidemiological study by Memon et al. (2002) in which they examined reproductive, hormonal characteristics and incidence of thyroid cancer in ME women. The authors reported that ME women experienced their first and last menstrual cycle at an average age of 13.2 years and 45.8 years respectively. These two values provide information on menstrual age which is the time between first and last menstrual cycle. According to the Memon et al. (2002) study their calculated menstrual age is

32.6 years, which is very close to the findings of this current study of 32.2 years. Little has been reported on menstrual age in the literature. This variable quantifies the length of reproductive hormone exposure in women, since estrogen, progesterone and testosterone have all been linked to influencing lipid metabolism this variable may be valuable in future assessments of disease risk in menopausal women.

The results also demonstrated that the ME women were heavier, had a higher BMI and a larger WC. As mentioned before in the review of literature, a large percentage of women in the Iranian population between the ages 55-64 are considered either overweight, obese or abdominally obese, as per the WHO BMI and WC guidelines (Janghorbani et al. 2007). The findings of this study are in accordance with the results of previous studies.

PA levels were measured objectively through the use of accelerometers and were also measured subjectively using the self-reported HPAPQ tool. The accelerometers did not detect any significant differences between groups. Step counts were comparable as well as EE. Activity counts, the measure of the exercise intensity, was slightly higher and more variable in the W women compared to the ME women but not significantly different. Based upon the HPAPQ, the W women generally took part in more intense physical activities, which may have contributed to their healthier body mass, BMI, WC and a higher VO_2 peak, even though overall EE is not different between groups. The HPAPQ asks questions about the frequency and intensity of PA plus the respondent's perceived fitness. Depending on the response to the questions, points are allocated accordingly higher score is representative of a higher perceived fitness and PA levels. The HPAPQ can be found in Appendix C. The W women scored higher on the HPAPQ and reported to participate in higher levels of PA and at a higher intensity then the ME women. As well, they had an elevated perception of their physical fitness. This can be related back to the results of the accelerometer which suggested that the W women tend to participate in regular PA for health benefits, while ME women tend to participate in PA for leisure purposes only and not

for the attainment of health benefits. The ME women may not perceive the activities that they participate in (household chores, walking etc.) as activities that are beneficial for their health and as a result underestimate their PA levels.

The accelerometer data revealed that the W women participated in higher intensity activities, even though the total step count and EE between groups were similar. The higher activity values that were obtained represented a higher frequency and intensity of PA participation. Upper and lower limits for what was considered low, moderate or high intensity was not provided by the accelerometer manufacture. A simple comparison between the data has been made which has shown that W women have a higher activity count than ME women, leading to the conclusion that W women take part in higher intensity PA than ME women. Another finding from this study was that ME women subjectively rated their PA and fitness levels lower than the W women. This illustrates that ME women do not perceive themselves as being healthy and physically fit. This may be related to the idea that ME women are aware that they do not take part in organized PA for the purpose of improving their physical fitness.

Al-Nozha et al. (2007) measured activity levels in the Saudi population, a sub-group of ME, and reported that 98.2-99.1 % of women between the ages of 50-70 years of age were inactive based on minimum of 30 minutes of moderate intensity PA at least 3x week (Haskell et al. 2007, Warburton et al. 2007, Tremblay et al. 2011, WHO, 2010). Al-Nozah et al. did not speculate on the low PA participation. Merom et al. (2012) compared the activity levels between Palestinian and Israeli men and women, and concluded that Palestinian women expended less energy during moderate to vigorous PA compared to the Israeli women, therefore implying that Palestinian women participate in lower levels of moderate to vigorous PA in comparison to Isreali women. Palestinian women received most of their PA from household chores and light to moderate intensity walking which was reported as their leisure-time PA (Merom et al. 2012). It can be seen from these two studies that ME women do not expend much energy in moderate to

vigorous intensity PA, which supports the findings from the present study. Walking and household chores are the two most common forms of PA that ME women engage in.

6.2 Cardiometabolic Characteristics

Resting BP, HR, VO_2 and exercise HR peak and VO_2 peak were measured for each participant. Resting measurements were similar between the groups. No significant differences were observed among absolute VO_2 peak and HR peak. Relative VO_2peak was significantly different between the two groups. The measured VO_2 peak values in this study are comparable to the VO_2 values measured in other studies using similar study participants. Johnson et al. (2010) measured a relative VO_2 peak of 25.5 $mLO_2 \cdot min^{-1} \bullet kg$ in postmenopausal women, which falls in the middle of the VO_2 peak values of the ME and W groups in the present study. The values from Johnson et al. (2010) coincide with the measured values from Numao et al. (2009) and Zarins et al. (2009) VO_2 peak of 25.3 $mLO_2 \cdot min^{-1} \bullet kg$ and 25.5 $mLO_2 \cdot min^{-1} \bullet kg$ respectively.

The peak exercise HR attained during the incremental to maximum exercise test in the present study demonstrated that the ME women were not able to reach their calculated age-predicted max HR whereas the W women did. The mean age of the ME women was lower and therefore their calculated age-predicted max HR is higher. The ME group had a measured HR peak of 158 beats per minute (bpm) while the mean calculated (220-age) HR max was 167 bpm, the W group had a measured HR peak of 164 bpm and their estimated HR max was 163 bpm. This demonstrates that the W group was able to tolerate higher intensity activity which is supported by the results of the PA measurements and participation in higher intensity PA.

6.3 Hormone and Lipid Panel

A wide variety of sex hormones were measured and compared in this study and no hormonal differences were detected between groups. Studies that have measured sex hormone levels in women pre and postmenopause have mainly been breast cancer research studies.

Researchers that have looked at the ethnicity and breast cancer risk have measured sex hormone levels and compared them between ethnicities. These studies that measured sex hormone levels, observed ethnic differences in estradiol, testosterone, FSH and SHBG both pre and postmenopausal stages (Randolph et al. 2004, Santoro et al. 2004, Setiawan et al. 2006, Sowers et al. 2006, Sutton-Tyrell et al. 2010). Ethnic groups that had been studied include White, Hispanic, Chinese, Japanese and African American women. ME women have not been studied and therefore the hormone measurements that were obtained in the current study contribute new information to the body of literature on women's reproductive health and menopausal transition.

When comparing the hormone and lipid values of the ME and W women to that of the laboratory normative data, all of the measurements fall within the acceptable range with the exception of LH and LDL-CH levels. LH levels fell below the normal range; the measured values are being compared to the normative values that Canadian Life Labs uses. The values and ranges set by this laboratory are specific to this lab and may differ to the normative values of other laboratory (Canadian Life Labs). HDL-CH levels are again elevated above normal for both the ME and W groups. The normative values for the lipid panel are not just applicable to menopausal women but these values apply to persons of all ages and sex. This study was cross-sectional in nature, which only provided a snapshot picture of the lipid profile of the study participants. Premenopausal values for the lipid measurement were not obtained and therefore not available to compare current values to. This comparison would provide a better understanding of the changes that may occur as a result of the menopausal transition on lipid profile.

6.4 Fat Oxidation

Contradictory to the hypothesis, differences were not observed in resting FOR, FOR at varying sub-maximal exercise intensities or FOR_{max}. This study acquired data on the FOR of W

and ME women over a wide range of exercise intensities that allowed for the data to be fit to a curve and provide information on FOR, FOR_{max} and FATmax. Once again, to our knowledge, no other study has investigated FOR in ME postmenopausal women.

The FATmax value obtained from the current study of 49.3% VO_2 peak and 44.8% of VO_2 peak for the W and ME women respectively do not coincide with the FATmax values that are similar to what has been reported in other studies of sedentary W females that utilized a treadmill protocol (reference). In younger and more fit populations, Atchen et al. (2003) had reported a FATmax at 59.2% of VO_2 peak and Chenevière et al. (2010) at 57.2% of VO_2 peak in treadmill exercise. The treadmill protocols used by Atchen and Chenevière differed slightly from the protocol that was employed by the current study and the participants in those studies were different. The study participants from the previous studies were healthy young men and women that had healthy body mass and BF. As mentioned in the review of literature, age and gender are influential factors of FOR. The FATmax from the current study with the FATmax of the previous studies cannot be compared due to inconsistency of the protocol and study participants utilized in the studies. FOR_{max} had not been measured during incremental to maximum exercise in postmenopausal women until the current study. Other studies (Treuth et al. 1995, Zarins et al. 2009, Johnson et al. 2010) that have examined FOR in postmenopausal women have only measured FOR at rest or at single % VO_2 peak. This has made it difficult to compare the results on FOR, FOR_{max} and FATmax of the current study to that of others thus making this study novel, in providing detailed information on FOR in ME women.

7.0 CONCLUSION

In summary, this study contributed to the ever-growing body of literature on fat oxidation and women's health by exploring an ethnic group that has not been studied previously. Significant differences were not observed between resting FOR, sub-maximal exercise FOR, FORmax , hormone levels, height, body fat%, skinfold measurements, HPAPQ, resting BP, resting HR, resting VO_2, HR max, fasting blood glucose and fasting blood lipid panel. Differences were observed in BMI, body mass, WC, age at first and last menstrual cycle, age at study participation and VO_2 peak. As a result of this research, new information was reported on FOR and hormonal characteristics for ME and W postmenopausal women. The current study is one of the few that utilized a treadmill protocol to measure and calculate FOR over a wide range of sub-maximal exercise intensities and provide values on FOR_{max} and FATmax. In addition, this study is the first to examine FOR and FATmax in ME women and to measure the hormones that may impact fat mobilization and oxidation during exercise

The next step in addressing the cardiometabolic risk factors that seem to linger among ME women is by addressing preventative strategies to reduce risk development. This study did reveal the differences in PA participation, W women participated in higher intensity activities than ME women. Regular moderate intensity PA has been associated with health benefits and high intensity with greater health benefits (Janssesn and LeBlanc, 2010) in body composition, blood lipid profile and BP (Gledhill and Jamnik, 2003). Encouraging ME women to take part in regular PA for the purpose of improving their overall physical fitness and health with allow for improvements in risk development. Developing cultural-specific PA strategies will allow ME women to freely participate and incorporate regular PA into their lifestyles. As mentioned before, regular PA for the purpose of improving one's health and physical fitness will allow for risk reductions in cardiometabolic diseases.

This study has some limitations that should be noted. First, the sample size was small and this may have had an influence on the lack of significance of the results. With the results of this study if one were to perform a power analysis, in order to attain power at the 0.05 level for a two-tailed alpha with an effect size of 0.8, the number of participants in each group would have to be 60. Second, the participants were chosen at random and were not matched for any variables, which could have confounded the results. And lastly, diet was not recorded or controlled for which does have an influence in the outcomes of the fat oxidation protocol.

This research study has shed light on FOR within an ethnic group of postmenopausal women who have not previously been investigated. As well, information was provided on FOR, FOR_{max}, FATmax using a treadmill protocol and hormonal measurements for ME and W postmenopausal women. A treadmill protocol was created and used which provided whole-body FOR at multiple sub-maximal PA intensities. PA levels and anthropometrics were also obtained in this study providing detailed data on the health and fitness characteristics of the study populations. The current study was exploratory in nature and offers a concrete basis for future research. Expansion on this study will provide a more complete picture of how the menopausal transition affects FOR at rest and during exercise in ME and W women. Following ethnic women longitudinally over the menopausal transition tracking FOR at sub-maximal PA intensities, sex hormone levels, PA levels and anthropometric characteristics would provide a better understanding of how the menopausal transition influences a women's physiology and disease risk.

APPENDIX A. Physical Activity Readiness Questionnaire for Everyone.

PAR-Q+

The Physical Activity Readiness Questionnaire for Everyone

Regular physical activity is fun and healthy, and more people should become more physically active every day of the week. Being more physically active is very safe for MOST people. This questionnaire will tell you whether it is necessary for you to seek further advice from your doctor OR a qualified exercise professional before becoming more physically active.

SECTION 1 - GENERAL HEALTH

	Please read the 7 questions below carefully and answer each one honestly: check YES or NO.	YES	NO
1.	Has your doctor ever said that you have a heart condition OR high blood pressure?	☐	☐
2.	Do you feel pain in your chest at rest, during your daily activities of living, OR when you do physical activity?	☐	☐
3.	Do you lose balance because of dizziness OR have you lost consciousness in the last 12 months? Please answer NO if your dizziness was associated with over-breathing (including during vigorous exercise).	☐	☐
4.	Have you ever been diagnosed with another chronic medical condition (other than heart disease or high blood pressure)?	☐	☐
5.	Are you currently taking prescribed medications for a chronic medical condition?	☐	☐
6.	Do you have a bone or joint problem that could be made worse by becoming more physically active? Please answer NO if you had a joint problem in the past, but it does not limit your current ability to be physically active. For example, knee, ankle, shoulder or other.	☐	☐
7.	Has your doctor ever said that you should only do medically supervised physical activity?	☐	☐

If you answered NO to all of the questions above, you are cleared for physical activity.

Go to Section 3 to sign the form. You do not need to complete Section 2.

- Start becoming much more physically active – start slowly and build up gradually.
- Follow the Canadian Physical Activity Guidelines for your age (www.csep.ca/guidelines).
- You may take part in a health and fitness appraisal.
- If you have any further questions, contact a qualified exercise professional such as a CSEP Certified Exercise Physiologist® (CSEP-CEP).
- If you are over the age of 45 yrs. and NOT accustomed to regular vigorous physical activity, please consult a qualified exercise professional (CSEP-CEP) before engaging in maximal effort exercise.

If you answered YES to one or more of the questions above, please GO TO SECTION 2.

Delay becoming more active if:

- You are not feeling well because of a temporary illness such as a cold or fever – wait until you feel better
- You are pregnant – talk to your health care practitioner, your physician, a qualified exercise professional, and/or complete the PARmed-X for Pregnancy before becoming more physically active OR
- Your health changes – please answer the questions on Section 2 of this document and/or talk to your doctor or qualified exercise professional (CSEP-CEP) before continuing with any physical activity programme.

SECTION 2 - CHRONIC MEDICAL CONDITIONS

Please read the questions below carefully and answer each one honestly: check YES or NO.			YES	NO
1	Do you have Arthritis, Osteoporosis, or Back Problems?		☐ If yes, answer questions 1a-1c	☐ If no, go to question 2
	1a.	Do you have difficulty controlling your condition with medications or other physician-prescribed therapies? (Answer NO if you are not currently taking medications or other treatments)	☐	☐
	1b.	Do you have joint problems causing pain, a recent fracture or fracture caused by osteoporosis or cancer, displaced vertebra (e.g., spondylolisthesis), and/or spondylolysis/pars defect (a crack in the bony ring on the back of the spinal column)?	☐	☐
	1c.	Have you had steroid injections or taken steroid tablets regularly for more than 3 months?	☐	☐
2	Do you have Cancer of any kind?		☐ If yes, answer questions 2a-2b	☐ If no, go to question 3
	2a.	Does your cancer diagnosis include any of the following types: lung/bronchogenic, multiple myeloma (cancer of plasma cells), head, and neck?	☐	☐
	2b.	Are you currently receiving cancer therapy (such as chemotherapy or radiotherapy)?	☐	☐
3	Do you have Heart Disease or Cardiovascular Disease? This includes Coronary Artery Disease, High Blood Pressure, Heart Failure, Diagnosed Abnormality of Heart Rhythm		☐ If yes, answer questions 3a-3e	☐ If no, go to question 4
	3a.	Do you have difficulty controlling your condition with medications or other physician-prescribed therapies? (Answer NO if you are not currently taking medications or other treatments)	☐	☐
	3b.	Do you have an irregular heart beat that requires medical management? (e.g. atrial fibrillation, premature ventricular contraction)	☐	☐
	3c.	Do you have chronic heart failure?	☐	☐
	3d.	Do you have a resting blood pressure equal to or greater than 160/90 mmHg with or without medication? (Answer YES if you do not know your resting blood pressure)	☐	☐
	3e.	Do you have diagnosed coronary artery (cardiovascular) disease and have not participated in regular physical activity in the last 2 months?	☐	☐
4	Do you have any Metabolic Conditions? This includes Type 1 Diabetes, Type 2 Diabetes, Pre-Diabetes		☐ If yes, answer questions 4a-4c	☐ If no, go to question 5
	4a.	Is your blood sugar often above 13.0 mmol/L? (Answer YES if you are not sure)	☐	☐
	4b.	Do you have any signs or symptoms of diabetes complications such as heart or vascular disease and/or complications affecting your eyes, kidneys, and the sensation in your toes and feet?	☐	☐
	4c.	Do you have other metabolic conditions (such as thyroid disorders, pregnancy-related diabetes, chronic kidney disease, liver problems)?	☐	☐
5	Do you have any Mental Health Problems or Learning Difficulties? This includes Alzheimers, Dementia, Depression, Anxiety Disorder, Eating Disorder, Psychotic Disorder, Intellectual Disability, Down Syndrome)		☐ If yes, answer questions 5a-5b	☐ If no, go to question 6
	5a.	Do you have difficulty controlling your condition with medications or other physician-prescribed therapies? (Answer NO if you are not currently taking medications or other treatments)	☐	☐
	5b.	Do you also have back problems affecting nerves or muscles?	☐	☐

Please read the questions below carefully and answer each one honestly: check YES or NO.			YES	NO
6	Do you have a Respiratory Disease? This includes Chronic Obstructive Pulmonary Disease, Asthma, Pulmonary High Blood Pressure		☐ If yes, answer questions 6a-6d	☐ If no, go to question 7
	6a.	Do you have difficulty controlling your condition with medications or other physician-prescribed therapies? (Answer NO if you are not currently taking medications or other treatments)	☐	☐
	6b.	Has your doctor ever said your blood oxygen level is low at rest or during exercise and/or that you require supplemental oxygen therapy?	☐	☐
	6c.	If asthmatic, do you currently have symptoms of chest tightness, wheezing, laboured breathing, consistent cough (more than 2 days/week), or have you used your rescue medication more than twice in the last week?	☐	☐
	6d.	Has your doctor ever said you have high blood pressure in the blood vessels of your lungs?	☐	☐
7.	Do you have a Spinal Cord Injury? This includes Tetraplegia and Paraplegia		☐ If yes, answer questions 7a-7c	☐ If no, go to question 8
	7a.	Do you have difficulty controlling your condition with medications or other physician-prescribed therapies? (Answer NO if you are not currently taking medications or other treatments)	☐	☐
	7b.	Do you commonly exhibit low resting blood pressure significant enough to cause dizziness, light-headedness, and/or fainting?	☐	☐
	7c.	Has your physician indicated that you exhibit sudden bouts of high blood pressure (known as Autonomic Dysreflexia)?	☐	☐
8.	Have you had a Stroke? This includes Transient Ischemic Attack (TIA) or Cerebrovascular Event		☐ If yes, answer questions 8a-c	☐ If no, go to question 9
	8a.	Do you have difficulty controlling your condition with medications or other physician-prescribed therapies? (Answer NO if you are not currently taking medications or other treatments)	☐	☐
	8b.	Do you have any impairment in walking or mobility?	☐	☐
	8c.	Have you experienced a stroke or impairment in nerves or muscles in the past 6 months?	☐	☐
9.	Do you have any other medical condition not listed above or do you live with two chronic conditions?		☐ If yes, answer questions 9a-c	☐ If no, read the advice on page 4
	9a.	Have you experienced a blackout, fainted, or lost consciousness as a result of a head injury within the last 12 months OR have you had a diagnosed concussion within the last 12 months?	☐	☐
	9b.	Do you have a medical condition that is not listed (such as epilepsy, neurological conditions, kidney problems)?	☐	☐
	9c.	Do you currently live with two chronic conditions?	☐	☐

Please proceed to Page 4 for recommendations for your current medical condition and sign this document.

PAR-Q+

If you answered NO to all of the follow-up questions about your medical condition, you are ready to become more physically active:

- It is advised that you consult a qualified exercise professional (e.g., a CSEP-CEP) to help you develop a safe and effective physical activity plan to meet your health needs.
- You are encouraged to start slowly and build up gradually – 20-60 min. of low- to moderate-intensity exercise, 3-5 days per week including aerobic and muscle strengthening exercises.
- As you progress, you should aim to accumulate 150 minutes or more of moderate-intensity physical activity per week.
- If you are over the age of 45 yrs. and NOT accustomed to regular vigorous physical activity, please consult a qualified exercise professional (CSEP-CEP) before engaging in maximal effort exercise.

If you answered YES to one or more of the follow-up questions about your medical condition:

- You should seek further information from a licensed health care professional before becoming more physically active or engaging in a fitness appraisal.

Delay becoming more active if:

- You are not feeling well because of a temporary illness such as a cold or fever – wait until you feel better
- You are pregnant - talk to your health care practitioner, your physician, a qualified exercise professional, and/or complete the PARmed-X for Pregnancy before becoming more physically active OR
- Your health changes - please talk to your doctor or qualified exercise professional (CSEP-CEP) before continuing with any physical activity programme.

SECTION 3 - DECLARATION

- You are encouraged to photocopy the PAR-Q+. You must use the entire questionnaire and NO changes are permitted.
- The Canadian Society for Exercise Physiology, the PAR-Q+ Collaboration, and their agents assume no liability for persons who undertake physical activity. If in doubt after completing the questionnaire, consult your doctor prior to physical activity.
- If you are less than the legal age required for consent or require the assent of a care provider, your parent, guardian or care provider must also sign this form.
- Please read and sign the declaration below:

I, the undersigned, have read, understood to my full satisfaction and completed this questionnaire. I acknowledge that this physical activity clearance is valid for a maximum of 12 months from the date it is completed and becomes invalid if my condition changes. I also acknowledge that a Trustee (such as my employer, community/fitness centre, health care provider, or other designate) may retain a copy of this form for their records. In these instances, the Trustee will be required to adhere to local, national, and international guidelines regarding the storage of personal health information ensuring that they maintain the privacy of the information and do not misuse or wrongfully disclose such information.

NAME _____ DATE _____

SIGNATURE _____ WITNESS _____

SIGNATURE OF PARENT/GUARDIAN/CARE PROVIDER _____

For more information, please contact:
Canadian Society for Exercise Physiology
www.csep.ca

KEY REFERENCES

1. Jamnik VJ, Warburton DER, Makarski J, McKenzie DC, Stephens RJ, Stone J, and Gledhill N. Enhancing the effectiveness of clearance for physical activity participation: background and overall process. APNM 36(3):533-543, 2011.
2. Warburton DER, Gledhill N, Jamnik VK, Bredin SSD, McKenzie DC, Stone J, Charlesworth S, and Stephens RJ. Evidence-based risk assessment and recommendations for physical activity clearance: Consensus Document. APNM 36(5):S266-s298, 2011.

The PAR-Q+ was created using the evidence-based AGREE process (1) by the PAR-Q+Collaboration chaired by Dr. Darren E. R. Warburton with Dr. Norman Gledhill, Dr. Veronica Jamnik, and Dr. Donald C. McKenzie (2). Production of this document has been made possible through financial contributions from the Public Health Agency of Canada and the BC Ministry of Health Services. The views expressed herein do not necessarily represent the views of the Public Health Agency of Canada or BC Ministry of Health Services.

APPENDIX B. Informed Consent Document.

Informed Consent

Ethnic Variations in Fat Oxidation Rates Among Postmenopausal Females at Rest and During Exercise

Veronica Jamnik PhD, Michael Riddell PhD, Faiza Abdullah MSc Candidate

Kinesiology and Health Sciences

Contact email: afaiza@yorku.ca Office phone: 416-736-2100 ext. 77236

Purpose of the research

The purpose of the research is to characterize the fat oxidation rates at rest and during exercise in postmenopausal females from the Middle Eastern and South Asian communities. The participants will undergo a series of incremental to maximal effort exercise tests to determine their: VO2 max, peak fat oxidation rates. Researchers have made the connection between a low fat oxidative capacity plus fat mass gain and an increased predisposition to cardiovascular and metabolic (eg diabetes) disease .We will be examining a number of hormones in an attempt to link the factors that influence the weight gain observed in postmenopausal females. Research has been conducted on fat oxidation rates of Caucasians and African-American females, but there is limited information on the Middle Eastern and South Asian populations, although related research findings suggest that these ethnic groups are likely to be anomalous and consequently at greater health risk. We hypothesize that the postmenopausal women in these ethnic communities have a diminished fat oxidation rates, contributing to the increase in fat mass, especially visceral fat mass experienced with menopause.

What you will be asked to do in the research

Once pre-exercise clearance has been obtained using the well-validated Physical Activity Readiness Questionnaire+ (PAR-Q+), the participants will attend the laboratory on 3 separate occasions to undergo exercise tests on the treadmill. Qualified exercise professionals (Canadian Society for Exercise Physiology-Certified Exercise Physiologist®: CSEP-CEP) will conduct the exercise tests.

Prior to the exercise tests each participant will have their resting blood pressure and heart rate measured. Body fat percentage will be measured on the Tanita Body Fat Scale, waist circumference, height and weight measurements will also be taken. Also, blood samples will be taken from the participants at the second session when they are in a fasted state by a certified phlebotomist who has medical delegation to characterize their blood lipid profile (HDL-cholesterol, LDL-cholesterol, triglycerides, total cholesterol, glucose) and hormone levels (estrogen, progesterone, testosterone, follicular stimulating hormone (FSH), luteinizing hormone (LH), insulin and cortisol).

Urine samples will also be obtained from the subject for comparison to hormone levels in the blood. The urine samples will be collected in laboratory containers while in a fasted state.

One of the exercise tests will determine the participant's Aerobic Power (VO_{2max}) and consists of an incremental to maximal effort graded exercise test on the treadmill, during which the participant will walk on the treadmill until she reaches volitional fatigue or until the tester asks

her to stop. This may take anywhere from 10-20 minutes in duration. The second and third treadmill tests are sub-maximal in nature and will be used to determine each participant's fat oxidation rate. The duration of the 2nd and 3rd treadmill tests is approximately 35 minutes. During the treadmill tests each participant will breathe into a mouthpiece and heart rate will be monitored via an electronic heart rate monitor.

Risks and discomforts

Taking part in any physical activity has some minor risks such as fatigue, soreness, or shortness of breath. To minimize these risks, participants will be required to complete a pre-screening PAR-Q+ and the physical activity will be supervised by exercise professionals (CSEP-CEP) who are qualified to conduct safe exercise tests on people with and without chronic diseases.

There is a very small risk of infection with the blood collection. All blood samples will be taken by experienced and qualified blood samplers (medical delegation) using sterile equipment and techniques. There may also be minor discomfort and/or bruising at the needle site.

Benefits of the research and benefits to you

The participants will better understand their health and the benefits of regular physical activity on their health. If unfamiliar, they will become accustomed to walking on a treadmill. They will be given information on how to stay physically active and on how to adopt a healthy lifestyle.

Voluntary participation

Your participation in the study is completely voluntary and you may choose to stop participating at any time. Your decision not to volunteer will not influence the relationship you may have with the researchers or study staff or the nature of your relationship with York University either now, or in the future.

Withdrawal from the study

You can stop participating in the study at any time, for any reason, if you so decide. Your decision to stop participating, or to refuse to answer particular questions, will not affect your relationship with the researchers, York University, or any other group associated with this project. In the event you withdraw from the study, all associated data collected will be immediately destroyed wherever possible.

Confidentiality

All participants will remain anonymous and the results will only be reported in aggregate format. All hard copy data will be stored in a secure filing cabinet. All participants will be issued ID numbers. The participant's name and corresponding ID number will be securely store separately. Computer based data will be stored on a secure system with identification by ID number. No individual data will be reported in publications, and to meet scientific standards, the data will be kept for 5 years following the publication of the study results then destroyed.

All electronic data will be password protected and only research personnel will have access. Hard copies of the data will be destroyed using a confidential shredding agency five years after publication of the study results. Confidentiality will be provided to the fullest extent possible by law.

Questions about the research?

If you have any general questions or questions about your role in this study feel free to contact the researcher at the above email or phone number, or contact the supervisor Veronica Jamnik, PhD at ronij@yorku.ca or at 416-736-2100 ext. 22995.

This research has been reviewed and approved by the Human Participants Review Sub-Committee, York University's Ethics Review Board and conforms to the standards of the Canadian Tri-Council Research Ethics guidelines. If you have any questions about this process, or about your rights as a participant in the study, your may contact the Senior Manager and Policy Advisor for the Office of Research Ethics, 5th Floor, York Research Tower, York University, telephone 416-736-5914 or e-mail ore@yorku.ca

Legal rights and signatures:

I, consent to participate in Ethnic variations in fat oxidation rates among postmenopausal women at rest and during exercise conducted by Faiza Abdullah. I have understood the nature of this project and wish to participate. I am not waiving any of my legal rights by signing this form. My signature below indicates my consent.

Signature _____ **Date** _____
Participant

Signature _____ **Date** _____
Principal Investigator

APPENDIX C. Healthy Physical Activity Participation Questionnaire.

TOOL #21 HEALTHY PHYSICAL ACTIVITY PARTICIPATION QUESTIONNAIRE

DETERMINING THE HEALTH BENEFITS OF YOUR PHYSICAL ACTIVITY PARTICIPATION IS AS EASY AS A, B, C ...

A. Answer the following questions:

#1 Frequency

Over a typical seven-day period (one week), how many times do you engage in physical activity that is sufficiently prolonged and intense to cause sweating and a rapid heart beat?

- ☐ At least three times
- ☐ Normally once or twice
- ☐ Rarely or never

#2 Intensity

When you engage in physical activity, do you have the impression that you:

- ☐ Make an intense effort
- ☐ Make a moderate effort
- ☐ Make a light effort

#3 Perceived Fitness

In a general fashion, would you say that your current physical fitness is:

- ☐ Very Good
- ☐ Good
- ☐ Average
- ☐ Poor
- ☐ Very Poor

B. Circle your score for each answer and total your score.

Scoring of Questionnaire Responses

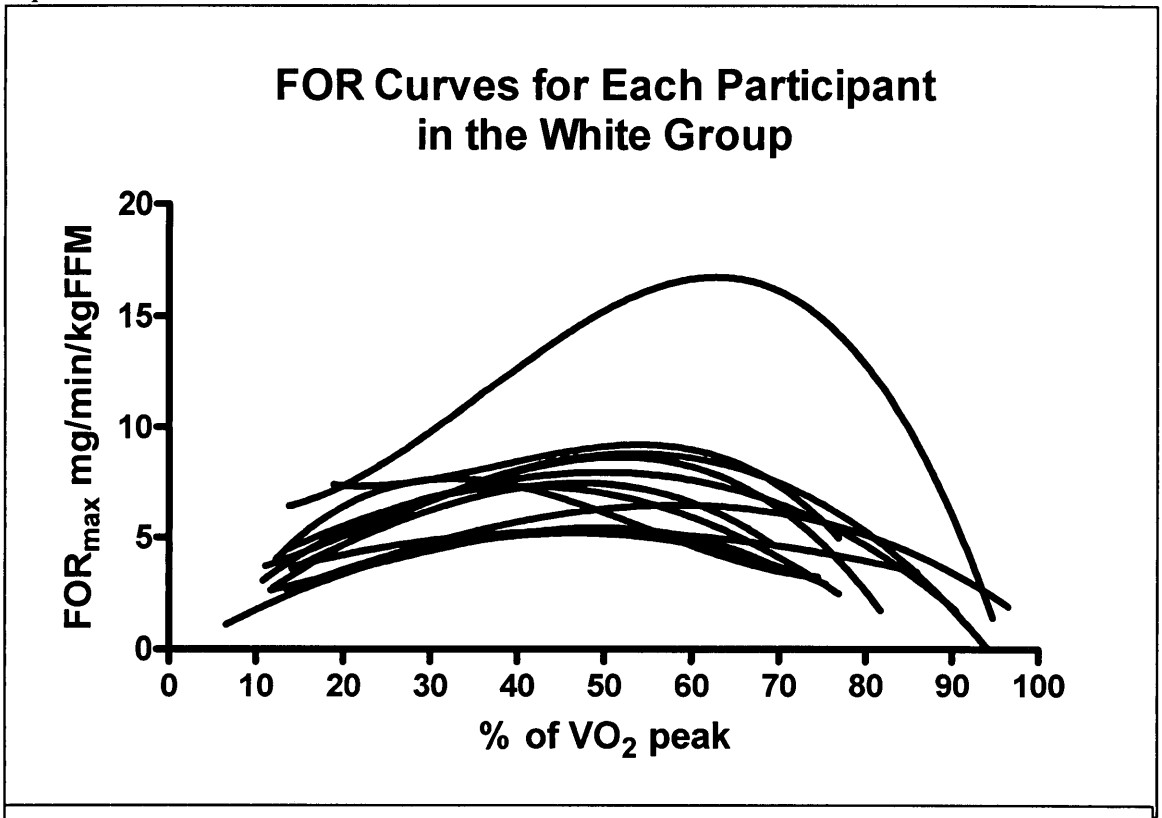
Item	Male	Female	Male	Female	Male	Female
#1 Frequency	Rarely or never		Normally once or twice		At least three times	
	0	0	2	2	5	5
#2 Intensity	Light effort		Moderate effort		Intense effort	
	0	0	1	2	5	5
#3 Perceived Fitness	Very Poor or Poor		Average		Good or Very Good	
	0	0	5	1	5	5

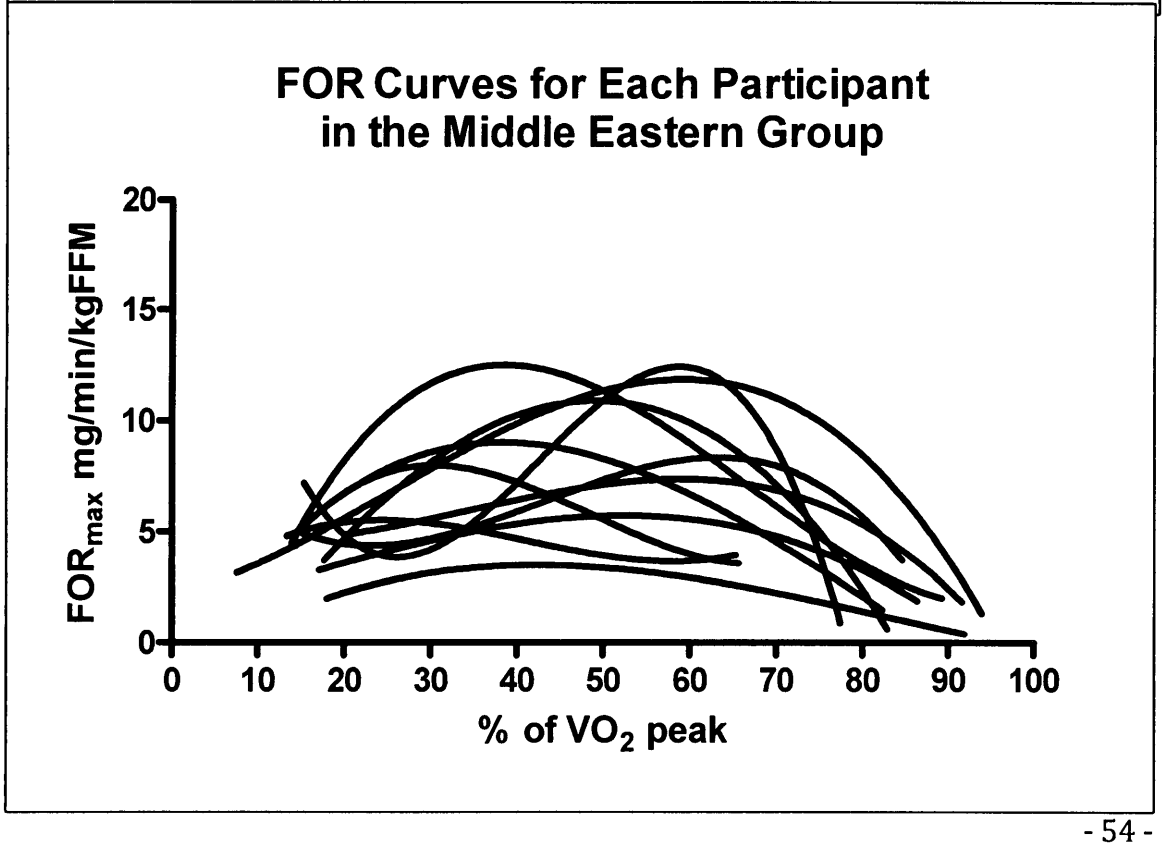
Total Score = _____

C. Determine your health benefit rating based on your score from B.

Health Benefit Zone	Total Score
Excellent	9–11
Very Good	6–8
Good	4–5
Fair	1–3
Needs Improvement	0

APPENDIX D. Individual fat oxidation rate curves for each participant compiled onto one graph, separate for Middle Eastern and White women.





APPENDIX E. Raw data measurements for each participant.

Participant	Age (years)	Age of first men cycle (years)	Age of last men cycle (years)	Height (cm)	BM (kg)	BMI	WC (cm)
White01	66	12	52	154	68.4	28.8	86
White02	57	11	50	150	51.6	22.9	86
White03	58	11.5	55	158.5	68.4	27.2	91.5
White04	52	11	50	159	68.1	26.9	86
White05	56	11	46	158	65	26.0	94.5
White06	56	13	52	170	63.9	22.1	81
White07	62	12	53	159	52.6	20.8	77
White08	56	13	53	168	99.6	35.3	114.5
White09	54	13	49	161	71.1	27.4	84
White10	55	13	52	163.5	76.1	28.5	97.5
White11	60	12	49	162.5	76	28.8	83.5

Participant	Age (years)	Age of first men cycle (years)	Age of last men cycle (years)	Height (cm)	BM (kg)	BMI	WC (cm)
ME01	52	12	38	168	85.9	30.4	106
ME02	57	14	54	176	99.1	32.0	113
ME03	53	14	46	162	76.1	29.0	100.5
ME04	54	12	51	154	72	30.4	101.5
ME05	51		42	164	82.7	30.7	105.5
ME06	47	15	45	162	109.2	41.6	122
ME07	49	13	37	161	101	39.0	118
ME08	55	14	53	158.6	94.2	37.4	117
ME09	53	14	50	162.6	68.1	25.8	94
ME10	54	12.5	50	156	51.3	21.1	84
ME11	54	11.5	45.5	160.5	84.2	32.7	106

Participant	Resting BP Auto LA Systolic (mmHg)	Resting BP Auto LA Diastolic (mmHg)	Resting BP Auto RA Systolic (mmHg)	Resting BP Auto RA Diastolic (mmHg)	Resting BP Manual LA Systolic (mmHg)	Resting BP Manual LA Diastolic (mmHg)	Resting BP Manual RA Systolic (mmHg)	Resting BP Manual RA Diastolic (mmHg)
White01	119	73	115	70	104	74	102	72
White02	157	83	143	77	136	86	140	82
White03	102	69	113	74	108	68	104	64
White04	110	75	115	73	110	64	106	62
White05	98	53	99	59	96	58	100	60
White06	141	91	146	91	140	78	150	80
White07	101	59	97	58	92	52	90	58
White08	110	75	113	81	110	74	110	70
White09	105	72	103	72	110	62	102	68
White10	102	68	105	73	102	68	98	60
White11	113	74	121	72	104	62	112	64
White12	111	79	118	84	120	72	110	74

Participant	Bicep SF (mm)	Tricep SF (mm)	Subcap SF (mm)	Illica Crest SF (mm)	Medial Calf SF (mm)	Total SF (mm)	CPAFLA Score	CPAFLA Rating	Body Fat %
ME01	21.1	25.3	23.1	25.2	30.1	124.8	0.8	Fair	43.5
ME02	18.3	25.2	37.3	28.3	22.5	131.6	0.8	Fair	45.8
ME03	9.7	22.7	21.2	30	18.9	102.5	1.8	Good	39.4
ME04	8	21.5	21.4	15.2	17.8	83.9	1.2	Fair	37.3
ME05	15.8	30.9	19.4	30.6	30.4	127.1	0.8	Fair	45.1
ME06	38.8	28.4	49.4	45.3	45.6	207.5	0	NI	47.2
ME07	28.4	37.8	39.8	47.1	42.7	195.8	0	NI	47.7
ME08	23.7	37	30.9	56.4	54.9	202.9	0	NI	47.3
ME09	13.6	24.7	26.1	23.5	18.9	106.8	1.8	Good	34.7
ME10	7.5	15.1	13.5	14	9.5	59.6	3.4	Very Good	27.4
ME11	14.7	26	37.7	41.8	34.6	154.8	0.4	NI	43.1

Participant	Bicep SF (mm)	Tricep SF (mm)	Subcap SF (mm)	Illica Crest SF (mm)	Medial Calf SF (mm)	Total SF (mm)	CPAFLA Score	CPAFLA Rating	Body Fat %
White01	10.4	16.4	22	19.8	17.4	86	3	Very Good	36.7
White02	6.6	15.3	19.6	23.5	15.3	80.3	3.4	Very Good	31.3
White03	12	24.2	30	20.5	18.5	105.2	1.8	Good	37.4
White04	12.6	16.7	21.5	21.7	23	95.5	3	Very Good	35.3
White05	16.1	24.9	23	22.9	22.9	109.8	1.8	Good	35.3
White06	6.7	11.1	16	13.9	11.4	59.1	3.4	Very Good	31.2
White07	7.8	15.3	8.6	13.3	13.8	58.8	4	Excellent	27.5
White08	26.1	33.5	49.1	35.7	40.3	184.7	0	Need Improvement	46.9
White09	31	29.9	22.8	19	15.3	118	2.6	Very Good	38.9
White10	13.8	31.6	26.4	25.8	22.2	119.8	1.4	Fair	36.4
White11	10.5	20.1	22.3	27.3	20.5	100.7	3	Very Good	39.1
White12	14.3	18.5	38	27.6	24.6	123	1.4	Fair	41.7

Participant	Resting BP Auto LA Systolic (mmHg)	Resting BP Auto LA Diastolic (mmHg)	Resting BP Auto RA Systolic (mmHg)	Resting BP Auto RA Diastolic (mmHg)	Resting BP Manual LA Systolic (mmHg)	Resting BP Manual LA Diastolic (mmHg)	Resting BP Manual RA Systolic (mmHg)	Resting BP Manual RA Diastolic (mmHg)
ME01	103	65	112	69	110	70	112	72
ME02	117	74	122	79	130	78	130	80
ME03	102	69	101	66	96	62	108	60
ME04	104	69	106	68	110	70	118	68
ME05	117	68	106	65	118	74	116	72
ME06	130	83	126	81	138	78	136	80
ME07	141	92	151	92	144	102	154	98
ME08	121	66	122	70	124	70	126	60
ME09	112	75	106	74	92	68	98	68
ME10	97	63	96	63	90	64	92	60
ME11	108	71	104	69	98	62	104	68

Participant	HPAPQ Score	HPAPQ Rating	Resting HR	Resting VO_2 relative ($mLO_2 \cdot min^{-1} \cdot kg$ of BM)	Resting VO_2 absolute ($LO_2 \cdot min^{-1}$)	VO_2 peak relative ($mLO_2 \cdot min^{-1} \cdot kg$ of BM)	VO_2 peak absolute ($mLO_2 \cdot min^{-1}$)	HR max (bpm)	Age Predicted Max HR (bpm)	RPE at max
White01	7	Very Good	61	3.48	242	20.90	1.44	156	154	20
White02	11	Excellent	62	2.87	148	29.31	1.50	161	163	20
White03	9	Excellent	62	3.12	215	26.80	1.83	168	162	14
White04	6	Very Good	60	3.67	250	25.77	1.75	171	168	20
White05	11	Excellent	57	3.29	210	26.05	1.69	152	164	16
White06	11	Excellent	65	5.59	357	40.40	2.58	170	164	17
White07	10	Excellent	81	4.08	213	29.17	1.53	147	158	18
White08	9	Excellent	75	3.98	395	22.40	2.23	162	164	NA
White09	11	Excellent	70	3.76	266	31.70	2.30	172	166	NA
White10	5	Good	62	3.44	262	28.15	2.14	169	165	19
White11	10	Excellent	67	4.08	310	28.00	2.12	160	160	19
White12	11	Excellent	64	3.29	228	23.18	1.61	175	158	11

Participant	HPAPQ Score	HPAPQ Rating	Resting HR	Resting VO_2 relative ($mLO_2 \cdot min^{-1} \cdot kg$ of BM)	Resting VO_2 absolute ($LO_2 \cdot min^{-1}$)	VO_2 peak relative ($mLO_2 \cdot min^{-1} \cdot kg$ of BM)	VO_2 peak absolute ($mLO_2 \cdot min^{-1}$)	HR max (bpm)	Age Predicted Max HR (bpm)	RPE at max
ME01	10	Excellent	68	4.06	349	22.90	1.97	165	168	20
ME02	11	Excellent	92	4.00	396	20.68	2.05	160	163	15
ME03	8	Very Good	66	4.20	322	28.01	2.13	165	167	12
ME04	10	Excellent	54	3.56	258	26.77	1.92	155	166	18
ME05	3	Fair	72	3.50	291	19.51	1.61	146	169	20
ME06	2	Fair	60	2.63	287	16.52	1.80	137	173	20
ME07	2	Fair	66	2.92	298	20.17	2.06	154	171	19
ME08	2	Fair	78	3.26	307	15.84	1.49	161	165	20
ME09	7	Very Good	78	5.03	244	21.29	1.45	158	167	13
ME10	6	Very Good	72	5.28	271	20.15	1.03	158	166	15
ME11	7	Very Good	91	4.45	374	24.06	2.03	177	166	16

Participant	FOR_{peak} Trial 1 ($g \bullet min^{-1} \bullet kg$ of fat free mass)	Fatmax Trial 1 (% VO_2 peak)	FOR_{max} Trial 2 ($g \bullet min^{-1} \bullet kg$ of fat free mass)	Fatmax Trial 2 (% VO_2 peak)	Total Step Count (steps)	Total EE (kcal)	Total Activity Counts
White01	8.35	45.33	7.018	48.88	9779	435.15	117583
White02	4.385	32.75	6.082	48.74	17527	472.58	280304
White03	7.451	49.07	7.404	37.11	7365	524.20	106604
White04	6.439	54.83	4.613	33.33	6593	387.17	92607
White05	8.248	55.51	9.494	49.35	9515	480	159175
White06	17.097	63.19	16.455	62.65	23358	1183	704131
White07	7.688	57.46	5.425	57.18	12357	472.72	260620
White08	8.826	36.8	11.167	62.79	6177	643.47	94315
White09	8.724	54.96	9.12	49.96	7203	581.59	142773
White10	6.88	35.23	9.21	31.88	10862	692.72	188386.67
White11	7.403	48.85	8.879	52.99	7044	423.89	125482.67
White12	5.247	44.6	6.516	54.6	4674	2409.37	78220.66

Participant	FOR_{peak} Trial 1 ($g \bullet min^{-1} \bullet kg$ of fat free mass)	Fatmax Trial 1 (% VO_2 peak)	FOR_{max} Trial 2 ($g \bullet min^{-1} \bullet kg$ of fat free mass)	Fatmax Trial 2 (% VO_2 peak)	Total Step Count (steps)	Total EE (kcal)	Total Activity Counts
ME01	7.798	31.71	8.256	28.64	7261	409.93	64343
ME02	NA	NA	14.363	43.27	8057	756.45	153845.33
ME03	5.909	22.01	5.722	36	8701	518.95	203751
ME04	12.973	57.57	11.33	62.75	15657	1063	284893
ME05	8.23	52.67	8.878	66.27	2663	140.08	26785
ME06	12.772	39.2	12.368	38.19	9362	835.94	135056
ME07	8.364	42.1	9.665	38.63	4215	2357.79	58457
ME08	7.197	61.85	7.662	56.09	9952	524.87	89259
ME09	7.477	61.88	5.658	37.16	4245	380.67	120665
ME10	3.095	34.36	3.183	45.62	4624	256.49	72626
ME11	7.985	52.97	13.464	40.7	5917	521.73	93680

Participant	Estradiol (pmol/L)	Estrone (pmol/L)	Progesterone (nmol/L)	Lutropin (IU/L)	FSH (IU/L)	Testosterone (nmol/L)	SHBG (nmol/L)	Androgen Index	Insulin (pmol/L)	Glucose (noml/L)
White01	<70	208	1	37	54	2.6	57.7	0.520	47	4.9
White02	<70	117	1	47	103	0.9		0.000	40	6.1
White03	125	72	1	25	75	0.9	26	0.081	49	5.1
White04	96	150	<1	36	77	1.8	50.1	0.313	22	5
White05	169	95	1	58	101	1	47.1	0.163	47	5.8
White06	<70	241	<1	45	136	1.6	60.7	0.337	39	4.8
White07	<70	254	2	49	115	1.5	29.5	0.153	15	5.2
White08	<70	152	1	32	57	1.3	33.3	0.150	97	5.4
White09	<70	94	<1	27	50	0.9	60.1	0.188	42	5.4
White10	<70	50	<1	57	93	0.69	40.5	0.097	60	6.2
White11	<70	102	<1	21	62	0.69	25	0.060	60	5.4
White12	<70	124	1	31	97	2.1	36.9	0.269	35	4.7

Participant	Estradiol (pmol/L)	Estrone (pmol/L)	Progesterone (nmol/L)	Lutropin (IU/L)	FSH (IU/L)	Testosterone (nmol/L)	SHBG (nmol/L)	Androgen Index	Insulin (pmol/L)	Glucose (noml/L)
ME01	85	213	3	30	51	1	35.1	0.122	27	5.7
ME02	<70	104	<1	34	64	1.1	38.7	0.148	56	4.9
ME03	<70		<1	33	76	1.1	49.3	0.188	26	5.1
ME04	<70	112	1	49	78	0.08		0.000	<14	4.8
ME05	<70	186	2	36	61	1.3	24.1	0.109	87	7.3
ME06	<70	225	2	19	37	1.8	22	0.137	128	5.4
ME07	<70	135	1	15	35	1.9	32	0.211	183	5.6
ME08	<70	NA	<1	NA	NA	NA	NA	NA	58	NA
ME09	<70	126	1	31	75	1.6	77.7	0.431	39	4.6
ME10	<70	81	1	51	138	0.08	71.7	0.020	30	4.7
ME11	<70	176	<1	36	71	1.2	18.8	0.078	56	5.8

Participant	Cortisol (nmol/L)	Cholesterol (nmol/L)	LDL-CH (nmol/L)	HDL-CH (nmol/L)	CH/HDL ratio	Triglycerides (nmol/L)
ME01	335	5.53	3.49	1.69	3.3	0.77
ME02	124	7.15	4.56	1.63	4.4	2.12
ME03	222	4.73	2.78	1.53	3.1	0.93
ME04	71	4.56	2.53	1.79	2.5	0.53
ME05	213	5.02	2.92	1.08	4.6	2.24
ME06	166	5.18	3.47	0.88	5.9	1.83
ME07	76	4.42	2.96	0.91	4.9	1.2
ME08	129	NA	NA	NA	NA	NA
ME09	269	6.95	4.1	2	3.5	1.86
ME10	119	3.31	2.31	0.075	4.4	0.55
ME11	367	3.98	2.1	1.23	3.2	1.24

Participant	Cortisol (nmol/L)	Cholesterol (nmol/L)	LDL-CH (nmol/L)	HDL-CH (nmol/L)	CH/HDL ratio	Triglycerides (nmol/L)
White01	66	9.35	6.71	1.49	6.4	2.6
White02	198	6.04	3.52	2.1	2.9	0.92
White03	335	4.66	3.08	1.2	3.9	0.83
White04	104	6.01	3.99	1.4	4.3	1.36
White05	429	5.13	2.95	1.86	2.8	0.71
White06	256	5.97	3.94	1.5	4	1.16
White07	1108	4.87	2.64	1.92	2.5	0.68
White08	84	4.87	2.79	1.55	3.1	1.16
White09	340	4.84	2.42	2.08	2.3	0.75
White10	5194	4.49	2.86	1.33	3.4	0.67
White11	223	4.78	2.75	1.28	3.7	1.64
White12	250	5.31	3.33	1.07	5	2

APPENDIX F. Data tables for the comparison of the 2nd order to the 3rd order polynomial graphs.

Middle Eastern		
Participant	2nd order R values	3rd order R values
1	0.6821	0.7876
2	0.249	0.6401
3	0.4906	0.4915
4	0.7129	0.7734
5	0.2591	0.3422
6	0.7808	0.8568
7	0.8462	0.8647
8	0.6071	0.667
9	0.3178	0.3233
10	0.6887	0.7124
11	0.6116	0.6132

White		
Participants	2nd order R values	3rd order R values
1	0.718	0.727
2	0.8477	0.5955
3	0.6628	0.6641
4	0.2055	0.2061
5	0.7693	0.779
6	0.7493	0.9039
7	0.6562	0.6689
8	0.1926	0.2434
9	0.6123	0.6731
10	0.4354	0.4945
11	0.6086	0.6137
12	0.4963	0.5211

Paired Samples Test For Middle Eastern Women

	Paired Differences					t	df	Sig. (2-tailed)
	Mean	Std. Deviation	Std. Error Mean	95% Confidence Interval of the Difference				
				Lower	Upper			
Second Order – Third Order	-0.0751	0.1108543	0.0334238	-0.149591	-0.00064	-2.247	10	0.048

Paired Samples Test For White Women

	Paired Differences					t	df	Sig. (2-tailed)
	Mean	Std. Deviation	Std. Error Mean	95% Confidence Interval of the Difference				
				Lower	Upper			
Second Order-Third Order	-0.01135	0.0937634	0.0270672	-0.070932	0.0482161	-0.42	11	0.683

REFERENCES

Achten J, Venables MC, Jeukendrup AE. Fat Oxidation Rates are Higher During Running Compared with Cycling Over a Wide Range of Intensities. *Metabolism.* 2003;52(6):747-752.

Al-Nozha M, Al-Hazzaa HM, Arafah MR et al. Prevalence of Physical Activity and Inactivity Among Saudis aged 30-70 years. *Saudi Medical Journal.* 2007;28(4):559-68.

Barrett-Connor E. Epidemiology and the Menopause: a Global Overview. *International Journal of Fertility and Menopausal Study.* 1993;38(Suppl 1):6-14.

Bouchard C, Shephard RJ. Physical Activity, Fitness, and Health: The Model and Key Concepts. *Physical Activity, Fitness, and Health International Proceedings and Consensus Statement.* 1994;Chpt 3:77-88.

Bjorntorp P. The Regulation of Adipose Tissue Distribution in Humans. *International Journal of Obesity and Related Metabolic Disorders.* 1996;20(4):291-302.

Bjorntorp P. Hormonal Control of Regional Fat Distribution. *Human Reproduction.* 1997;12(S1):21-25.

Burger HG, Dudley EC, Robertson DM, Dennerstein L. Hormonal Changes in the Menopause Transition. *The Endocrine Society.* 2002;57:257-75.

Campbell SE, Febbraio MA. Effect of ovarian hormones on mitochondrial enzyme activity in fat oxidation pathway of skeletal muscle. *American Journal of Physiology, Endocrinology and Metabolism.* 2001;281:E803.

Carr MC. The Emergence of the Metabolic Syndrome with Menopause. *Journal of Clinical Endocrinology & Metabolism.* 2003;88(6):2404-2411.

Christensen EH, Hanse O. Arbeitsfahigkeit und Ernahrung. S*candanavian Archives of Physiology.* 1939;81:160-71.

Chenevière X, Malatesta D, Gojanovic B, Borrani F. Differences in whole-body fat oxidation kinetics between cycling and running. *European Journal of Applied Physiology.* 2010;109(6):1037-45.

Chu, L. The effect of obesity and pubertal development on fat oxidation during exercise in youth. *ProQuest Dissertations and Theses;* 2011; Dissertations & Theses @ York University.

Davies CT, Few J, Foster C, et al: Plasma catecholamine concentration during dynamic exercise involving different muscle groups. *European Journal of Applied Physiology.* 1974;32:195-206.

D'eon T, Braun B. Carbohydrate and Fat Utilization at Rest and During Exercise. *Journal of Women's Health and Gender-Based Medicine.* 2002;11(3).

Elavsky S, McAuley E. Physical activity and mental health outcomes during menopause: a randomized controlled trial. *Annals of Behavioral Medicine.* 2007;33(2):132-42.

Ellis GS, Lanza-Jacoby S, Gow A, Kendrick ZV. Effects of estradiol on lipoprotein lipase activity and lipid availability in exercised male rats. *Journal of Applied Physiology.* 1994;77:209.

Ferrara CM, Lynch NA, Nicklas BJ, Ryan AS, Berman DM. Differences in Adipose Tissue Metabolism between Postmenopausal and Perimenopausal Women. *Journal of Clinical Endocrinology & Metabolism.* 2002;87(9):4166-4170.

Field A. Discovering statistics using SPSS. SAGE Publication Ltd. 3rd ed. 2009.

Frayn KN. Calculation of substrate oxidation from gaseous exchange rates in vivo. *Journal of Applied Physiology.* 1983;55(2):628-634.

Friedlander AL, Casazza GA, Horning MA, Buddinger TF, Brooks GA. Effects of exercise intensity and training on lipid metabolism in young women. *American Journal of Physiology.* 1998;275:E853-E863.

Gledhill, N. and V. Jamnik. Canadian Physical Activity and Lifestyle Approach. *Ottawa: Canadian Society for Exercise Physiology.* 3rd ed. 2003.

Hackney AC, McCraken-Compton MA, Ainsworth B. Substrate Response to Submaximal Exercise in the Midfollicular and Midluteal Phases of the Menstrual Cycle. *International Journal of Sports Nutrition.* 1994;4:299.

Hall LML, Moran CN, Milne GR, et al. Fat Oxidation, fitness and Skeletal Muscle Expression of Oxidative/Lipid Metabolism Genes in South Asians: Implication for Insulin Resistance? *PloS one.* 2010;5(12):e14197.

Hansen PA, McCarthy TJ, Pasia EN, Spina RJ, Gulve E. Effects of ovariectomy and exercise training on muscle GLUT-4 content and glucose metabolism in rats. *Journal of Applied Physiology.* 1996;80:1605.

Haskell WL, Lee I, Pate RR et al. Physical Activity and Public Health Updated Recommendation for Adults From the American College of Sports Medicine and the American Heart Association. *Circulation.* 2007;116:1081-1093

Hatta H, Atomi Y, Shinohara S, Yamamoto Y, Yamada S. The effects of ovarian hormones on glucose and fatty acid oxidation during exercise in female ovariectomized rats. *Hormone and Metabolism Resarch.* 1988;20:609–611.

Heyward V. Advanced fitness assessment and exercise prescription. *Human Kinetics.* 5th ed. 2009.

Hickner RC, Privette J, Mciver K, et al. Fatty acid oxidation in African-American and Caucasian women during physical activity. *American Journal of Clinical Nutrition.* 2011:2319-2324.

Howely ET, Bassett Jr DR, Welch HG. Criteria for maximal oxygen uptake: review and commentary. *Medicine and Science in Sport and Exercise.* 1995;27(9):1292-1301.

Janghorbani M, Amini M, Willett WC, et al. First nationwide survey of prevalence of overweight, underweight, and abdominal obesity in Iranian adults. *Obesity.* 2007;15(11):2797-808.

Janssen, I. LeBlanc AG. Systematic review of the health benefits of physical activity and fitness in school-aged children and youth. *International Journal of Behavioral Nutrition and Physical Activity* 2010.

Janssen I, Powell L, Crawford S, Lasley B, Sutton-Tyrrell K. Menopause and the Metabolic Syndrome. The Study of Women's Health across the Nation. *Archives of Internal Medicine.* 2008;168(14):1568-1575.

Johnson ML, Zarins Z, Fattor JA, et al. Twelve weeks of endurance training increases FFA Mobilization and Reesterfication in Postmenopausal Women. *Journal of Applied Physiology.* 2010;109:1573-81.

Kalkhoff R. Metabolic effects of progesterone. *American Journal of Obstetrician and Gynecology.* 1982;142:735.

Kalyani RR, Franco M, Dobs AS, et al. The Association of Endogenous Sex Hormones, Adiposity, and Insulin Resistance with Incident Diabetes in Postmenopausal Women. *Journal of Clinical Endocrinology and Metabolism.* 2009;94(11):4127– 4135.

Kendrick ZV, Steffen C, Rumsey W, Goldberg D. Effect of estradiol on tissue glycogen metabolism in exercised oophorectomized rats. *Journal of Applied Physiology.* 1987;63:492.

Kohrt WM, Kirwan JP, Staten MA, et al. Insulin Resisitane in aging is Related to Abdominal Obesity. *Diabetes.* 1993;42:273-81.

Levine JA. Non-Exercise Activity Thermogenesis (NEAT). Nutrition Reviews. 2004;62(S2):S82-S97.

Lusk G. The Elements of the Science of Nutrition. *Johnson Reprint Corp.* 3rd ed. 1976.

Main P, Darin N, Amemiya T, et al. Cortisol secretion in regulation to body fat distribution in obese premenopausal women. *Metabolism.* 1992;41(8):882-886.

Marin PL, Lönn L, Andersson B, Odén B, Olbe L, Bengtsson BA, Björntorp P. Assimilation of triglycerides in subcutaneous and intraabdominal adipose tissues in vivo in men: effects of testosterone. *Journal of Clinical Endocrinology and Metabolism.* 1996;81(3):1018–1022.

Mayes JS, Watson GH. Direct effects of sex steroid hormones on adipose tissue and obesity. *Obesity Review.* 2004;5:197-216.

Mead JR, Irvine, SA, Ramji DP. Lipoprotein lipase: structure, function, regulation, and role in disease. *Journal of molecular Medicine.* 2002;80:753-769.

Merom D, Sinnreich R, Aboudi V, Kark JD, Nassar H. Lifestyle Physical Activity Among Urban Palestinian and Israelis: A Cross-sectional Comparison in the Palestinian-Israeli Jerusalem Risk Factor Study. *Biomed Central Public Health.* 2012;12:90.

Nelson H, Haney E, Humphrey L, et al. Management of menopause-related symptoms. *Agency for Healthcare Research and Quality.* 2005.

Park Y, Zhu S, Palaniappan L, Heshka S, Carnethon MR, Heymsfield SB. The Metabolic Syndrome: Prevalence and Associated Risk Factor Findings in the US Population From the Third National Health and Nutrition Examination Survey, 1988-1994. *Archives of Internal Medicine*. 2003;163(4):427–436.

Peckett AJ, Wright DC, Riddell MC. The Effects of Glucocorticoids on Adipose Tissue Metabolism. *Metabolism*. 2011;60(11)1500-10.

Pedersen SB, Kristensen K, Hermann JA, et al. Estrogen controls lipolysis by up-regulating alpha2A-adrenergic receptors directly in human adipose tissue through estrogen receptor alpha. Implications for the female fat distribution. *The Journal of Clinical Endocrinology and Metabolism*. 2004;89(4):1869-78.

Price TM, O'Brien SN, Welter BH et al. Estrogen regulation of adipose tissue lipoprotein lipase – Possible mechanism of body fat distribution. *American Journal of Obstetrician and Gynecology*. 1998;178:101-7.

Randolph JF Jr, Sowers M, Bondarenko IV, et al. Changes in Estradiol and Follicle-Stimulating Hormone across the Early Menopausal Transition: Effects of Ethnicity and Age. *The Journal of Clinical Endocrinology& Metabolism*. 2004;89(4):1555-1561.

Rising R, Tataranni P, Snitker S, Ravussin E. Decreased ratio of fat to carbohydrate oxidation with increasing age in pima indians. *Journal of American College of Nutrition*. 1996;15: 309–312.

Saad F, Gooren IL. The Role of Testosterone in the Etiology and Treatment of Obesity, the Metabolic Syndrome and Diabetes Mellitus Type 2. *Journal of Obesity*. 2011.

Sallis JF, Frank LD, Saelens BE, Kraft MK. Active transportation and physical activity: opportunities for collaboration on transportation and public health research. *Transport Res A-Pol*. 2004;38:249–268.

Saltiel AR, Kahn RC. Insulin signaling and the regulation of glucose and lipid metabolism. *Nature*. 2001;414(6865):799-806.

Santa-Clara H, Szymanski L, Ordille T, Fernhall B. Effects of exercise training on resting metabolic rate in postmenopausal African American and Caucasian women. *Metabolism: Clinical and Experimental*. 2006;55(10):1358-64.

Setiawan VW, Haiman CA, Stanczyk FZ, Marchand L Le, Henderson BE. Racial/ethnic differences in postmenopausal endogenous hormones: the multiethnic cohort study. *Cancer Epidemiology, Biomarkers & Prevention*. 2006;15(10):1849-55.

Sherman S. Defining the menopausal transition. *The American Journal of Medicine*. 2005;118 Suppl :3-7.

Sial S, Coggan AR, Hickner RC, Klein S. Training-induced alterations in fat and carbohydrate metabolism during exercise in elderly subjects. *American Journal of Physiology Endocrinology Metabolism. 1998;274 E785–E790.*

Sial S, Coggan AR, Carroll R, Goodwin J, Klein S. Fat and carbohydrate metabolism during exercise in elderly and young subjects. *American Journal of Physiology Endocrinology Metabolism.* 1996;271 E983–E989.

Sidossis L, Wolfe R, Coggan A. Regulation of fatty acid oxidation in untrained vs. trained men during exercise. *American Journal of Physiology Endocrinology Metabolism.* 1998;274:E510-E515

Soler JT, Folsom AR, Kaye SA, Prineas RJ. Associations of abdominal adiposity, fasting insulin, sex hormone binding globulin, and estrone with lipids and lipoproteins in postmenopausal women. *Atherosclerosis.* 1989:79(1):21-27.

Soules MR, Sherman S, Parrott E, et al. Executive summary : Stages of Reproductive Aging Workshop (STRAW). *Climacteric.* 2001:267-272.

Sowers MR, Wilson AL, Karvonen-Gutierrez CA, Kardia SR. Sex steroid hormone pathway genes and health-related measures in women of 4 races/ethnicities: the Study of Women's Health Across the Nation (SWAN). *The American Journal of Medicine.* 2006;119(9 Suppl 1):S103-110.

Stefanick ML, Wood PD. Physical Activity, Lipid and Lipoprotein Metabolism and Lipid transport. *Physical Activity, Fitness, and Health International Proceedings and Consensus Statement.* 1994;Chpt 26:417-31.

Sutton-Tyrrell K, Zhao X, Santoro N, et al. Reproductive Hormones and Obesity: 9 Years of Observation From the Study of Women's Health Across the Nation. *American Journal of Epidemiology.* 2010;171(11):1203-1213.

Svendsen OL, Hassager C, Christiansen C. Relationships and independence of body composition, sex hormones, fat distribution and other cardiovascular risk factors in overweight postmenopausal women. *International Journal of Obesity.* 1993;17:459-63.

Toth EL, Suthijumronn A, Crockford PM, Ryan EA. Insulin Action Does Not Change During the Menstrual Cycle in Normal Women. *Journal of Clinical Endocrinology and Metabolism. 1987;64:74.*

Treuth MS, Hunter GR, Weinsier RL, Kell SH. Energy Expenditure and Substrate Utilization in Older Women After Strength Training: 24-hr Calorimeter Results. *Journal of Applied Physiology (Bethesda, Md. : 1985).* 1995;78(6):2140-2146.

Vankrieken L. Testosterone and the free androgen index. Diagnostic Products Corporation. 1997.

Villaverde_Gutierrez C, Araujo E, Cruz F et al. Quality of Life of Rural Menopausal Women in Response to a Customized Exercise Programme. *Journal of Advanced Nursing.* 2006;54(1):11-19.

Warburton, DER, Jamnik, VK, Bredin, SSD, Gledhill, N. The Physical Activity Readiness Questionnaire (PAR-Q+) and electronic Physical Activity Readiness Medical Examination (ePARmed-X+). *Health & Fitness Journal of Canada.* 2011;4(2): 3-23.

Warburton DER, Katzmarzyk PT, Rhodes RE, Shephard RJ. Evidence-informed physical activity guidelines for Canadian adults. *Applied Physiology Nutrition and Metabolism.* 2007;32:S16–S68.

World Health Organization. Global recommendations on physical activity for health. Report of a WHO Consultation. *World Health Organization Technical Report Services*. 2010.

World Health Organization. Obesity: preventing and managing the global epidemic. Report of a WHO Consultation. *World Health Organization Technical Report Services*. 2000.

Zamboni M, Armellini F, Milani MP et al. Body fat distribution in pre- and postmenopausal women: metabolic and anthropometric variables and their inter-relationships. *International Journal of Obesity and Related Metabolic Disorders*. 1992;16(7):495-504.

Zarins ZA, Wallis GA, Faghihnia N, et al. Effects of endurance training on cardiorespiratory fitness and substrate partitioning in postmenopausal women. *Metabolism*. 2010;58(9):1338-1346.