Effects of maternal exercise type and metrics on maternal and infant body composition and blood biomarkers

By

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July, 2025

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ABSTRACT

Obesity is a growing global health issue, particularly among reproductive-aged women, increasing risks during pregnancy such as gestational diabetes, hypertension, and preeclampsia. These complications affect both maternal and fetal health and can have lasting consequences. A major concern in obese pregnancies is metabolic dysfunction, including lipid and glucose dysregulation. While cholesterol is vital, excess levels raise cardiovascular and oxidative stress risks. Elevated lactate and insulin resistance, assessed by HOMA-IR, are common in obesity and worsen during pregnancy.

Body composition significantly influences pregnancy outcomes—greater muscle mass and lower fat levels enhance insulin sensitivity and energy. Regular exercise improves body composition, regulates lipids, lowers lactate, and boosts insulin sensitivity, reducing obesity-related risks. Maternal exercise also benefits offspring, lowering the child's risk of metabolic disorders like dyslipidemia and type 2 diabetes by improving maternal glucose control and influencing fetal epigenetics.

Emerging research shows maternal exercise reduces inflammation and supports healthy fetal growth. This study investigates how prenatal exercise affects infant body

composition and metabolic biomarkers. We hypothesize that aerobic exercise during pregnancy lowers infant adiposity and improves insulin and lipid profiles.

Healthy pregnant women (<16 weeks gestation) were randomized to supervised moderate-intensity exercise (aerobic, resistance, or combined) or a control group. Maternal and infant body composition was measured, and blood biomarkers were collected. Findings from Aim 1 showed that higher weekly exercise volume and longer duration were linked to lower maternal weight and improved biomarkers, particularly with aerobic exercise.

Aims 2 and 3 demonstrated that maternal exercise influenced infant body composition and lipid profiles. Aerobic exercise was associated with lower non-HDL and higher HDL cholesterol in infants, while resistance training reduced LDL cholesterol. Greater exercise frequency, intensity, and volume were linked to improved infant growth and metabolic health, emphasizing the importance of structured prenatal exercise for optimizing early-life outcomes.

Effects of maternal exercise type and metrics on maternal and infant body composition and blood biomarkers

A Dissertation

Presented to the Faculty of the Department of Kinesiology

East Carolina University

In Partial Fulfillment of the Requirements for the Degree

Doctor of Philosophy in Bioenergetics and Exercise Science (Concentration: Exercise Physiology and Behavioral Sciences)

By

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July, 2025

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AE	aerobic exercise group
AERE	combination exercise group
BF%	body fat percentage
BMI	body mass index
BW	body weight
CDC	center of disease control and prevention
CMD	cardiometabolic disease
CON	control group
FFM%	fat free mass percent
GDM	gestational diabetes mellitus
GWG	gestational weight gain
HDL	high-density lipoprotein
HOMA-IR	homeostatic model assessment for insulin resistance
HR	heart rate
IR	insulin resistance
LDL	low-density lipoprotein
ME	maternal exercise
MET	metabolic equivalent
NonHDL	non-high-density lipoprotein
RE	resistance exercise group
TC	total cholesterol
TG	triglyceride

CHAPTER ONE

Introduction

Public Health Implications of Obesity

Overview of the prevalence of adult obesity:

The prevalence of adult obesity in the United States has reached alarming levels, becoming one of the most pressing public health challenges. According to data from the National Health Statistics Report, as of 2021, approximately 41.9% of adults aged 20 years and older were classified as obese. This figure represents a significant increase from previous decades, indicating a troubling upward trend[1-2]. Obesity rates among adults show considerable variation based on age; for instance, young adults (aged 20-39 years) have a prevalence of 40%, which underscores the widespread nature of obesity across age groups[2-3]. Several factors have driven the rise in adult obesity rates in the United States. For example, decreased physical activity and sedentary behaviors, including prolonged screen time, further exacerbate the problem. Adult obesity poses serious health risks, both immediate and long-term. Obese individuals are at higher risk for developing chronic conditions such as type 2 diabetes, hypertension, cardiovascular diseases, certain cancers, and musculoskeletal disorders, which can significantly impact overall quality of life and life expectancy[4].

Overview of the prevalence of obesity during pregnancy:

In the United States, the prevalence of obesity among pregnant women varies significantly by race and ethnicity. Approximately 50% of pregnant non-Hispanic Black

women are classified as obese, making this group the most affected by obesity during pregnancy. Approximately 40% of pregnant Hispanic women are classified as obese. Non-Hispanic White women have a lower prevalence, with around 30% to 35% classified as obese during pregnancy. Asian women experience the lowest prevalence during pregnancy, with estimates ranging from 20% to 25%[5-6].

Risks associated with pregnancies affected by obesity:

Obesity during pregnancy presents several risks for both the mother and the baby. One major risk is the development of gestational diabetes, which can lead to complications, such as excessive fetal growth and preterm birth. Additionally, pregnant women with obesity are at a higher risk of developing preeclampsia, a condition characterized by high blood pressure and organ damage, which can pose serious dangers to both mother and baby. The likelihood of preterm birth is also increased, potentially leading to respiratory and developmental issues for the baby. Obesity is associated with a higher probability of requiring a cesarean section due to complications during labor or difficulties with vaginal delivery[7-9]. Excessive weight gain can result in fetal macrosomia, where the baby grows larger than average, augmenting the risk of birth injuries and delivery complications. There is also an increased risk of stillbirth, though the exact mechanisms are not understood[9-11]. Postpartum, obese women may face more complications such as infections, longer recovery times, and challenges with breastfeeding. Additionally, both mother and child are at greater risk for long-term health issues, including obesity and cardiovascular diseases later in life[10-12]. Managing weight gain, maintaining regular

prenatal care, and adopting a healthy lifestyle are crucial for mitigating these risks and improving outcomes for both the mother and the baby.

Overview of the prevalence of childhood obesity:

When pregnant women are obese and/or gain excessive weight gain during pregnancy, this usually leads to larger babies at birth[12]. This pattern leads to children who develop obesity earlier and more severe. In the United States, the prevalence of childhood obesity has become a critical public health issue over the past several decades. According to the National Health Statistics Report , approximately 19.7% of children and adolescents aged 2-19 years were classified as obese in 2021. This equates to about 14.4 million children, reflecting a significant increase from previous decades[1,13-15]. Childhood obesity poses significant immediate and long-term health risks. Obese children are more likely to develop health issues such as type 2 diabetes, hypertension, and psychological problems. Additionally, they are at a higher risk of becoming obese adults, leading to increased morbidity and mortality due to chronic diseases like cardiovascular disease and certain cancers[15].

To address the issue of childhood obesity, various public health initiatives have been implemented in the United States. These initiatives focus on promoting healthy eating, increasing physical activity, and reducing sedentary behaviors among children. Policy measures such as improving school nutrition standards, regulating the marketing of unhealthy foods to children, and creating environments that support physical activity are

also crucial in combating this epidemic[16]. Comprehensive efforts involving parents, schools, communities, and policymakers are essential to reverse the trend of childhood obesity and improve the health outcomes of future generations.

Cardiometabolic disease and how it relates to body composition and blood biomarkers of obesity:

Obesity is related to other diseases, including cardiovascular diseases (such as coronary artery disease, stroke, and hypertension) and metabolic diseases (such as type 2 diabetes and metabolic syndrome)[17-20]. These conditions are interrelated, they are referred to as cardiometabolic disease and share common risk factors, including body composition, blood cholesterol, glucose levels, insulin levels, and insulin resistance as measured by the Homeostasis Model Assessment of Insulin Resistance (HOMA-IR)[17-26].

Body composition, particularly the amount and distribution of body fat, plays a significant role in cardiometabolic health. Excess adiposity, especially visceral fat, is a major risk factor for developing cardiometabolic diseases[27-28]. Adipose tissue is metabolically active and releases free fatty acids, inflammatory cytokines, and hormones that impair insulin signaling and promote systemic inflammation, contributing to insulin resistance, dyslipidemia, and hypertension. Excessive body fat, especially around the abdomen, leads to a higher risk of type 2 diabetes, dyslipidemia, and hypertension, all of which are core components of cardiometabolic disease[29-34].

Blood cholesterol levels, including low-density lipoprotein (LDL), high-density lipoprotein (HDL), and triglycerides, are critical markers of cardiometabolic health. Elevated LDL cholesterol and triglycerides, along with low HDL cholesterol, characterize dyslipidemia, a core component of metabolic syndrome and a major risk factor for atherosclerosis and cardiovascular diseases. Blood glucose levels and insulin function are tightly regulated in healthy individuals but are often disrupted in cardiometabolic diseases[20,35-38]. Elevated fasting blood glucose and impaired glucose tolerance are hallmarks of prediabetes and type 2 diabetes. Chronic hyperglycemia results from insulin resistance and/or insufficient insulin secretion by the pancreas[39]. High blood glucose levels can damage blood vessels, leading to cardiovascular complications. Insulin, a hormone produced by the pancreas, facilitates glucose uptake by cells for energy production[40]. In cardiometabolic diseases, insulin resistance impairs this process, leading to elevated blood glucose levels and increased demand for the pancreas to produce more insulin[41-43].

The Homeostasis Model Assessment of Insulin Resistance (HOMA-IR) is a widely used method to estimate insulin resistance from fasting glucose and insulin levels. Insulin resistance is a condition in which cells become less responsive to insulin, leading to higher blood glucose and insulin levels. HOMA-IR provides an index of this resistance, with higher values indicating greater insulin resistance[24-25]. Insulin resistance is a central feature of metabolic syndrome and is closely associated with obesity, particularly abdominal obesity, and other cardiometabolic risk factors[20,44].

Fasting blood lactate levels can serve as a valuable biomarker for cardiometabolic health, as elevated levels are associated with insulin resistance, metabolic syndrome, and cardiovascular diseases. Increased fasting lactate often reflects impaired glucose metabolism and insulin resistance, as insulin-resistant tissues rely more on anaerobic metabolism, producing excess lactate even in the absence of hypoxia[45]. This elevation is also linked to metabolic syndrome, characterized by central obesity, dyslipidemia, hypertension, and hyperglycemia, highlighting systemic metabolic dysregulation and mitochondrial inefficiency. High fasting lactate levels are further associated with increased cardiovascular risk, potentially indicating underlying endothelial dysfunction, systemic inflammation, and altered oxidative metabolism. Elevated lactate may also signify mitochondrial dysfunction, a hallmark of many cardiometabolic diseases, as impaired mitochondrial energy production leads to greater lactate generation from glycolysis[46-49]. Chronic low-grade inflammation, commonly observed in cardiometabolic conditions, can also contribute to higher lactate levels through altered energy metabolism. Moreover, fasting lactate levels have prognostic value, as elevated levels predict worse outcomes in individuals with diabetes, heart failure, and other cardiometabolic conditions[48]. While fasting lactate is a useful marker, it is non-specific and should be interpreted in conjunction with other clinical parameters for comprehensive risk assessment and diagnosis.

The interrelationship between body composition, blood cholesterol, glucose, insulin, lactate, and HOMA-IR underpins the pathophysiology of cardiometabolic disease. Excess adiposity contributes to insulin resistance and dyslipidemia through various mechanisms,

including increased release of free fatty acids and pro-inflammatory cytokines from adipose tissue[50-51]. Insulin resistance exacerbates hyperglycemia and prompts compensatory hyperinsulinemia, which can further impair lipid metabolism and promote hypertension and are key processes in the development of cardiovascular diseases[52-54]. Collectively, these interconnected factors create a vicious cycle that perpetuates and exacerbates cardiometabolic risk. Effective management of cardiometabolic diseases involves addressing these underlying risk factors through lifestyle interventions such as physical activity and weight management, as well as pharmacological treatments to improve lipid profiles, blood glucose levels, and insulin sensitivity[55-56]. Understanding the complex interplay among these factors, particularly the role of obesity is crucial for developing comprehensive strategies to prevent and treat cardiometabolic diseases[55].

II. Changes in body composition, blood lipids, glucose, and lactate during pregnancy

Changes in maternal body composition:

During pregnancy, body composition undergoes significant changes to support the growing fetus and prepare the mother for childbirth and lactation. On average, women gain about 25-35 pounds during pregnancy, although this can vary depending on pre-pregnancy weight and whether the pregnancy involves multiple fetuses. This weight gain is distributed among the fetus, placenta, amniotic fluid, increased breast tissue, additional blood volume, and stored fat[57]. Fat mass increases notably, particularly in the first and second trimesters, as the body stores energy to support fetal growth and prepare for breastfeeding. This fat accumulation is typically seen in the abdominal, thigh, and hip

areas. Concurrently, lean body mass, including muscle, bones, organs, and fluids, also rises to support the additional weight and physiological demands of pregnancy. Gestational weight gain and fat distribution during pregnancy are both important markers for maternal and fetal health. The primary purpose of weight gain during pregnancy is to provide adequate nutrients and energy for the growth and development of the fetus[58]. However, weight gain above the appropriate limit can lead to health complications for both mother and fetus. Such complications can include hypertension, gestational diabetes, preterm birth, low birth weight, and large-for-gestational-age infants[59]. Overall, these changes in body composition are crucial for the healthy development of the fetus and the well-being of the mother, highlighting the importance of these physiological adjustments and ensuring a healthy pregnancy outcome. Proper weight management and nutritional intake during pregnancy are crucial for achieving these goals and ensuring a successful pregnancy outcome.

Blood lipid, glucose, lactate, and insulin changes during pregnancy:

During pregnancy, cholesterol levels undergo significant changes due to hormonal fluctuations and metabolic adaptations essential for fetal development and maternal health. In the early stages of pregnancy, total cholesterol levels typically increase. This rise is primarily due to elevated levels of lipoproteins, including low-density lipoprotein (LDL) and high-density lipoprotein (HDL). The increase in cholesterol is necessary for several reasons. LDL cholesterol provides essential building blocks for the synthesis of cell membranes and hormones, while HDL cholesterol helps to transport cholesterol away from the tissues and back to the liver[60-61]. Total cholesterol levels generally rise by about

25-50% during pregnancy. This increase is attributed to elevated levels of estrogen, which stimulates the liver to produce more lipoproteins[61]. Specifically, LDL cholesterol, which is often referred to as “bad” cholesterol, tends to increase, while HDL cholesterol, known as “good” cholesterol, also rises but not to the same extent. Despite this increase in LDL cholesterol, the overall lipid profile is in a physiological range that supports pregnancy[62]. Triglyceride levels also rise during pregnancy, which is associated with increased fat storage and energy needs for fetal growth. Triglycerides are a type of fat found in the blood, and their levels can increase by about 30-50% during pregnancy. This rise supports the additional caloric needs and helps provide energy for the developing fetus[60-62].

Fasting plasma lactate levels during pregnancy provide valuable insights into maternal and fetal metabolism. Lactate, a byproduct of anaerobic metabolism, typically decreases during pregnancy due to several physiological adaptations and increased metabolic demands. As pregnancy progresses, maternal metabolism increases to support the growing fetus, leading to enhanced aerobic metabolism and reduced reliance on anaerobic processes that produce lactate[63]. Additionally, improved oxygen delivery and utilization, due to increased blood volume and cardiovascular changes, contribute to lower lactate production[63-66]. Changes in glucose metabolism also play a role; pregnancy involves alterations such as insulin resistance and modified glucose utilization, which can influence lactate levels. Furthermore, the body’s ability to clear lactate from the bloodstream improves during pregnancy, thanks to enhanced liver and muscle function, which processes and removes lactate more efficiently[66]. While decreased fasting plasma lactate levels are generally normal, significant deviations can signal potential issues.

Elevated lactate levels might be associated with conditions like maternal hypoxia, impaired glucose metabolism, or preeclampsia[63-66]. Monitoring these levels can be an important aspect of assessing overall metabolic health, especially if there are symptoms or concerns about maternal or fetal well-being. Overall, the changes in fasting plasma lactate during pregnancy reflect the body's complex adjustments to meet the needs of both the mother and the developing fetus.

During pregnancy, insulin levels and the Homeostasis Model Assessment of Insulin Resistance (HOMA-IR) are critical indicators of metabolic changes and insulin sensitivity. Insulin levels typically rise during pregnancy to meet the increased metabolic demands of both the mother and the growing fetus. This increase is a response to hormonal changes produced by the placenta, which induce insulin resistance, particularly in the second and third trimesters. This insulin resistance ensures a steady supply of glucose to the fetus, prompting the mother's body to produce more insulin to maintain normal blood glucose levels[100-102]. HOMA-IR, which estimates insulin resistance based on fasting glucose and insulin levels, generally increases during pregnancy due to this insulin resistance. Elevated HOMA-IR values reflect the body's need to produce more insulin to counteract reduced insulin sensitivity and maintain glucose homeostasis. While some degree of insulin resistance is normal, significantly high HOMA-IR values can signal a higher risk of developing gestational diabetes mellitus (GDM), a condition marked by elevated blood glucose levels during pregnancy[67-70]. Monitoring insulin levels and HOMA-IR is essential for assessing metabolic health and identifying potential issues. Elevated values may indicate the need for further evaluation and management to ensure

the well-being of both mother and fetus. Regular screening for gestational diabetes and appropriate interventions, such as lifestyle modifications, are crucial for managing insulin resistance and maintaining healthy glucose levels throughout pregnancy[67].

III. Recommendations for Prenatal Exercise Guidelines

Synthesis of findings to inform exercise recommendations:

The current exercise guidelines for pregnant women emphasize the importance of regular physical activity for maintaining health and well-being during pregnancy, as outlined by the American College of Obstetricians and Gynecologists (ACOG) and other health organizations. Pregnant women are advised to complete at least 150 minutes of moderate-intensity exercise each week. Activities that increase heart rate and induce mild to moderate sweating, such as walking, swimming, stationary cycling, and low-impact aerobics, are ideal. Muscle-strengthening activities, including resistance exercises, weight training, and yoga, are also encouraged[71-72].

Safety considerations are paramount, with pregnant women advised to avoid activities that increase the risk of falling or abdominal trauma, such as contact sports, skiing, horseback riding, and scuba diving[73]. Exercises involving lying flat on the back after the first trimester should be avoided to prevent vena cava compression, which can reduce blood flow to the heart and fetus. Hydration and proper nutrition are essential, with recommendations to drink plenty of fluids before, during, and after exercise, and to maintain a balanced diet to support increased energy needs[73]. Pregnant women should

listen to their bodies and adjust activity levels as needed, stopping exercise and consulting healthcare providers if any unusual symptoms, such as dizziness, shortness of breath, chest pain, or vaginal bleeding, occur[73]. Incorporating a warm-up and cool-down period in exercise routines is important to prepare the body for activity and to gradually return it to a resting state. Before starting or continuing an exercise program, consulting with healthcare providers is crucial, particularly for those with pre-existing medical conditions or pregnancy-related complications[73].

Postpartum, women are advised to gradually resume physical activity, with specific timing and intensity to be discussed with healthcare providers, considering the mode of delivery, recovery, and overall health[74]. Regular physical activity during pregnancy offers numerous benefits, including improved mood, reduced risk of gestational diabetes and hypertension, enhanced fitness, and better weight management. Following these guidelines ensures that exercise is safe and beneficial for both mother and baby[73].

IV. Influence of Exercise on Body Composition and Blood Biomarkers during Pregnancy

Overview of blood lipid changes from exercise

Regular exercise significantly impacts blood lipid profiles, promoting healthier levels and reducing the risk of cardiovascular diseases. It helps decrease total cholesterol by improving lipid metabolism and enhancing the liver's ability to process and remove excess cholesterol from the bloodstream. Exercise is particularly effective at lowering low-density

lipoprotein (LDL) cholesterol, with aerobic activities like running, swimming, or cycling being the most beneficial. Additionally, regular physical activity increases high-density lipoprotein (HDL) cholesterol, which helps remove LDL cholesterol from the blood. Exercise also reduces triglyceride levels, a type of fat in the blood associated with cardiovascular risk, especially when combined with a healthy diet and weight loss. Moreover, it promotes a shift toward larger, less dense LDL and HDL particles, which are less likely to contribute to atherosclerosis. By enhancing the body's ability to use fat as a fuel source, exercise further improves overall lipid balance[75-76]. To optimize these benefits, individuals should engage in at least 150–300 minutes of moderate-intensity or 75–150 minutes of high-intensity aerobic exercise per week, with resistance training also playing a supportive role[77].

Impact of exercise on maternal lipid profile

Regular exercise during pregnancy can have a positive impact on a woman's lipid profile, supporting better cardiovascular health. It can help reduce total cholesterol and low-density lipoprotein levels, which naturally rise during pregnancy to support fetal development. At the same time, physical activity can increase high-density lipoprotein providing protective cardiovascular benefits. While triglyceride levels also increase during pregnancy to supply energy for the growing fetus, exercise can help moderate excessive increases, keeping them within a healthy range. Additionally, regular physical activity improves lipid metabolism, enhancing the body's ability to utilize lipids for energy and reducing the risk of lipid-related complications, such as gestational hyperlipidemia[78-81].

V. Maternal and Fetal Outcomes

The relationship between maternal body composition, blood cholesterol, glucose, insulin, and lactate levels during pregnancy and offspring outcomes is complex and significant. Increased maternal adiposity, whether due to high BMI or excessive gestational weight gain, is associated with a greater risk of fetal macrosomia (large birth weight), childhood obesity, and long-term metabolic disorders[82-84]. Conversely, maintaining a healthy body composition with adequate nutrition supports optimal fetal growth and development. Maternal cholesterol levels also play a crucial role, as elevated cholesterol during pregnancy has been linked to increased fetal lipid deposition, childhood atherosclerosis risk, and long-term cardiovascular issues. Balanced lipid levels, however, contribute to proper fetal organ development, particularly in the cardiovascular and nervous systems.

Similarly, maternal glucose levels have profound effects on offspring health. High blood glucose, especially in gestational diabetes, can lead to fetal hyperinsulinemia, excessive fat storage, and an increased risk of obesity and type 2 diabetes in childhood[79,85]. Poor glucose control has also been associated with preterm birth and metabolic syndrome in offspring. Maternal insulin levels further influence fetal development, as insulin resistance during pregnancy can impair fetal insulin sensitivity and predispose the child to obesity, insulin resistance, and diabetes later in life[79,85].

Additionally, maternal lactate levels play a role in fetal metabolism, as lactate serves as an important energy substrate for the fetus. Altered maternal lactate metabolism may impact fetal energy supply and overall development, potentially signaling underlying metabolic disturbances[86]. These maternal metabolic factors collectively influence various offspring outcomes. Children born to mothers with metabolic imbalances may face an increased risk of obesity, insulin resistance, type 2 diabetes, and dyslipidemia. They may also have a higher susceptibility to hypertension and early-onset atherosclerosis. Neurodevelopmental outcomes can also be affected, as maternal metabolic imbalances have been linked to cognitive and behavioral differences in offspring. Additionally, birth outcomes such as macrosomia, preterm birth, and neonatal complications are more common in pregnancies with poor maternal metabolic control[87-88].

Maintaining a healthy maternal body composition and metabolic profile through balanced nutrition and regular physical activity is essential for optimizing fetal development and reducing the risk of adverse outcomes in offspring. By managing cholesterol, glucose, insulin, and overall metabolic health, mothers can significantly improve their children's long-term health and well-being.

VI. Pediatric Outcomes

Influence of exercise during pregnancy and what we know related to child body composition, blood lipids, glucose, lactate, insulin and insulin resistance:

Exercise during pregnancy has profound and lasting effects on both maternal and offspring health, particularly in terms of body composition, blood lipids, glucose, insulin, and lactate metabolism. Regular maternal physical activity helps regulate weight gain during pregnancy, reducing the risk of excessive fetal growth and promoting a healthier birth weight[89]. This, in turn, influences the child's body composition by lowering fat mass and increasing lean body mass, which may contribute to a lower risk of obesity and metabolic disorders later in life. Studies suggest that children born to mothers who engaged in consistent exercise during pregnancy tend to have improved body composition and healthier weight trajectories throughout childhood and beyond[90-91].

Maternal exercise is also associated with improved blood lipid profiles, including lower maternal triglycerides and LDL cholesterol, as well as higher HDL cholesterol[78,80-81]. These favorable lipid changes create a healthier intrauterine environment, which can positively influence fetal lipid metabolism. Offspring of physically active mothers may have improved blood lipid profiles, reducing their long-term risk of cardiovascular diseases. Some research suggests that prenatal exercise helps regulate lipid transport to the fetus, thereby decreasing early-life predisposition to dyslipidemia and associated metabolic disorders[87].

Glucose regulation during pregnancy is another key area affected by maternal exercise. Physical activity enhances maternal insulin sensitivity and glucose metabolism, reducing the risk of gestational diabetes[79]. Since maternal hyperglycemia is strongly linked to excessive fetal fat deposition and macrosomia, maternal exercise can prevent these

outcomes, leading to improved metabolic health in offspring[79]. Children of mothers who exercised during pregnancy tend to have better glucose tolerance, a lower risk of insulin resistance, and a reduced likelihood of developing type 2 diabetes. Additionally, maternal exercise may enhance fetal insulin sensitivity, setting the stage for better metabolic control throughout life[79,91].

Lactate metabolism, a less commonly discussed factor, is also influenced by maternal exercise. Lactate serves as an important energy substrate for the fetus, and regular maternal physical activity improves lactate clearance and utilization. Some studies suggest that enhanced maternal lactate metabolism may positively impact fetal metabolic adaptations, promoting better energy efficiency and utilization in offspring[46-48]. Although more research is needed in this area, preliminary findings indicate that maternal exercise could help optimize fetal metabolic programming, leading to long-term benefits in energy regulation[65].

Overall, maternal exercise has significant implications for offspring health, influencing body composition, lipid metabolism, glucose regulation, insulin sensitivity, and lactate utilization. Encouraging regular, moderate-intensity exercise during pregnancy can provide lifelong metabolic and cardiovascular benefits for the child, reducing the risk of obesity, diabetes, and cardiovascular disease. Continued research is needed to further clarify the long-term effects and refine exercise recommendations for pregnant women to maximize both maternal and offspring health outcomes.

VI. Conclusion

Summary of key findings

Exercise during pregnancy is crucial for both maternal and infant health, influencing key aspects such as body composition, blood lipids, glucose, insulin, and blood lactate. For mothers, regular physical activity helps manage weight gain, reduce the risk of excessive fat accumulation, and improve overall body composition. It also supports healthy blood lipid levels by lowering triglycerides and LDL cholesterol while increasing HDL cholesterol, contributing to better cardiovascular health. Maternal exercise enhances glucose metabolism and insulin sensitivity, reducing the risk of gestational diabetes and improving long-term metabolic health.

For infants, the benefits of maternal exercise are also significant. Babies born to mothers who engage in regular physical activity tend to have healthier birth weights, with reduced risk of macrosomia (excessive birth weight) and adiposity. This helps set the stage for healthier body composition and metabolic profiles in childhood and later life. Exercise during pregnancy also positively influences fetal glucose metabolism, potentially reducing the risk of insulin resistance and type 2 diabetes in the child. Furthermore, maternal exercise enhances lactate metabolism, providing a better fetal energy supply and supporting optimal fetal development. Overall, exercise during pregnancy promotes better health outcomes for both the mother and her baby, reducing the risk of obesity, metabolic disorders, and cardiovascular disease in the long term.

Identification of gaps in the literature

The majority of maternal exercise studies primarily focus on aerobic exercise routines, limiting their scope in terms of exploring how various metrics of exercise—such as frequency, intensity, duration, and total volume—affect maternal and infant outcomes. While these studies have provided valuable insights into the general benefits of exercise during pregnancy, there remains a significant gap in understanding how the specific parameters of exercise influence maternal changes in body composition, metabolic health, and blood biomarkers throughout the pregnancy. Additionally, more research is needed to investigate how these factors impact infants once they are born, particularly about their growth, body composition, and long-term metabolic health. The current lack of comprehensive data regarding the interplay between exercise type, intensity, and duration during pregnancy and its effects on both maternal and infant health highlights the need for more detailed and diverse studies. Such research is essential for developing evidence-based recommendations for prenatal exercise that consider the full range of exercise parameters and their specific effects on both mothers and their children.

Implications for future research and practice

This research question focuses on the gaps identified in the existing literature. Specifically, it aims to explore how different maternal exercise and exercise metrics (such as frequency, intensity, duration, and total volume) influence maternal body morphology and blood biomarkers throughout pregnancy. Additionally, to seek to examine how

maternal exercise type affects infant body morphology and blood biomarkers at one and six months of age.

Dissertation Objectives

The overall objective of this dissertation was to elucidate the effects of maternal exercise type and metrics on maternal and infant body composition and blood biomarkers of obesity. Specifically, in **Aim 1** we determined the effect of maternal exercise type and metrics on 36wk and the change score 16wk – 36wk maternal body composition (BF%, FFM%, BW, and circumferences) and blood biomarkers of obesity (TC, LDL, HDL, TG, NonHDL, Lactate, Glucose, Insulin, and HOMA-IR). **Aim 2**, we determined the effect of maternal exercise type on 1-month-old infant body composition (BF%, FFM%, BW, and circumferences) and blood biomarkers of obesity (TC, LDL, HDL, TG, NonHDL, Lactate, and Glucose). Finally in **Aim 3**, we determined the effect of exercise metrics on 1- and 6-month-old infant body composition and blood biomarkers of obesity (BF%, FFM%, BW, and circumferences) and blood biomarkers of obesity (TC, LDL, HDL, TG, NonHDL, Lactate, and Glucose).

Chapter 2:

Gestational exercise metrics influence on maternal body composition and blood biomarkers

ABSTRACT:

Purpose: To assess the effects of prenatal exercise metrics on maternal blood biomarker profile and body composition. We hypothesized that any prenatal exercise type and increased volume would improve maternal blood biomarkers relative to non-exercisers.

Study Design: Healthy pregnancy women were recruited and randomly assigned to 150 min/wk of aerobic, muscular strength, or combined (aerobic+strength) moderate-intensity exercise, or a non-exercising stretching/breathing (control) group for ~24 weeks. Exercise metrics (frequency, intensity, duration, volume, attendance) were tracked across pregnancy. At 16- and 36-weeks gestation, maternal blood draw was performed to assess blood lipid profile. The differences between 36 – 16 wks are reported as the change score. Data were analyzed using analysis-of-variance and regression.

Results: Data from 290 participants was analyzed. Maternal and neonatal descriptors were similar between groups, except prepregnancy BMI. For per-protocol analysis, resistance and combination groups have lower 36 wks insulin and HOMA-IR compared to controls. Stratifying by prepregnancy BMI, aerobic and resistance exercisers have significantly lower lactate levels in both BMI groups compared to controls. Controlling for exercise attendance, 36 wk HDL is predicted (R^2=.53, p<.0001) by 16wk HDL, maternal age, and exercise group [lower in **c**ontrol]. Maternal 36 wk insulin is predicted (R^2=.90, p<.0001) by 16 wk insulin, total exercise trainings, and exercise attendance. Controlling

for gravida, 36 wk HOMA-IR is predicted R^2=.91, p<.0001) by 16 wk HOMA-IR, maternal age, prepregnancy BMI, and weekly exercise frequency. Controlling for maternal race, insulin change score is predicted (R^2=.75, p<.0001) by exercise attendance, total exercise training, and 16 wk insulin. Controlling for exercise attendance, HOMA-IR change score (R^2=.92, p<.0001) is predicted by 16 wk HOMA-IR, prepregnancy BMI, maternal age, and total exercise training. Controlling for exercise, attendance, lactate change score is predicted (R^2=.42, p<.0001) by 16 wk lactate, exercise group [↓Control, ↑Aerobic], and parity.

Conclusions: These findings suggest that an increased number of total exercise sessions across pregnancy is important in healthy maternal blood biomarkers. Additionally, resistance exercise has a positive influence on maternal blood biomarker outcomes. Overall, research should focus on exercise volume and exercise type across pregnancy for maternal health.

Introduction

Obesity is a growing global health issue, with its prevalence significantly increasing among reproductive-aged women and posing critical health risks during pregnancy. Excess adiposity in expectant mothers can lead to complications like gestational diabetes, hypertension, preeclampsia, and macrosomia, all of which can negatively impact both maternal and fetal health [7-12]. Excess adiposity during pregnancy may also have long-term health consequences for both the mother and child, emphasizing the importance of addressing obesity during this crucial life stage[7-12,15,69]. One of the

primary metabolic concerns in obese pregnancies is the dysregulation of lipids, such as cholesterol, and the altered glucose metabolism, both of which can affect fetal development and maternal health. Although cholesterol is a crucial lipid for synthesizing cell membranes and hormones, excess cholesterol can lead to adverse cardiovascular outcomes and increased oxidative stress, especially in obese pregnant women[60-62]. Furthermore, elevated lactate levels, a byproduct of anaerobic metabolism, suggest a shift in metabolic flexibility, potentially leading to insulin resistance, which is a common issue in obesity[63-66]. During pregnancy, insulin resistance is a naturally progressive occurrence to ensure glucose supply for the fetus; however, in obese pregnancies, it can be amplified, leading to gestational diabetes and other metabolic complications[67,69,-70]. The Homeostatic Model Assessment of Insulin Resistance (HOMA-IR) is frequently used to assess insulin resistance, which is often elevated in obese pregnancies and highlights the increased risk of metabolic disorders[23-26]. Body composition—the balance of fat, muscle, and bone—also plays a crucial role in health outcomes during pregnancy, as an increased muscle with less fat body composition can enhance insulin sensitivity and energy levels, which are essential for a healthy pregnancy[50-52]. Lifestyle interventions, including regular exercise, are critical for promoting healthy body composition and weight, optimizing maternal health, and supporting positive outcomes for both mother and child. Maternal exercise has emerged as a valuable factor in improving pregnancy outcomes. Regular exercise can help regulate lipid profiles, decrease lactate production, and enhance insulin sensitivity, thereby reducing HOMA-IR scores and mitigating the adverse effects of maternal obesity[78-81].

Methods

Participants and Recruitment

This randomized, blinded, prospective study is focused on determining the influence of maternal exercise types on maternal and offspring health outcomes. All protocols were approved by the East Carolina University Institutional Review Board. Recruitment is focused on low-risk, healthy women with a singleton pregnancy. Based on preliminary maternal blood biomarker data, power analysis indicated we would need a sample of 19 per group to detect group differences in fasting blood lactate. Women enrolled in the study met the following criteria: clearance from an obstetric provider to participate in exercise; between ages 18 and 40; pre-pregnancy BMI of 18.5–39.9; gestational age ≤ 16 weeks; no current alcohol, tobacco, or medication use. Criteria for exclusion were pre-existing diabetes mellitus, hypertension, cardiovascular disease, and comorbidities known to affect fetal growth and well-being. Written informed consent is obtained from each participant; each participant subsequently completed a treadmill test and 1-repetition maximum strength tests. Using concealed randomization methods, participants were randomly assigned to aerobic, resistance, combination (aerobic + resistance) exercise, or stretching-breathing attention control. Due to the nature of an exercise intervention, the participants and trainers knew the group assignment.

Pre-Intervention Exercise Testing

Participants recruited before March 2020, the COVID-19 pandemic, completed a submaximal modified Balke treadmill test following previously described protocols[92] to determine the target heart rate (HR) zone for exercise training. To minimize exposure and potential risk associated with COVID-19, women recruited following March 2020 did not complete the treadmill protocol, and target HR zones for aerobic exercise were determined based on their pre-pregnancy physical activity level and age, using published guidelines[92]. Target HR zones for the aerobic exercise intervention corresponded to maternal HR at 60 to 80% of maximal oxygen consumption, reflecting moderate intensity[92].

Exercise Intervention

Participants trained according to American College of Obstetricians and Gynecologists guidelines for approximately 24 weeks, beginning at 16 weeks gestation and continuing until delivery (~40 weeks gestation)[93]. Each exercise training session included a 5-minute warmup, a 50-minute exercise period, and a 5-minute cooldown. Every training session, regardless of training type, is supervised; participants exercised within their target HR zone corresponding to moderate intensity (60%-80% maximal oxygen consumption and 12-14 rated perceived exertion) throughout the exercise session. HR is monitored (Polar FS2C HR Monitor) throughout each exercise session to ensure that participants remain within their target HR ranges for the duration of the session. All sessions were performed three times per week until delivery. The aerobic exercise (AE) group completed moderate-intensity training for 50 minutes on the treadmill, elliptical,

recumbent bicycle, rowing, and/or stair-stepping equipment. To maintain the appropriate HR zone, speed, grade, and/or resistance were adjusted. The resistance exercise (RE) group completed sessions consisting of two to three sets of 15 repetitions of each exercise at moderate intensity[94]. Seated Cybex machines (leg extension, leg curl, shoulder press, chest press, triceps extension, latissimus dorsi pull-down), dumbbells (biceps curls, lateral shoulder raises, front shoulder raises), resistance bands, dumbbells, exercise balls, benches, and/or mats were used. The combination exercise (CE) group performed five 4.5-5 minutes long alternating bouts of aerobic and resistance exercise, each for 50 minutes per workout. The CE group performed aerobic and resistance exercises on the same equipment as the AE and RE groups. The control (CON) group performed supervised stretching, breathing, and flexibility exercises for the duration of the session. The breathing exercises and stretches of CON participants targeted major muscle groups[94] and maintained a low intensity (<40% VO_{2peak}) throughout the session. Maternal exercise intensity (METs)is determined based on the published compendium of physical activity for the exercise performed in each session[95]. Average maternal exercise dose throughout pregnancy, expressed as MET·min/wk, is quantified (frequency X duration of session) and then multiplied by the intensity (METs) level of their exercise. The ability of this protocol to elicit maternal physiological adaptations to exercise has been previously reported[96].

Maternal Measurements

Maternal age, gravida, parity, pre-pregnancy weight and height, gestational weight gain (GWG), length of gestation, infant sex, birth length, and birth weight were abstracted from

various sources, including pre-screening eligibility, postpartum questionnaires, as well as maternal and neonatal electronic health records. Exercise metrics such as frequency, intensity, duration, and type were recorded from the training logs across pregnancy. Gestational weight gain is calculated by subtracting maternal pre-pregnancy weight from weight at delivery and is extracted from electronic health records, while weight before pregnancy (lbs.) is collected via the pre-screening eligibility questionnaires. At 16 and 36 weeks of gestation, maternal height and weight were obtained using a stadiometer and calibrated medical grade scale; BMI is calculated as [weight (kg)] / [height (m) ^{2}] (healthy BMI 18.5-24.9; overweight 25-29.9, obese $\geq$30). Maternal percent body fat is determined using a previously validated skinfold technique and age-adjusted equations; sites include triceps, subscapular, supra iliac, and thigh[97-98]. Circumferences were gathered at minimum waist and hip. Further, non-fasting lipid profiles were assessed at enrollment and 36 weeks gestation. Blood samples were acquired from a peripheral vein and then centrifuged and stored using standard procedures as described previously. Plasma samples were assayed in duplicates. Two drops of blood from the sample were used to measure glucose, lipids (NonHDL, TC, TG, HDL, LDL), and lactate with a Cholestech and lactate analyzer, respectively. Plasma vacutainers were immediately centrifuged in a Fisher Scientific accuSpin 1R at 4 degrees Celsius. After centrifugation, plasma samples were aspirated by use of a pipette into a micro-vial for storage at -80 degrees Celsius until analysis. Plasma samples were used via Luminex xMAP technology (Millipore, Darmstadt, Germany) and chemiluminescence immunoassay (Beckman Coulter Unicel DXL 800 Access Analyzer, California, USA) to evaluate maternal insulin levels within the plasma samples. A custom MAP 11-plex Human Cytokine/Adipokine Magnetic Bead

Panel Kit, which includes an Anti-Human Insulin Bead, is used. The 96-well plate is analyzed on Luminex 200 (EMD Millipore, San Jose, CA) using Milliplex Software (EMD Millipore, San Jose, CA) for data analysis. HOMA-IR is calculated using the established formula: $HOMA - IR = \left[insulin_{\frac{\mu IU}{mL}} \cdot glucose_{\frac{mg}{dL}}\right] \div 405$ The change in pregnancy blood biomarker levels and body composition is calculated by subtracting the 16-week (enrollment) values from the 36-week level.

Statistical Analysis

ANOVA is used to determine the significance between groups where appropriate. Kruskal-Walls (4-group comparison) and Mann-Whitney (2-group comparison) tests were used to assess the difference between not normally distributed variables (gravida and parity). Linear Regressions were completed to assess predictors of maternal body composition and biomarker (lipid, glucose, insulin, HOMA-IR) outcomes. ANCOVA is used to control for maternal covariates, such as pre-pregnancy BMI. Statistical significance is set at $p \leq 0.05$. Statistical analyses were performed using IBM SPSS Statistics (IBM Corp. in Armonk, NY) and JMP Statistical Discovery (Cary, NC) for Mac.

Results

Maternal and infant birth descriptors. We were contacted by 454 pregnant women interested in the study (Figure 1). Our final analysis included 272 participants (CON n=68, AE n=82, RE n=57, or AERE n=65). The intervention is conducted from September 1, 2018, to January 30, 2023. For the intention-to-treat (ITT) analysis, participants were similar in age, gravida, and parity. Pre-pregnancy BMI is significantly higher in the control group, and Maternal exercise metrics were substantially lower in the Control group, as expected by group protocol (Table 2.1). For the per-protocol analysis of the exercising women, a total of 184 women met ≥75% adherence to exercise training throughout the ~24 weeks (AE n=46, RE n=30, AERE n=40). There were no differences between groups for age, gravida, or parity. Pre-pregnancy BMI is significantly higher in the control group, and maternal exercise metrics were substantially lower in the control group, as expected based on group protocol (Table 2.2).

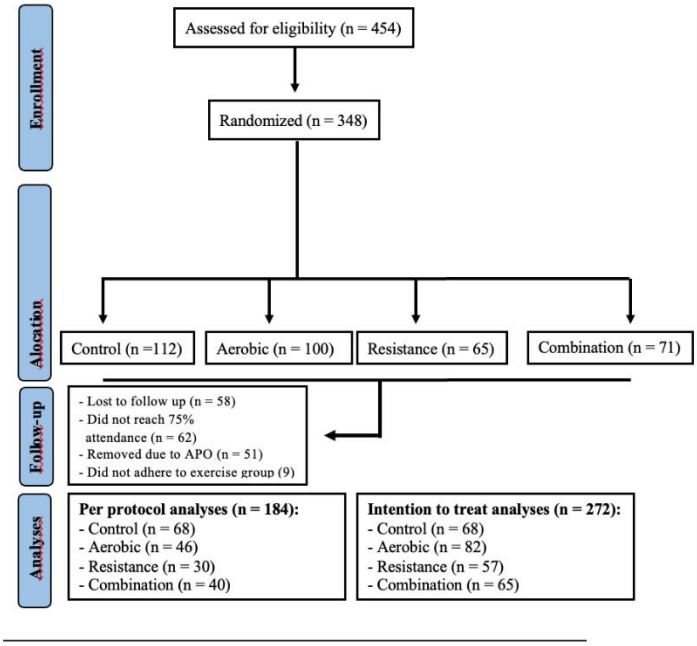

Fig. S1 Flow chart of the study participants.

Table 2.1: Intention to Treat Maternal and Birth Descriptors						
	Full Sample (N=272)	Control (N=68)	Aerobic (N=82)	Resistance (N=57)	Combination (N=65)	p-value
Maternal Measures						

Age (years)	30.49 ± 4.31	30.25 ± 4.44	30.36 ± 4.45	31.17 ± 4.06	30.38 ± 4.20	0.64
Pre-pregnancy BMI (kg/m^2)	25.99 ± 5.01	27.25 ± 5.55	25.04 ± 4.94**	25.24 ± 4.21	26.00 ± 4.59	**0.01**
Gravida†	2.0 (1, 8)	2.0 (1, 4)	2.0 (1, 4)	2.0 (1, 5)	2.0 (1, 8)	0.59
Parity†	1.0 (0, 4)	1.0 (0, 3)	1.0 (0, 2)	1.0 (0, 4)	1.0 (0, 3)	0.60
Attendance (percentage)	86.58 ± 18.83	--	78.13 ± 19.94	77.70 ± 19.80	82.42 ± 19.97	0.40
Race (n,%)						0.23
White	242 (83.3%)	57 (83.8%)	66 (78.7%)	53 (90.6%)	40 (93%)	
Black	29 (10%)	8 (11.8%)	7 (8.33%)	4 (6.3%)	2 (4.7%)	
Other	19 (6.7%)	3 (4.4%)	8 (10.6%)	2 (3.1%)	1 (2.3%)	
GWG (lbs)	30.19 ± 11.45	28.87 ± 12.76	29.66 ± 10.43	32.42 ± 10.47	31.10 ± 10.56	0.20
Length of Gestation (wks)	39.69 ± 1.08	39.41 ± 1.26	39.74 ± 1.06	39.94 ± 0.90	39.63 ± 1.09	0.29
Maternal Exercise Measures						
Average METs (METs)	3.94 ± 0.49	2.93 ± 0.31***	3.98 ± 0.49	3.92 ± 0.49	4.06 ± 0.52	**<.001**
Weekly Frequency (days)	2.51 ± 0.48	2.21 ± 0.93*	2.46 ± 0.52	2.51 ± 0.39	2.63 ± 0.37	**.025**
Weekly Duration (minutes)	129.35 ± 26.56	114.89 ± 43.11**	130.32 ± 24.62	130.32 ± 24.62	137.86 ± 20.94	**.003**
Total # of Pregnancy Trainings	51.25 ± 15.09	50.82 ± 21.43	52.48 ± 13.42	52.80 ± 14.94	51.25 ± 15.09	.458
Total Exercise Volume (METminutes)	10135.64 ± 3887.09	6835.94 ± 3850.48**	9761.21 ± 3595.15	10425.37 ± 3782.04	10885.58 ± 4094.01	**.008**
Infant Measures						
Infant Sex (n,% Male)	153 (52.7%)	39 (57.4%)	45 (55.3%)	32 (56.2%)	25 (39.5%)	0.76
Birth Length (m)	0.50 ± 0.02	3.54 ± 0.57	0.49 ± 0.03	0.50 ± 0.02	0.50 ± 0.02	0.35
Birth Weight (kg)	3.56 ± 0.46	0.50 ± 0.03	3.52 ± 0.41	3.60 ± 0.48	3.58 ± 0.42	0.87

Presented as mean +/- SD. †due to non-normal distribution, Mann Whitney U test performed and expressed as median (minimum, maximum). GWG - Gestational Weight Gain, BMI - Body Mass Index. *p < 0.05, **p < 0.01, p*** < 0.001 relative to control group.

Table 2.2: Per Protocol - Maternal and Birth Descriptors

	Control (n=68)	Aerobic (N=46)	Resistance (N=30)	Combination (N=40)	p-value
Maternal Measures					
Age (years)	30.25 ± 4.44	31.56 ± 4.42	30.84 ± 2.87	30.31 ± 4.34	0.17
Pre-pregnancy BMI (kg/m^2)	27.25 ± 5.55	24.03 ± 3.98 **	23.93 ± 2.87 *	25.71 ± 3.73	**0.004**
Gravida†	2.00 (1, 4)	2.00 (1, 4)	2.00 (1,4)	2.00 (1,3)	0.23
Parity†	1.00 (0,3)	1.00 (0, 2)	1.00 (0, 3)	1.00 (0, 1)	0.06
Attendance (Percentage)	--	90.06 ± 0.80	88.62 ± 0.80	90.14 ± 0.80	0.26
Race					0.18
White	57 (83.8%)	36 (78.3%)	26 (86.7%)	38 (95.0%)	
Black	8 (11.8%)	5 (10.9%)	1 (3.3%)	1 (2.5%)	
Other	3 (4.4%)	5 (10.9%)	3 (10%)	1 (2.5%)	
GWG (lbs)	28.87 ± 12.76	29.26 ± 9.08	33.22 ± 9.84	31.94 ± 10.01	0.23
Length of Gestation (wks)	39.41 ± 1.26	39.83 ± 0.99	40.00 ± 0.79	39.70 ± 0.95	0.20
Average METs (METs)	2.93 ± 0.31***	4.05 ± 0.47	3.95 ± 0.09	4.02 ± 0.58	**<.001**
Week Frequency (days)	2.21 ± 0.93*	2.68 ± 0.29	2.67 ± 0.25	2.74 ± 0.34	**<.001**
Week Duration (minutes)	114.89 ± 43.11**	135.90 ± 15.63	139.43 ± 16.37	142.34 ± 20.54	**<.001**
Total Trainings	50.82 ± 21.43	56.60 ± 12.13	59.64 ± 9.32	60.00 ± 9.85	**.006**
Total Exercise Volume (METminutes)	6835.94 ± 3850.48**	11562.60 ± 2970.62	12358.07 ± 2741.74	12288.77 ± 3851.80	**<.001**
Infant Measures					
Infant Sex (Male)	39 (57.4%)	29 (63.0%)	15 (51.72%)	17 (42.5%)	0.54
Birth Weight (kg)	3.54 ± 0.57	3.51 ± 0.43	3.57 ± 0.44	3.59 ± 0.42	0.91
Birth Length (m)	0.50 ± 0.03	0.49 ± 0.03	0.50 ± 0.02	0.50 ± 0.02	0.20

Presented as mean +/- SD. † due to non-normal distribution, Mann Whitney U test performed and expressed as median (minimum, maximum). GWG - Gestational Weight Gain. *p < 0.05, **p < 0.01, p*** < 0.001 compared to control

16-week Maternal Outcomes. For the per protocol, there were between-group differences in pre-pregnancy BMI (p=<.001; CON to AE and RE), Subscapular (p-value=.003; CON to RE), and suprailiac (p-value=.01; CON to RE) skinfold thickness, body fat percent (p=.04; CON to RE), hip circumference (p=.02; CON to AE), and total cholesterol to HDL cholesterol ratio (p=.02; CON to AERE) (Table 2.3).

Table 2.3: Per Protocol - 16wk Maternal Measures

	Control (n=68)	Aerobic (n=46)	Resistance (n=29)	Combination (n=40)	p-value
Weight (lbs)	165.39 ± 32.05	152.06 ± 28.84	156.80 ± 24.33	159.71 ± 22.28	.21
BMI	27.43 ± 5.38	24.24 ± 4.19*	23.93 ± 4.18**	25.71 ± 3.73	<.001
Maternal Skinfolds (mm)					
Triceps	24.54 ± 7.84	22.71 ± 7.32	21.99 ± 5.87	22.36 ± 7.23	.40
Subscapular	22.29 ± 7.75	21.45 ± 8.20	16.19 ± 6.44**	18.16 ± 6.52	.003
Suprailiac	24.38 ± 8.16	20.47 ± 7.72	19.30 ± 5.76*	20.08 ± 6.91	.01
Thigh	36.15 ± 10.35	34.83 ± 10.90	33.69 ± 10.20	32.71 ± 10.88	.50
Sum	105.38 ± 32.70	99.46 ± 29.55	91.16 ± 23.22	93.31 ± 27.57	.15
Fat Free Mass Percent	65.32 ± 4.37	66.97 ± 4.46	68.01 ± 3.51	67.61 ± 4.33	.07
Body Fat Percent	34.70 ± 4.10	33.27 ± 4.74	32.04 ± 3.44*	32.53 ± 4.29	.04
Maternal Circumferences					
Waist (in)	34.48 ± 8.36	30.43 ± 6.73	31.35 ± 2.84	31.00 ± 5.82	.09
Hip (in)	43.67 ± 9.57	39.22 ± 5.46*	40.83 ± 3.14	39.51 ± 6.50	.02
WHR	0.78 ± 0.07	0.78 ± 0.13	0.77 ± 0.03	0.78 ± 0.07	.90
Maternal Blood Lipids and markers					
TC (mg/dL)	182.12 ± 28.23	175.22 ± 32.53	173.12 ± 29.90	174.43 ± 28.58	.72
HDL (mg/dL)	63.83 ± 15.06	54.64 ± 18.36*	65.96 ± 11.16	64.07 ± 11.90	.02
LDL (mg/dL)	100.63 ± 25.12	98.92 ± 30.19	88.92 ± 24.40	92.04 ± 28.61	.39
TG (mg/dL)	104.40 ± 30.82	101.89 ± 39.30	91.21 ± 31.02	102.50 ± 55.43	.67
NonHDL (mg/dL)	120.92 ± 26.67	119.41 ± 32.43	107.25 ± 26.90	111.93 ± 31.58	.33
TC/HDL ratio	3.05 ± 0.82	3.56 ± 1.60	2.70 ± 0.67*	2.87 ± 0.78	.02
Lactate (mmol/L)	1.16 ± 1.08	1.00 ± 0.48	0.96 ± 0.49	0.93 ± 0.45	.65
Insulin (mU/L)	5.39 ± 6.82	2.95 ± 2.18	0.84 ± 0.68	2.51 ± 3.49	.18
Glucose (mg/dL)	81.28 ± 8.20	79.68 ± 7.22	80.29 ± 5.65	80.86 ± 7.23	.86
HOMA-IR	1.09 ± 1.33	0.60 ± 0.42	0.16 ± 0.13	0.52 ± 0.83	.16

FFM: Fat Free Mass, BMI: Body Mass Index, SkF: Skin Fold, WHR: Waist to Hip Ratio, BF%: Body Fat Percent, TC: Total Cholesterol, HDL: High-Density Lipoprotein, LDL: Low-Density Lipoprotein, TG: Triglycerides, NonHDL: Non-High-Density Lipoprotein *p < 0.05, **p < 0.01, p*** < 0.001 relative to control group.

36-week and 16 - 36 wk Maternal Outcomes. For the per-protocol analysis, we found differences for 36 wk hip circumference *(p=.02; CON to AE)*, waist-to-hip ratio *(p=.03; CON to AE),* fasting insulin *(p=.01; CON to RE),* and HOMA-IR *(p=.02; CON to RE and CON to AERE)* (Table 2.4). We also found trends or significant differences in the 16wk to 36wk change score for the sum of skinfolds *(p=.05; CON to AE),* body fat percent *(p=.04; CON to AE)*, fasting lactate *(p=.003; CON to AE and CON to RE),* and fasting insulin *(p=.03; CON to RE and CON to AERE)* (Table 2.5).

Table 2.4: Per Protocol - 36wk Measures ANOVA

	Control (n=68)	Aerobic (n=46)	Resistance (n=29)	Combination (n=40)	p-value
Weight (lbs)	184.51 ± 27.50	173.58 ± 20.01	179.04 ± 26.61	185.43 ± 25.12	.15
BMI (kg/m^2)	30.67 ± 3.99	28.56 ± 3.24	29.04 ± 4.53	30.36 ± 3.72	.06
Maternal Skinfolds (mm)					
SkF Triceps	24.00 ± 6.70	21.43 ± 6.07	24.63 ± 7.30	23.28 ± 7.09	.22
SkF Subscapular	23.76 ± 7.94	21.97 ± 8.34	19.72 ± 8.68	24.09 ± 7.17	.13
Skf Suprailiac	24.27 ± 8.46	20.43 ± 7.90	22.47 ± 8.26	23.73 ± 7.26	.17
Skf Thigh	37.93 ± 11.76	35.14 ± 10.68	37.49 ± 11.03	37.99 ± 9.89	.63
Skf Sum	107.87 ± 26.10	98.31 ± 28.23	103.69 ± 30.64	109.09 ± 25.05	.32
Fat Free Mass (lbs)	120.04 ± 14.08	114.53 ± 10.33	117.75 ± 14.58	120.50 ± 12.54	.23
Fat Free Mass Percent	64.40 ± 3.28	66.61 ± 4.01	66.39 ± 4.16	65.12 ± 3.63	.10
Body Fat Percent	34.99 ± 3.63	33.51 ± 3.89	33.77 ± 4.17	34.85 ± 3.58	.27
Maternal Circumferences					
Waist (in)	36.42 ± 3.38	33.83 ± 4.71	34.48 ± 3.38	35.21 ± 4.95	.11
Hip (in)	43.47 ± 2.84	41.22 ± 2.53*	42.17 ± 2.77	42.90 ± 3.29	.02
WHR	0.82 ± 0.04	0.82 ± 0.06	0.80 ± 0.04*	0.83 ± 0.04	.03
Maternal Blood Lipids and markers					
TC (mg/dL)	229.75 ± 35.15	235.96 ± 51.90	225.10 ± 40.41	232.87 ± 43.50	.87
HDL (mg/dL)	59.19 ± 16.89	62.04 ± 15.67	69.05 ± 13.23	68.14 ± 14.46	.14
LDL (mg/dL)	126.63 ± 37.24	133.15 ± 50.29	125.60 ± 43.10	123.41 ± 42.83	.88
TG (mg/dL)	211.47 ± 47.28	199.04 ± 59.84	177.76 ± 45.09	194.24 ± 49.38	.27

NonHDL (mg/dL)	170.38 ± 38.83	173.92 ± 54.44	160.90 ± 46.11	166.57 ± 46.92	.83
TC/HDL ratio	3.76 ± 1.06	3.80 ± 1.12	3.38 ± 0.97	3.58 ± 1.08	.60
Lactate (mmol/L)	1.31 ± 0.48	1.12 ± 0.44	1.01 ± 0.37	1.17 ± 0.38	.29
Insulin (mU/L)	8.70 ± 8.67	4.11 ± 3.34	1.06 ± 1.12*	1.66 ± 1.10*	.01
Glucose (mg/dL)	80.13 ± 8.03	78.58 ± 7.96	78.19 ± 8.88	79.21 ± 6.69	.89
HOMA-IR	1.83 ± 1.83	0.84 ± 0.67	0.21 ± 0.26*	0.35 ± 0.26*	.02
FFM: Fat Free Mass, BMI: Body Mass Index, SkF: Skin Fold, WHR: Waist to Hip Ratio, BF%: Body Fat Percent, TC: Total Cholesterol, HDL: High-Density Lipoprotein, LDL: Low-Density Lipoprotein, TG: Triglycerides, NonHDL: Non-High-Density Lipoprotein *p < 0.05, **p < 0.01, p*** < 0.001 relative to control group.					

Table 2.5: Per Protocol - 16-36wk Measures ANOVA					
	Control	Aerobic	Resistance	Combination	p-value
GWG (lbs)	28.87 ± 12.76	28.93 ± 9.25	33.22 ± 9.84	31.94 ± 10.01	.20
BMI (kg/m^2)	4.34 ± 1.54	4.66 ± 1.40	5.11 ± 1.45	4.82 ± 2.06	.31
Maternal Skinfolds					
Skinfold Sum (mm)	7.21 ± 22.29	1.94 ± 22.41*	13.96 ± 20.58	15.36 ± 21.53	.05
Fat Free Mass (lbs)	5.43 ± 27.41	13.88 ± 5.44	12.66 ± 6.65	11.03 ± 4.34	.14
Fat Free Mass Percent (lbs)	-0.93 ± 3.38	-0.58 ± 3.14	-1.83 ± 3.02	-2.21 ± 3.36	.19
Body Fat Percent	0.72 ± 3.10	0.24 ± 3.09*	1.97 ± 3.02	2.20 ± 3.31	.04
Maternal Circumferences					
WHR	0.04 ± 0.04	0.03 ± 0.04	0.03 ± 0.04	0.04 ± 0.05	.91
Maternal Blood Lipids and markers					
TC (mg/dL)	48.75 ± 34.83	53.05 ± 23.97	54.15 ± 34.90	55.55 ± 33.99	.93
HDL (mg/dL)	-0.53 ± 7.45	6.54 ± 11.39	1.90 ± 12.88	3.95 ± 12.17	.27
LDL (mg/dL)	23.67 ± 27.77	33.50 ± 33.87	31.79 ± 31.32	28.50 ± 32.59	.81
TG (mg/dL)	108.13 ± 41.84	106.13 ± 50.66	84.33 ± 36.47	102.70 ± 40.77	.28
NonHDL (mg/dL)	44.53 ± 28.23	48.55 ± 29.80	48.00 ± 33.39	51.45 ± 33.63	.93
TC/HDL ratio	0.70 ± 0.43	0.49 ± 0.78	0.64 ± 0.80	0.90 ± 1.08	.45
Lactate (mmol/L)	0.71 ± 0.50	0.04 ± 0.54**	0.16 ± 0.49**	0.38 ± 0.38	.003*
Insulin (mU/L)	1.72 ± 1.10	0.91 ±1.15	0.13 ± 0.30*	0.22 ± 1.01*	.03
Glucose (mg/dL)	-2.14 ± 8.25	-1.32 ± 7.48	-2.71 ± 7.63	-1.52 ± 8.53	.94
HOMA-IR	0.26 ± 0.36	0.19 ± 0.25	0.02 ± 0.05	0.06 ± 0.25	.37
FFM: Fat Free Mass, BMI: Body Mass Index, SkF: Skin Fold, WHR: Waist to Hip Ratio, BF%: Body Fat Percent, TC: Total Cholesterol, HDL: High-Density Lipoprotein, LDL: Low-Density Lipoprotein, TG: Triglycerides, NonHDL: Non-High-Density Lipoprotein *p < 0.05, **p < 0.01, p*** < 0.001 relative to control group.					

ANCOVA Maternal Outcomes. For the per protocol, ANCOVA analysis, we found differences for 16-36 wk lactate *(p=.02; CON to AE, p=.04; CON to RE)* when controlling for maternal prepregnancyBMI (Table 2.6).

Table 2.6. 36wk Maternal ANCOVA

	Estimate	STD Error	t Ratio	Lower 95%	Upper 95%	p-value
16-36wk Lactate (mmol/L)	**p = .98**					
16wk Lactate						<0.001
Exercise Group [Aerobic]						0.02
Exercise Group [Resistance]						.04

Regressions for Maternal Morphometric Outcomes. Controlling for the exercise group, 36wk body weight is predicted (R^2=0.89, p=<.0001) by 16wk weight and total exercise volume. 36wk body weight is predicted (R^2=0.89, p=<.0001) by 16wk weight, weekly exercise duration, and weekly exercise frequency. Controlling for exercise attendance, exercise group, maternal age, and race, 36wk fat-free mass percent is predicted (R^2=.061, p=<.0001) by parity, 16wk fat-free mass percent, prepregnancy BMI, weekly exercise duration, and weekly exercise frequency. Controlling for maternal race, exercise attendance, and maternal race, 36wk body fat percent is predicted (R^2=.059, p=<.0001) by 16wk body fat percent, parity, prepregnancy BMI, weekly exercise frequency, weekly exercise duration, and exercise group [aerobic]. Controlling for the exercise group, 36wk triceps skinfold thickness is predicted (R^2=0.46, p=<.0001) by 16wk triceps skinfold thickness, weekly exercise duration, weekly exercise frequency, parity, and prepregnancy

BMI. Controlling for prepregnancy BMI and gravida, 36wk triceps skinfold thickness is predicted (R^2=0.47, p=<.0001) by 16wk triceps skinfold thickness, parity, total exercise volume, and exercise group [control]. Controlling for prepregnancy BMI, and maternal age, 36wk subscapular skinfold thickness is predicted (R^2=0.47, p=<.0001) by 16wk subscapular skinfold thickness, weekly exercise frequency, and total exercise training. Controlling for maternal race, and weekly exercise duration, 36wk supra iliac skinfold thickness is predicted (R^2=0.60, p<.0001) by prepregnancy BMI, parity, 16wk supra iliac skinfold thickness, exercise attendance, weekly exercise frequency, and exercise group [aerobic]. Controlling for prepregnancy BMI and maternal race, 36wk thigh skinfold thickness is predicted (R^2=0.43, p=<.0001) by 16wk thigh skinfold thickness, total exercise volume, and exercise group [combination]. Controlling for weekly exercise duration, 36wk thigh skinfold thickness is predicted (R^2=0.41, p=<.0001) by 16wk thigh skin fold, weekly exercise frequency, total exercise training, and prepregnancy BMI. Controlling for weekly exercise duration, total exercise training, maternal age, maternal race, and gravida, the 36wk sum of skinfolds is predicted by (R^2=0.60, p=<.0001) by weekly exercise frequency, 16wk sum of skinfolds, parity, prepregnancy BMI, exercise attendance, and exercise group [aerobic]. Controlling for maternal age, average weekly exercise METs, 16wk waist circumference, maternal race, and parity, 36wk waist circumference is predicted (R^2=0.39, p=.0005) by prepregnancy BMI, weekly exercise duration, and weekly exercise frequency (Table 2.7).

Table 2.7. 36wk Maternal Body Composition Regression models

		Estimate	STD Error	t Ratio	Lower 95%	Upper 95%	p-value
36wk Body Weight[1]	**R^2 = 0.89**	**p = <0.0001**					

16wk Weight	0.99	0.04	24.20	0.91	1.07	<0.0001
Total Exercise Volume	-0.0006	0.0003	-2.00	-0.001	-0.000004	0.05
36wk Body Weight[2] R^2 = 0.89 p = <0.0001						
16wk Weight	0.98	0.04	25.31	0.91	1.06	<0.0001
Weekly Exercise Duration	-0.28	0.10	-2.68	-0.49	-0.07	0.009
Weekly Exercise Frequency	12.73	5.35	2.38	2.09	23.37	0.02
Fat Free Mass Percent[3] R^2 = 0.61 p = <0.0001						
Parity	2.16	0.66	3.27	0.83	3.49	0.002
16wk Fat Free Mass Percent	0.36	0.11	3.17	0.13	0.59	0.003
PrePregnancy Body Mass Index	-0.26	0.12	-2.22	-0.49	-0.02	0.03
Weekly Exercise Duration	0.11	0.05	2.17	0.008	0.21	0.03
Weekly Exercise Frequency	-6.26	2.93	-2.14	-12.14	-0.39	0.04
Body Fat Percent[4] R^2 = 0.59 p = <0.0001						
16wk Body Fat Percent	0.35	0.10	3.47	0.15	0.56	0.0009
Parity	-1.69	0.56	-3.04	-2.80	-0.58	0.003
PrePregnancy Body Mass Index	0.26	0.10	2.53	0.06	0.47	0.01
Weekly Exercise Frequency	5.17	2.14	2.42	0.91	9.44	0.02
Weekly Exercise Duration	-0.09	0.04	-2.19	-0.17	-0.007	0.03
Exercise Group [Aerobic]	-1.54	0.71	-2.17	-2.96	-0.12	0.03
Triceps Skin Fold[5] R^2 = 0.46 p = <0.0001						
16wk Triceps Skinfold	0.40	0.11	3.77	0.19	0.61	0.0003
Weekly Exercise Duration	-0.22	0.08	-2.89	-0.38	-0.07	0.005
Weekly Exercise Frequency	9.70	4.00	2.42	1.72	17.68	0.02
Parity	-2.01	0.99	-2.04	-3.99	-0.05	0.04
PrePregnancy Body Mass Index	0.36	0.18	1.99	-0.0006	0.71	0.05
Triceps Skin Fold[6] R^2 = 0.47 p = <0.0001						
16wk Triceps Skin Fold	0.44	0.10	4.21	0.23	0.65	<0.0001
Parity	-2.97	1.34	-2.21	-5.65	-0.29	0.03
Total Exercise Volume	-0.0004	0.0002	-2.21	-0.0008	-0.00004	0.03
Exercise Group [Control]	-4.82	2.37	-2.04	-9.54	-0.11	0.05
Subscapular Skin Fold[7] R^2 = 0.47 p = <0.0001						
16wk Subscapular Skin Fold	0.51	0.11	4.51	0.28	0.73	<0.0001
Weekly Exercise Frequency	5.91	2.82	2.10	0.30	11.52	0.04

Total Exercise Trainings	-0.19	0.10	-1.97	-0.38	0.002	0.05
Suprailiac Skin Fold[8]	**R^2 = 0.60**	**p = <0.0001**				
PrePregnancy Body Mass Index	0.61	0.19	3.16	0.23	1.00	0.002
Parity	-2.98	1.18	-2.51	-5.34	-0.61	0.01
16wk Suprailiac Skin Fold	0.30	0.12	2.46	0.06	0.54	0.02
Attendance	-25.61	10.76	-2.38	-47.07	-4.15	0.02
Weekly Exercise Frequency	9.43	4.53	2.08	0.39	18.46	0.04
Exercise Group [Aerobic]	-3.84	1.50	-2.56	-6.84	-0.85	0.01
Thigh Skin Fold[9]	**R^2 = 0.43**	**p = <0.0001**				
16wk Thigh Skin Fold	0.46	0.11	4.40	0.25	0.67	<0.0001
Total Exercise Volume	-0.0008	0.0003	-2.93	-0.001	-0.0003	0.004
Exercise Group [Combination]	3.96	1.87	2.12	0.24	7.69	0.04
Thigh Skin Fold[10]	**R^2 = 0.41**	**p = <0.0001**				
16wk Thigh Skin Fold	0.38	0.10	3.74	0.18	0.57	0.0003
Weekly Exercise Frequency	12.42	5.27	2.36	1.95	22.90	0.02
Total Exercise Trainings	-0.32	0.13	-2.35	-0.58	-0.05	0.02
PrePregnancy Body Mass Index	0.53	0.26	2.03	0.01	1.05	0.05
Sum of Skin Folds[11]	**R^2 = 0.60**	**p = <0.0001**				
Weekly Exercise Frequency	58.06	19.75	2.94	18.42	97.70	0.005
16wk Sum of Skin Folds	0.39	0.13	2.92	0.12	0.66	0.005
Parity	-15.50	6.36	-2.44	-28.27	-2.74	0.02
PrePregnancy Body Mass Index	2.04	0.86	2.36	0.30	3.77	0.02
Exercise Group [Aerobic]	-16.18	6.64	-2.44	-29.49	-2.86	0.02
Attendance	-94.31	46.62	-2.02	-187.86	-0.77	0.05
Waist Circumference[12]	**R^2 = 0.39**	**p = 0.0005**				
PrePregnancy Body Mass Index	0.39	0.08	4.70	0.23	0.56	<0.0001
Weekly Exercise Duration	-0.09	0.04	-2.23	-0.17	-0.009	0.03
Weekly Exercise Frequency	3.92	1.98	1.98	-0.04	7.88	0.05

Model 1: 16wk Weight, Total Exercise Volume, Exercise Group
Model 2: 16wk Weight, Weekly Exercise Duration, Weekly Exercise Frequency
Model 3: Parity, 16wk Fat Free Mass Percent, PrePregnancy Body Mass Index, Weekly Exercise Duration, Weekly Exercise Frequency, Attendance, Maternal Race, Exercise Group, Age
Model 4: 16wk Body Fat Percent, Parity, PrePregnancy Body Mass Index, Weekly Exercise Frequency, Weekly Exercise Duration, Attendance, Exercise Group, Maternal Race
Model 5: 16wk Triceps Skin Fold, Weekly Exercise Duration, Weekly Exercise Frequency, Parity, PrePregnancy Body Mass Index, Exercise Group
Model 6: 16wk Triceps Skin Fold, Parity, Total Exercise Volume, Exercise Group, PrePregnancy Body Mass Index, Gravida
Model 7: 16wk Subscapular Skin Fold, Weekly Exercise Frequency, Total Exercise Trainings, PrePregnancy Body Mass Index, Age
Model 8: 16wk Prepregnancy Body Mass Index, Parity, Suprailiac Skin Fold, Attendance, Weekly Exercise Frequency, Exercise Group, Maternal Race, Weekly Exercise Duration
Model 9: 16wk Thigh Skin Fold, Total Exercise Volume, Exercise Group, PrePregnancy Body Mass Index, Maternal Race
Model 10: 16wk Thigh Skin Fold, Weekly Exercise Frequency, Total Exercise Trainings, PrePregnancy Body Mass Index, Weekly Exercise Duration

Model 11: Weekly Exercise Frequency, 16wk Sum of Skin Folds, Parity, PrePregnancy Body Mass Index, Attendance, Exercise Group, Weekly Exercise Duration, Total Exercise Trainings, Age, Race, Gravida
Model 12: PrePregnancy Body Mass Index, Weekly Exercise Duration, Weekly Exercise Frequency, Age, Average Weekly METs, 16wk Waist Circumference, Maternal Race, Parity
*p<0.05, **p<0.01, ***p<0.001

Regressions for Maternal Morphometric Change Score Outcomes. Controlling for exercise group, gravida, maternal age, weekly exercise frequency, GWG is predicted (R^2=0.34, p=.0004) by prepregnancyBMI, 16wk body weight, and weekly exercise duration. GWG is predicted (R^2=0.26, p=<.0001) by prepregnancyBMI, 16wk body weight, maternal age, and exercise attendance. 16-36wk BMI is predicted (R^2=0.14, p=.0005), by 16wk BMI, maternal age, and exercise attendance. Controlling for prepregnancyBMI and maternal age, 16-36wk skinfold sum is predicted (R^2=0.53, p=<.0001) by 16wk sum of skin folds, weekly exercise frequency, parity, exercise group [aerobic], maternal race [Asian], attendance, total exercise trainings, weekly exercise duration. Controlling for weekly exercise frequency, exercise group, and prepregnancyBMI, 16-36wk WTH ratio is predicted (R^2=0.34, p=<.0001) by 16wk WTH ratio and weekly exercise frequency. Controlling for prepregnancyBMI and maternal age, 16-36wk body fat percent is predicted (R^2=0.54, p=<.0001) by 16wk body fat percent, parity, weekly exercise frequency, exercise group [aerobic], weekly exercise duration, maternal race [Asian], and exercise attendance. Controlling for exercise attendance, 16-36wk fat free mass percent is predicted (R^2=0.51, p=<.0001) by 16wk fat free mass percent, weekly exercise frequency, parity, total trainings, maternal race [Asian], and prepregnancyBMI (Table 2.8).

Table 2.8. 16-36wk Maternal Body Composition Regression models

	Estimate	STD Error	t Ratio	Lower 95%	Upper 95%	p-value
Gestational Weight Gain[1] R^2 = 0.34 p = 0.0004						
PrePregnancy Body Mass Index	-2.03	0.49	-4.20	-3.01	-1.07	<0.0001
16wk Weight	0.29	0.08	3.76	0.14	0.45	0.0004
Weekly Exercise Duration	-0.32	0.13	-2.46	-0.58	-0.06	0.02
Gestational Weight Gain[2] R^2 = 0.26 p = <0.0001						
PrePregnancy Body Mass Index	-2.26	0.44	-5.17	-3.13	-1.40	<0.0001
16wk Weight	0.33	0.07	4.60	0.19	0.47	<0.0001
Age	-0.48	0.23	-2.10	-0.93	-0.03	0.04
Attendance	-23.23	11.08	-2.10	-45.19	-1.27	0.04
Body Mass Index[3] R^2 = 0.14 p = 0.0005						
16wk Body Mass Index	-0.11	0.03	-3.53	-0.18	-0.05	0.0006
Age	-0.08	0.04	-2.34	-0.15	-0.01	0.02
Attendance	-4.18	1.79	-2.33	-7.72	-0.63	0.02
Sum of Skin Folds[4] R^2 = 0.53 p = <0.0001						
16wk Sum of Skin Folds	-0.51	0.12	-4.18	-0.76	0.27	0.0001
Weekly Exercise Frequency	66.29	17.91	3.70	30.35	102.22	0.0005
Parity	-12.92	4.49	-2.88	-21.93	-3.92	0.006
Exercise Group [Aerobic]	-16.62	5.91	-2.81	-28.47	-4.76	0.007
Maternal Race [Asian]	15.72	6.86	2.29	1.95	29.49	0.03
Attendance	-93.06	41.39	-2.25	-176.12	-9.99	0.03
Total Exercise Trainings	-0.67	0.33	-2.01	-1.33	-0.001	0.05
Weekly Exercise Duration	-0.74	0.37	-2.01	-1.48	-0.0004	0.05
Waist to Hip ratio[5] R^2 = 0.34 p = <0.0001						
16wk Waist to Hip ratio	-0.28	0.08	-3.55	-0.44	-0.12	0.0006
Weekly Exercise Frequency	-0.03	0.01	-2.17	-0.05	-0.002	0.03
Body Fat Percent[6] R^2 = 0.54 p = <0.0001						
16wk Body Fat Percent	-0.53	0.11	-4.90	-0.74	-0.31	<0.0001
Parity	-2.26	0.62	-3.63	-3.51	-1.01	0.0006
Weekly Exercise Frequency	7.96	2.56	3.11	2.83	13.08	0.003
Exercise Group [Aerobic]	-2.30	0.83	-2.77	-3.96	-0.64	0.008
Weekly Exercise Duration	-0.12	0.05	-2.62	-0.22	-0.03	0.01

Maternal Race [Asian]	2.36	0.99	2.37	0.37	4.35	0.02
Attendance	-11.93	5.65	-2.11	-23.26	-0.61	0.04
Fat Free Mass Percent[7]	**R^2 = 0.51**	**p = <0.0001**				
16wk Fat Free Mass Percent	-0.58	0.10	-5.90	-0.78	-0.39	<0.0001
Weekly Exercise Frequency	-3.66	1.25	-2.92	-6.16	-1.15	0.005
Parity	1.31	0.50	2.62	0.31	2.31	0.01
Total Trainings	0.10	0.04	2.42	0.02	0.18	0.02
Maternal Race [Asian]	-1.71	0.82	-2.08	-3.36	-0.06	0.04
PrePregnancy Body Mass Index	-0.21	0.10	-2.05	-0.41	-0.005	0.04

Model 1: 16wk Weight, PrePregnancy Body Mass Index, Weekly Exercise Duration, Exercise Group, Gravida, Age, Weekly Exercise Frequency
Model 2: 16wk Weight, PrePregnancy Body Mass Index, Age, Attendance
Model 3: 16wk Body Mass Index, Age, Attendance
Model 4: 16wk Sum of Skin Folds, Weekly Exercise Frequency, Parity, Maternal Race, Attendance, Exercise Group, Total Exercise Trainings, Weekly Exercise Duration, PrePregnancy Body Mass Index, Age
Model 5: 16wk Waist to Hip ratio, Maternal Race, Weekly Exercise Frequency, Exercise Group, PrePregnancy Body Mass Index
Model 6: 16wk Body Fat Percent, Parity, Weekly Exercise Frequency, Weekly Exercise Duration, Maternal Race, Exercise Group, Attendance, PrePregnancy Body Mass Index, Age
Model 7: 16wk Fat Free Mass Percent, Weekly Exercise Frequency, Parity, Total Exercise Trainings, PrePregnancy Body Mass Index, Maternal Race, Attendance
*p<0.05, **p<0.01, ***p<0.001

Regressions for Maternal Biomarker Outcomes. Total cholesterol is predicted (R^2=0.47, p=<.0001) by 16wk total cholesterol, parity, and average weekly METs. Controlling for exercise attendance, 36wk HDL cholesterol is predicted (R^2=0.53, p=<.0001) by 16wk HDL cholesterol, maternal age, and exercise group [control]. 36wk LDL cholesterol is predicted (R^2=0.48, p=<.0001) by 16wk LDL cholesterol, parity, and average weekly METs. Controlling for exercise group, 36wk triglycerides is predicted (R^2=0.40, p=<.0001) by 16wk triglycerides and average weekly METs. NonHDL cholesterol is predicted (R^2=0.49, p=<.0001) by 16wk NonHDL cholesterol, parity, and average weekly METs. Controlling for maternal race, maternal age, and average weekly METs, 36wk lactate is predicted (R^2=0.27, p=.02) by parity, weekly exercise frequency, and 16wk lactate. Controlling for exercise attendance and maternal race, 36wk lactate is predicted (R^2=0.23, p=.04) by 16wk lactate, parity, and exercise group [control]. 36wk insulin is

predicted (R^2=0.90, p=<.0001) by 16wk insulin, total exercise trainings, and exercise attendance. Controlling for prepregnancyBMI, 36wk glucose is predicted (R^2=0.34, p=.0003) by 16wk glucose, total exercise volume, exercise attendance, and exercise group [control]. Controlling for gravida, 36wk HOMA-IR is predicted (R^2=0.91, p=<.0001) by 16wk HOMA-IR, maternal age, prepregnancyBMI, and weekly exercise frequency (Table 2.9).

Table 2.9. 36wk Maternal Blood Biomarker Regression models

	Estimate	STD Error	t Ratio	Lower 95%	Upper 95%	p-value
Total Cholesterol[1]	**R^2 = 0.47**	**p = <0.0001**				
16wk Total Cholesterol	0.98	0.14	6.84	0.70	1.27	<0.0001
Parity	16.46	5.90	2.79	4.69	28.24	0.007
Average Weekly METs	15.46	6.83	2.26	1.82	29.10	0.03
HDL Cholesterol[2]	**R^2 = 0.53**	**p = <0.0001**				
16wk HDL Cholesterol	0.71	0.10	7.30	0.51	0.90	<0.0001
Age	0.77	0.31	2.46	0.14	1.39	0.02
Exercise Group [Control]	-6.17	2.78	-2.22	-11.73	-0.62	0.03
LDL Cholesterol[3]	**R^2 = 0.48**	**p = <0.0001**				
16wk LDL Cholesterol	1.12	0.16	7.05	0.80	1.44	<0.0001
Parity	18.34	5.99	3.06	6.38	30.30	0.003
Average Weekly METs	15.55	7.41	2.10	0.75	30.26	0.04
Triglycerides[4]	**R^2 = 0.40**	**p = <0.0001**				
16wk Triglycerides	0.83	0.16	5.20	0.51	1.14	<0.0001
Average Weekly METs	25.53	10.34	2.47	4.88	46.19	0.02
NonHDL Cholesterol[5]	**R^2 = 0.49**	**p = <0.0001**				
16wk NonHDL Cholesterol	1.08	0.15	7.38	0.79	1.38	<0.0001
Parity	17.05	6.30	2.71	4.48	29.63	0.009
Average Weekly METs	14.76	7.30	2.02	0.19	29.33	0.05
Lactate[6]	**R^2 = 0.27**	**p = 0.02**				

Parity	-0.25	0.09	-2.83	-0.43	-0.07	0.007
Weekly Exercise Frequency	-0.42	0.15	-2.75	-0.73	-0.11	0.009
16wk Lactate	0.22	0.09	2.35	0.03	0.41	0.02
Lactate[7]	**R^2 = 0.23**	**p = 0.04**				
16wk Lactate	0.18	0.08	2.32	0.02	0.33	0.02
Parity	-0.17	0.07	-2.30	-0.32	-0.02	0.03
Exercise Group [Control]	0.37	0.13	2.83	0.11	0.62	0.006
Insulin[8]	**R^2 = 0.90**	**p = <0.0001**				
16wk Insulin	1.27	0.11	11.71	1.04	1.50	<0.0001
Total Exercise Trainings	-0.04	0.01	-3.67	-0.06	-0.02	0.001
Attendance	5.77	2.23	2.59	1.14	10.41	0.02
Glucose[9]	**R^2 = 0.34**	**p = 0.0003**				
16wk Glucose	0.43	0.13	3.33	0.17	0.68	0.001
Total Exercise Volume	0.0006	0.0003	2.51	0.0001	0.001	0.01
Attendance	-25.59	12.13	-2.11	-49.83	-1.36	0.04
Exercise Group [Control]	6.20	3.08	3.01	0.05	12.35	0.05
HOMA-IR[10]	**R^2 = 0.91**	**p = <0.0001**				
16wk HOMA-IR	1.47	0.14	10.72	1.18	1.77	<0.0001
Age	0.06	0.02	3.68	0.02	0.09	0.003
PrePregnancy Body Mass Index	0.03	0.01	2.80	0.007	0.05	0.02
Weekly Exercise Frequency	-0.22	0.09	-2.45	-0.42	-0.03	0.03

Model 1: 16wk Total Cholesterol, Parity, Average Weekly METs
Model 2: 16wk HDL Cholesterol, Age, Attendance, Exercise Group
Model 3: 16wk LDL Cholesterol, Parity, Average Weekly METs
Model 4: 16wk Triglycerides, Average Weekly METs, Exercise Group
Model 5: 16wk NonHDL Cholesterol, Parity, Average Weekly METs
Model 6: Parity, Weekly Exercise Frequency, 16wk Lactate, Race, Age, Average Weekly METs
Model 7: 16wk Lactate, Parity, Exercise Group, Attendance, Maternal Race
Model 8: 16wk Insulin, Total Exercise Trainings, Attendance
Model 9: 16wk Glucose, Total Exercise Volume, Attendance, PrePregnancy Body Mass Index, Exercise Group
Model 10: 16wk HOMA-IR, Age, PrePregnancy Body Mass Index, Weekly Exercise Frequency, Gravida
*p<0.05, **p<0.01, ***p<0.001

Regressions for Maternal Biomarker Change Score Outcomes. Controlling for parity, 16-36wk HDL is predicted (R^2=0.27, p=.0005) by exercise group [control] and [aerobic], maternal age, exercise attendance, and 16wk HDL cholesterol. Controlling for 16wk LDL cholesterol, 16-36wk LDL cholesterol is predicted (R^2=0.14, p=.03) by parity and average

weekly METs. Controlling for 16wk triglycerides and gravida, 16-36wk triglycerides is predicted (R^2=0.16, p=.05) by average weekly METs, total exercise trainings, and parity. Controlling for exercise group, 16-36wk lactate is predicted (R^2=0.41, p=<.0001) by 16wk lactate, parity, and weekly exercise frequency. 16-36wk lactate is predicted (R^2=0.42, p=<.0001) by 16wk lactate, exercise group [control] and aerobic, and parity. Controlling for prepregnancyBMI, 16-36wk insulin is predicted (R^2=0.60, p=.001) by exercise attendance, 16wk insulin and total exercise volume. Controlling for maternal race, 16-36wk insulin is predicted (R^2=0.75, p=<.0001) by exercise attendance, total exercise trainings, and 16wk insulin. Controlling for prepregnancyBMI, maternal age, and weekly exercise frequency, 16-36wk glucose is predicted (R^2=0.23, p=.008) by 16wk glucose and average weekly METs. Controlling for prepregnancyBMI and parity, 16-36wk glucose is predicted (R^2=0.21, p=.003) by 16wk glucose and total exercise volume. Controlling for exercise attendance, 16-36wk HOMA-IR is predicted (R^2=0.92, p=<.0001) by 16wk HOMA-IR, prepregnancyBMI, maternal age, and total trainings (Table 2.10).

Table 2.10. 16-36wk Maternal Blood Biomarker Regression models

		Estimate	STD Error	t Ratio	Lower 95%	Upper 95%	p-value
HDL Cholesterol[1]	**R^2 = 0.27**	**p = 0.005**					
Exercise Group [Control]		-5.87	2.59	-2.27	-11.04	-0.70	0.03
Age		0.66	0.30	2.22	0.06	1.26	0.03
Attendance		39.77	18.17	2.19	3.43	76.10	0.03
Exercise Group [Aerobic]		4.28	2.10	2.04	0.07	8.48	0.04
16wk HDL Cholesterol		-0.18	0.09	-2.02	-0.35	-0.002	0.04
LDL Cholesterol[2]	**R^2 = 0.14**	**p = 0.03**					
Parity		13.64	5.56	2.45	2.52	24.75	0.02

Average Weekly METs	14.10	6.62	2.13	0.86	27.33	0.04
Triglycerides[3]	**R^2 = 0.16**	**p = 0.05**				
Average Weekly METs	20.87	8.96	2.33	2.95	38.79	0.02
Total Exercise Trainings	-0.94	0.42	-2.21	-1.79	-0.09	0.03
Parity	-23.75	11.29	-2.10	-46.32	-1.18	0.04
Lactate[4]	**R^2 = 0.41**	**p = <0.0001**				
16wk Lactate	-0.65	0.14	-4.73	-0.92	-0.37	0.0001
Parity	-0.20	0.09	-2.27	-0.37	-0.02	0.03
Weekly Exercise Frequency	-0.36	0.17	-2.12	-0.70	-0.02	0.04
Lactate[5]	**R^2 = 0.42**	**p = <0.0001**				
16wk Lactate	-0.63	0.14	-4.66	-0.91	-0.36	<0.0001
Exercise Group [Control]	0.48	0.12	4.00	0.24	0.73	0.0002
Exercise Group [Aerobic]	-0.24	0.09	-2.67	-0.42	-0.06	0.01
Parity	-0.17	0.08	-2.03	-0.33	-0.002	0.05
Insulin[6]	**R^2 = 0.60**	**p = 0.001**				
Attendance	7.82	2.44	3.20	2.70	12.93	0.005
16wk Insulin	0.34	0.11	3.12	0.11	0.57	0.006
Total Exercise Volume	-0.00007	0.00003	-2.06	0.0001	0.000001	0.05
Insulin[7]	**R^2 = 0.75**	**p = <0.0001**				
Attendance	9.99	2.16	4.63	5.45	14.52	0.0002
Total Exercise Trainings	-0.04	0.01	-4.12	-0.06	-0.02	0.0006
16wk Insulin	0.37	0.09	3.95	0.17	0.56	0.0009
Glucose[8]	**R^2 = 0.23**	**p = 0.008**				
16wk Glucose	-0.51	0.14	-3.76	-0.79	-0.24	0.0004
Average Weekly METs	3.39	1.69	2.01	0.01	6.77	0.05
Glucose[9]	**R^2 = 0.21**	**p = 0.003**				
16wk Glucose	-0.53	0.14	-3.84	-0.80	-0.25	0.0003
Total Exercise Volume	0.0005	0.0002	1.98	-0.000005	0.001	0.05
HOMA-IR[10]	**R^2 = 0.92**	**p = <0.0001**				
16wk HOMA-IR	1.40	0.14	10.02	1.09	1.70	<0.0001
PrePregnancy Body Mass Index	0.04	0.01	2.95	0.01	0.06	0.01
Age	0.05	0.02	2.82	0.01	0.09	0.02
Total Trainings	-0.007	0.003	-2.21	-0.01	-0.00004	0.05

Model 1: 16wk HDL Cholesterol, Attendance, Age, Exercise Group, Parity
Model 2: 16wk LDL Cholesterol, Parity, Average Weekly METs
Model 3: 16wk Triglycerides, Average Weekly METs, Total Exercise Trainings, Parity, Gravida
Model 4: 16wk Lactate, Parity, Weekly Exercise Frequency, Exercise Group
Model 5: 16wk Lactate, Exercise Group, Parity, Attendance
Model 6: Attendance, 16wk Insulin, Total Exercise Volume, PrePregnancy Body Mass Index
Model 7: Attendance, Total Exercise Trainings, 16wk Insulin, Maternal Race
Model 8: 16wk Glucose, Average Weekly METs, PrePregnancy Body Mass Index, Weekly Exercise Frequency, Age
Model 9: 16wk Glucose, Total Exercise Volume, PrePregnancy Body Mass Index, Parity
Model 10: 16wk HOMA-IR, PrePregnancy Body Mass Index, Total Exercise Trainings, Age, Attendance
*p<0.05, **p<0.01, ***p<0.001

Discussion

The current study aimed to determine the influence of maternal exercise FITT-V on maternal body composition and blood biomarker outcomes. We hypothesized that maternal exercise FITT-V would improve maternal body composition and blood biomarker outcomes, such as decreasing gestational weight gain, body fat percent, waist-to-hip ratio, cholesterol levels, lactate, insulin, and HOMA-IR, while increasing lean mass percent, and HDL-C. We found a plethora of significant associations between exercise FITT-V and maternal body composition and blood biomarkers. Relationships were found for many specific FITT-V metrics. In brief, weekly exercise volume is related to decreased 36wk body weight; weekly exercise duration decreases maternal blood biomarker measures; weekly exercise frequency is associated with decreased lactate and waist-to-hip ratio; while exercise intensity increases maternal cholesterol levels. These effects were specific to exercise type, with AE showing more associations than RE or AERE, especially in lactate, insulin, and HOMA-IR.

Maternal Exercise Volume

Increased weekly exercise volume improves regional body composition and metabolic biomarkers. Our study observed that greater exercise volume decreases fasting insulin levels, aligning with findings from studies in non-pregnant adults showing that higher volumes of moderate-intensity exercise improve insulin regulation[99]. This suggests that an increased exercise volume can positively impact insulin sensitivity in pregnant populations. However, our findings diverged from studies on non-pregnant adults regarding fasting glucose levels[99-101], as we observed an increase in fasting glucose at the 36-week gestation relative to total exercise volume. This difference may reflect unique metabolic adaptations during pregnancy that influence glucose regulation, likely due to the physiological demands of pregnancy on glucose availability and insulin sensitivity.

Total exercise volume predicts decreased 36wk body weight as well as triceps and thigh skinfold thicknesses in late pregnancy, which highlights the impact of maternal exercise on improving body composition. This aligns with other studies that show moderate to high exercise volume—ideally 150–300 minutes per week of moderate-intensity activity—can limit excessive fat gain, support lean muscle retention, and help manage overall weight gain, all of which are beneficial for both maternal and fetal health[102-103]. Aerobic and resistance exercises in particular help maintain fat-free mass, which is essential for metabolic function, insulin sensitivity, and sustained energy levels[99-101,103]. Moderate exercise volume is sufficient to improve body composition without risks to maternal or fetal health. Women who engage in regular, moderate exercise during pregnancy gain less excess fat, and experience fewer metabolic disruptions, which can support faster postpartum recovery and healthier body composition outcomes[73,104-105].

Maternal Exercise Duration

Increased weekly exercise duration decreases nearly all maternal body composition measures. In our study increased weekly maternal exercise duration is strongly associated with improved body composition outcomes at the 36-week time point. Pregnant women engaging in longer weekly exercise durations have lower body fat percentage, triceps skinfold thickness, and waist circumference, along with increased fat-free mass percentage. Similar to findings in non-pregnant individuals, we also demonstrate exercise duration is negatively associated with GWG and body fat gain[105]. Moderate-duration workouts of 45–60 minutes of exercise can enhance fat loss, muscle growth, cardiovascular health, and endurance without excessive fatigue. For pregnant women, 50 min-duration workouts and longer seem to support body composition improvements, potentially offering a sustainable and balanced approach to maintaining health during pregnancy[73].

However, exercise duration did not correlate with fasting cholesterol, glucose, insulin, or HOMA-IR values at the 36-week mark, nor did it significantly impact changes in these biomarkers between the 16-week and 36-week assessments. This is contrary to what is seen in non-pregnant adult populations, extending the duration of exercise sessions improves metabolic and cardiovascular health, with reductions in LDL cholesterol, increases in HDL cholesterol[106-108], and improvements in fasting glucose[101], fasting insulin[109], and insulin sensitivity, as indicated by lower HOMA-IR values[110]. These

benefits highlight the positive impact of longer exercise durations on lipid profiles and glucose regulation. This suggests that while exercise duration may offer immediate metabolic benefits in non-gravid populations, sustained benefits during pregnancy may depend on additional factors, such as frequency or intensity adjustments in the exercise regimen.

Maternal Exercise Frequency

Increased weekly exercise frequency improves metabolic efficiency and insulin sensitivity. In our study, higher weekly exercise frequency within structured programs decreases HOMA-IR values at 36 weeks and reductions in blood lactate levels throughout the intervention period. Similarly, in non-pregnant adult populations, increasing the frequency of weekly exercise sessions has been shown to significantly improve metabolic and body composition outcomes. Regular exercise is associated with reductions in fasting cholesterol[107], glucose levels[101], as well as improved insulin sensitivity[110], reflected in decreased insulin levels and lower HOMA-IR scores[110]. This underscores the importance of consistent exercise frequency in managing metabolic health and mitigating risks related to insulin resistance and associated disorders during pregnancy.

Our study found that increased exercise frequency also plays a pivotal role in shaping body composition, by reducing WHR. In non-gravid populations, increased exercise frequency influences maximize calorie expenditure, reduces body fat, and promotes lean muscle growth. This is particularly beneficial for lowering visceral fat, which supports

cardiovascular health[111-112]. Higher-frequency resistance training, performed at least four days per week, enables targeted muscle group training, promoting muscle growth and strength while avoiding excessive strain. Frequent exercise stimulates consistent muscle protein synthesis, essential for maintaining and growing lean muscle mass[113-115].

For long-term sustainability and effectiveness, exercise frequency should be balanced with intensity and recovery[114]. A moderate frequency of three to four days per week provides an effective balance, driving body composition improvements while allowing adequate recovery time for muscle repair and fatigue prevention[113-114]. This approach supports both aerobic and resistance training goals, optimizing fat loss and muscle gain while allowing flexibility in workout focus, such as alternating between upper body, lower body, cardio, and full-body exercises. Overall, consistent and appropriately frequent exercise is essential for achieving and maintaining metabolic and body composition improvements[83-84].

Maternal Exercise Intensity

Increased weekly exercise intensity increases cholesterol and glucose levels. Our study found that weekly exercise intensity, using metabolic equivalents (METs), a measure of intensity, did not predict maternal body composition outcomes at the 36-week mark or changes across pregnancy. Higher METs were associated with increases in triglycerides, LDL cholesterol, and total cholesterol, contrasting with findings in non-pregnant populations. In non-pregnant adults, research highlights the significant impact of exercise

intensity on metabolic health and body composition. High-intensity exercise, such as high-intensity interval training (HIIT) and vigorous aerobic or resistance training, provides substantial benefits for metabolic markers, including fasting cholesterol, glucose, insulin, and HOMA-IR. High-intensity aerobic exercise effectively improves lipid profiles by reducing LDL cholesterol and increasing HDL cholesterol, enhancing cardiovascular health and lowering atherosclerosis risk[107-108]. For glucose regulation, HIIT and vigorous exercise significantly improve fasting blood glucose, particularly in individuals with impaired glucose tolerance or type 2 diabetes, while also reducing fasting insulin levels and enhancing insulin sensitivity as measured by HOMA-IR[109-110]. These benefits are often more pronounced compared to moderate- or low-intensity exercise.

High-intensity exercise also supports body composition improvements in non-gravid populations. Short, intense bursts of activity increase caloric burn both during and after workouts through excess post-exercise oxygen consumption (EPOC), contributing to significant fat loss over time[112]. Additionally, high-intensity resistance training promotes muscle hypertrophy by stimulating muscle repair and growth, which helps preserve lean muscle mass during fat loss. HIIT, in particular, is effective at reducing body fat while maintaining strength and lean mass, making it a valuable strategy for improving overall fitness[116].

Moderate-intensity exercise, such as brisk walking, jogging, or moderate circuit training, supports gradual fat loss while improving cardiovascular health and endurance. Typically

performed at 50–70% of maximum heart rate, moderate-intensity continuous training (MICT) utilizes fat as a primary fuel source, making it ideal for steady and sustainable improvements in body composition[103,112]. Circuit training at moderate intensity can also enhance muscular endurance and help preserve lean mass, contributing to overall fitness[116].

Low-intensity exercise, such as walking or gentle cycling, is primarily fueled by fat and offers a low-impact approach to improving body composition over time. While caloric expenditure is lower than with higher-intensity exercise, low-intensity activities can be maintained for longer durations, supporting gradual fat loss. This type of exercise is especially beneficial for beginners or individuals with lower fitness levels, as it provides a sustainable and low-stress option for improving body composition[97]. Additionally, low-intensity activities help increase daily physical activity, aiding in muscle maintenance and weight management[117-118].

These discrepancies suggest unique physiological responses to exercise intensity during pregnancy, potentially influenced by the hormonal and metabolic changes that accompany gestation. This highlights the need for tailored exercise recommendations to balance intensity and metabolic benefits safely during pregnancy.

Maternal Exercise Type

The type of exercise significantly influences metabolic health markers and maternal body composition during pregnancy. In this study, participants engaged in AE, RE, or AERE programs, with varying impacts on metabolic and body composition outcomes. While most maternal blood biomarkers did not differ significantly between groups, AE showed notable decreases in lactate and insulin change scores compared to the control group. Resistance exercise led to reductions in insulin, HOMA-IR, and lactate at 36 weeks. Interestingly, the AE group demonstrated stronger correlations between exercise volume (FITT-V) and favorable birth outcomes, particularly in change scores for HDL cholesterol. However, the AERE group did not yield the expected metabolic differences, potentially due to the combined modalities diluting the dose of each exercise type.

Regarding maternal body composition, AE is associated with favorable changes, such as reductions in the delta sum of skinfolds, delta body fat percentage, BMI, and hip circumference at 36 weeks compared to controls. This aligns with research showing that moderate-intensity aerobic exercise helps control gestational weight gain, supports cardiovascular health, and enhances energy levels, contributing to a more balanced maternal body composition[104-105]. Resistance exercise showed reductions in waist-to-hip ratio at 36 weeks and maintained lean body mass, which is critical for metabolism, postnatal recovery, and supporting the physical demands of pregnancy and childbirth. Additionally, RE improved joint stability, and posture, and reduced pregnancy-related discomforts like back pain, further highlighting its value during pregnancy[93].

Aerobic exercise (AE), such as walking, swimming, and cycling, is particularly effective at improving lipid profiles, reducing LDL cholesterol and triglycerides, and increasing HDL cholesterol[75]. This type of exercise also lowers fasting glucose, and insulin levels and improves insulin sensitivity, as indicated by decreased HOMA-IR scores[101]. Resistance exercise (RE) provides additional benefits, including reductions in fasting insulin, HOMA-IR, and modest decreases in LDL and total cholesterol[75]. The combination of aerobic and resistance exercises (AERE) has been shown in non-pregnant populations to provide comprehensive metabolic benefits by enhancing insulin sensitivity, reducing fasting glucose and cholesterol levels, and improving lactate management[119-120].

Surprisingly, the AERE group did not demonstrate the anticipated advantages, which may reflect the challenges of balancing both exercise modalities in a single program. Despite this, research supports that combining exercise types can maintain a healthy weight, preserve muscle mass, and reduce the risks of gestational diabetes and hypertensive disorders. A balanced approach to prenatal fitness that incorporates varied exercise types supports both maternal and fetal health, as well as postpartum recovery[73].

Overall, these findings emphasize the significant benefits of exercise, particularly aerobic activity, in enhancing metabolic health and maternal body composition during pregnancy, while resistance exercise provides complementary advantages for maintaining strength and lean mass.

Strengths & Limitations

The current analysis is the first to demonstrate a direct effect of maternal exercise FITT-V on maternal body composition and blood biomarker outcomes throughout pregnancy. The study is strengthened by its large sample size, derived from rigorous supervised RCT exercise intervention trials. The diverse range of outcomes measured provides a comprehensive picture of the effects of maternal exercise FITT-V on maternal body composition and blood biomarker outcomes. However, the study has some limitations. First, maternal exercise FITT-V is controlled within a prescribed range, limiting our investigation of high and low FITT-V; nonetheless, this step is crucial for demonstrating the dose-dependent influence of exercise on beneficial pregnancy outcomes. Second, although some body composition measures were obtained as standard procedures, skinfold measurements may have interindividual differences between study staff. These are standard processes in the research team and, thus likely have a small margin of error. Overall, these initial findings highlight the need for further research in this area and support continued work on this topic.

Conclusion

The study revealed significant associations between maternal exercise FITT-V metrics—frequency, intensity, time, and type—and both maternal blood biomarkers and body composition. Increased exercise frequency is linked to improved metabolic efficiency, as indicated by lower lactate levels, and a reduction in waist-to-hip ratio, highlighting its role in enhancing metabolic and body composition outcomes. Total exercise volume and

duration were strong predictors of decreased body weight, improved body fat percentage, and overall better body composition. Aerobic exercise emerged as particularly beneficial, demonstrating significant reductions in fasting insulin levels, HOMA-IR scores, and hip circumference, underscoring its impact on enhancing insulin sensitivity, glucose metabolism, and body composition.

In contrast, higher exercise intensity is associated with elevated cholesterol levels, possibly reflecting adaptations in lipid metabolism due to vigorous activity. These findings emphasize the importance of individualized and structured exercise regimens during pregnancy, as specific combinations of frequency, intensity, and type of exercise can optimize maternal health outcomes. By promoting tailored exercise plans that include aerobic activities and consider appropriate intensity and volume, healthcare providers can support maternal health, reduce the risk of gestational diabetes, and improve postpartum recovery.

These results underscore the need for further research to refine optimal exercise strategies for pregnant women, ensuring safety while maximizing health benefits for both mothers and their developing children. Structured exercise during pregnancy can significantly enhance metabolic and body composition outcomes, contributing to healthier pregnancies and long-term maternal and child health.

Chapter 3:

Effects of Maternal Exercise Type on 1-month-old Infant Body Composition and Blood Biomarkers

ABSTRACT:

Purpose: To assess the effects of different types of prenatal exercise on infant 1-month body composition measures and lipid profile. We hypothesized that all types of prenatal exercise would improve infant body composition and blood lipids relative to infants of non-exercisers.

Study Design: Participants were randomly assigned to 150 minutes/week of either aerobic, muscular strength, or combined (aerobic+strength) moderate-intensity exercise, or a non-exercising stretching/breathing (control) group for ~24 weeks. At 4 weeks post-delivery, 1 month skinfold, length, and circumference measures and a blood draw were performed to assess infant body composition and lipid profile. Data were analyzed using analysis-of-variance and regression.

Results: Data from 180 participants was analyzed. Maternal descriptors were similar between groups, except prepregnancy BMI (p=.004) and GWG (p=.04). Groups were similar in gestational age at birth. There were group differences in one-month measures of HDL (p=.05) and nonHDL (p=.04). Infant outcomes were described as AEROBIC>COMBINATION>RESISTANCE>Control.

Conclusions: These findings demonstrate that prenatal exercise may improve infant body composition and blood lipids measures. Further research is needed to determine if these differences persist past 1 month of age.

Introduction

Childhood obesity is a critical public health concern, closely linked to detrimental metabolic conditions such as dyslipidemia and impaired glucose regulation. Obese children often exhibit elevated low-density lipoprotein (LDL) cholesterol, reduced high-density lipoprotein (HDL) cholesterol, and insulin resistance, which significantly increase their risk of cardiovascular disease, obesity, and type 2 diabetes in adulthood[1-5,15]. These metabolic challenges often originate during crucial developmental periods, shaped by genetic and environmental factors[15].

Maternal health and behaviors during pregnancy play a pivotal role in determining the metabolic health of children. Among these, maternal exercise has emerged as a promising intervention to lower the risk of childhood obesity and associated metabolic disorders. Engaging in regular physical activity during pregnancy enhances maternal glucose regulation and decreases the likelihood of gestational diabetes, a condition strongly correlated with adverse metabolic outcomes in offspring[79,82-85,88]. The benefits extend to children, with evidence showing that maternal exercise supports healthier lipid profiles, improved insulin sensitivity, and better cardiovascular health markers in offspring[79,89].

Maternal exercise influences fetal epigenetics related to lipid and glucose metabolism and body composition, which are linked to metabolic programming. Studies highlight how exercise-induced improvements in maternal metabolism, such as enhanced insulin signaling and lipid regulation, translate into healthier metabolic trajectories for offspring[79].

These changes contribute to long-term resistance against increased adiposity and high blood lipids, which are risks of cardiometabolic diseases.

Furthermore, emerging research underscores the role of maternal exercise in reducing systemic inflammation and promoting optimal fetal growth. Such effects are vital for preventing early-life metabolic disorders like dyslipidemia and hyperglycemia, which often serve as precursors to lifelong health challenges[80].

The purpose of this research paper is to examine the effects of maternal exercise during pregnancy on infant body composition and blood biomarkers associated with obesity risk. Given the rising prevalence of childhood obesity and its long-term health consequences, identifying early-life interventions is critical. This paper will explore how maternal physical activity influences infant adiposity and metabolic health, with a focus on key biomarkers such as insulin sensitivity and lipid profiles. By synthesizing current evidence and identifying research gaps, this study aims to provide insights into the potential role of maternal exercise as a preventive strategy against early-onset obesity, ultimately contributing to improved lifelong health outcomes for offspring. We hypothesize that prenatal exercise, any type, will decrease infant body composition and blood biomarkers, with aerobic exercise having the greatest change, relative to non-exercise-exposed infants.

Methods

Participants and Recruitment

This is a post-hoc secondary analysis of a randomized, blinded, prospective study focused on determining the influence of maternal exercise types on maternal and offspring health outcomes. All protocols were approved by the East Carolina University Institutional Review Board. Recruitment was focused on low-risk, healthy women with a singleton pregnancy. Women enrolled in the study met the following criteria: clearance from an obstetric provider to participate in exercise; between ages 18 and 40; pre-pregnancy BMI of 18.5–39.9; gestational age ≤ 16 weeks; no current alcohol, tobacco, or medication use. Criteria for exclusion were pre-existing diabetes mellitus, hypertension, cardiovascular disease, and medications known to affect fetal growth and well-being. Written informed consent was obtained from each participant; each participant subsequently completed a treadmill test and 1-repetition maximum strength tests. Using concealed randomization methods (GraphPad Prism Software), participants were randomly assigned to aerobic, resistance, combination (aerobic + resistance) exercise, or stretching-breathing attention control. Due to the nature of an exercise intervention, the participants and trainers knew the group assignment.

Pre-Intervention Exercise Testing

Participants recruited before March 2020, the COVID-19 pandemic, completed a submaximal modified Balke treadmill test following previously described protocols[92] to determine the target heart rate (HR) zone for exercise training. To minimize exposure and potential risk associated with COVID-19, women recruited following March 2020 did not complete the treadmill protocol, and target HR zones for exercise were determined based

on their pre-pregnancy physical activity level and age, using published guidelines for pregnant women[92]. Target HR zones for exercise corresponded to maternal HR at 60 to 80% of maximal oxygen consumption, reflecting moderate intensity[92].

Exercise Intervention

Participants trained according to American College of Obstetricians and Gynecologists guidelines for approximately 24 weeks, beginning at 16 weeks gestation and continuing until delivery (~40 weeks gestation)[93]. Each exercise training session included a 5-minute warmup, a 50-minute exercise period, and a 5-minute cooldown. Every training session, regardless of training type, was supervised; participants exercised within their target HR zone corresponding to moderate intensity (60%-80% maximal oxygen consumption and 12-14 rated perceived exertion) throughout the exercise session. HR was monitored (Polar FS2C HR Monitor) throughout each exercise session to ensure that participants remained within their target HR ranges for the duration of the session. All sessions were performed three times per week until delivery. The aerobic exercise (AE) group completed moderate-intensity training for 50 minutes on the treadmill, elliptical, recumbent bicycle, rowing, and/or stair-stepping equipment. To maintain the appropriate HR zone, speed, grade, and/or resistance were adjusted. The resistance exercise (RE) group completed sessions consisting of two to three sets of 15 repetitions of each exercise at moderate intensity[94]. Seated Cybex machines (leg extension, leg curl, shoulder press, chest press, triceps extension, latissimus dorsi pull-down), dumbbells (biceps curls, lateral shoulder raises, front shoulder raises), resistance bands, dumbbells,

exercise balls, benches, and/or mats were used. The combination exercise (AERE) group performed five 4.5-5 minutes long alternating bouts of aerobic and resistance exercise, each for 50 minutes per workout. The AERE group performed aerobic and resistance exercises on the same equipment as the AE and RE groups. The control group (CON) performed supervised stretching, breathing, and flexibility exercises for the duration of the session. The breathing exercises and stretches of CON participants targeted major muscle groups[94] and maintained a low intensity (<40% VO_{2peak}) throughout the session. Maternal exercise intensity in METs was determined based on the published compendium of physical activity for the exercise performed in each session[95]. The ability of this protocol to elicit maternal physiological adaptations to exercise has been previously reported[96].

Maternal Measurements

Maternal age, gravida, parity, pre-pregnancy weight and height, gestational weight gain (GWG), length of gestation, infant sex, birth length, and birth weight were abstracted from various sources, including pre-screening eligibility, postpartum questionnaires, as well as maternal and neonatal electronic health records. Gestational weight gain was calculated by subtracting maternal pre-pregnancy weight from weight at delivery and was extracted from electronic health records, while weight before pregnancy (lbs.) was collected via the pre-screening eligibility questionnaires. At 16 and 36 weeks of gestation, maternal height and weight were obtained using a stadiometer and calibrated medical grade scale; BMI was calculated as [weight (kg)] / [height (m) ^{2}] (healthy BMI 18.5-24.9; overweight 25-29.9, obese ≥30).

Birth Outcomes

At birth, delivery type (spontaneous vaginal delivery, C-section), neonate sex, gestational age, and neonatal measures (heart rate, chest, head, and abdominal circumferences, birth weight, length) were measured by labor & delivery nurses, using standard protocols, and entered into the electronic health record. Head circumference-abdominal circumference (HC: AC) and weight-length ratios were calculated. Apgar scores were measured at 1 and 5 minutes after birth by the labor and delivery nurse per standard hospital protocol.

One-month Infant Measurements

Infant percent body fat was determined using a previously validated skinfold technique and age-adjusted equations[121-123]; sites include triceps, subscapular, and biceps. Circumferences were measured at the head and abdomen. Blood biomarkers, glucose, lactate, and lipid profiles were assessed where venipuncture was completed. Two drops of blood from the sample were used to measure glucose, lipids (NonHDL, TC, TG, HDL, LDL), and lactate with Cholestech (Abbott, Orlando FL) and lactate analyzer (Nova Biomedical, Waltham MA), respectively.

Statistical Analysis

ANOVA was used to determine statistical significance between groups where appropriate. Kruskal-Wallis (4-group comparison) and Mann-Whitney (2-group comparison; exercise vs. control) tests were used to assess the difference between non-normally distributed variables (gravida and parity). Based on preliminary infant blood biomarker data, power analysis indicated a sample of 9 per group was needed to detect group differences in 1-month NonHDL cholesterol. Statistical significance was set at $p \leq 0.05$. Statistical analyses were performed using IBM SPSS Statistics (IBM Corp., Armonk, NY) and JMP Statistical Discovery (Cary, NC) for Mac.

Results

Maternal and infant birth descriptors. We were contacted by 454 pregnant women interested in the study (Figure 2). Our final analysis included 150 participants (CON=28, AE=47, RE=32, AERE=43). The intervention is conducted from September 1, 2018, to January 30, 2023.

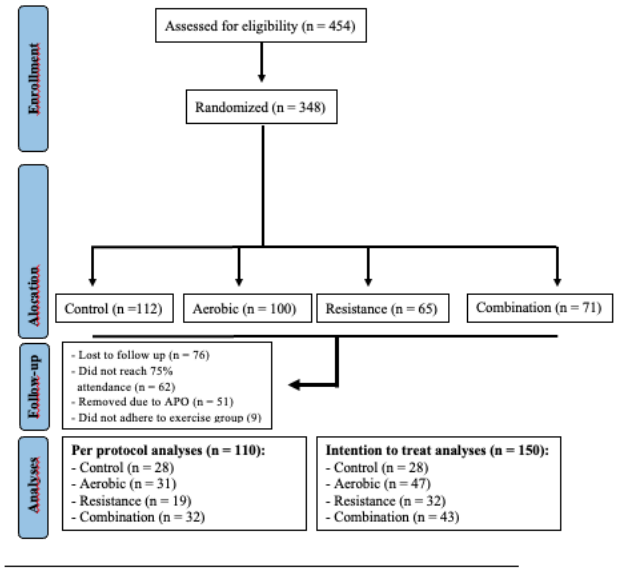

Fig. S2 Flow chart of the study participants.

For the intention-to-treat (ITT) analysis, participants were similar in age, gravida, parity, race, and gestational length. Pre-pregnancy BMI is higher in the Control group (Table 3.1).

For the per-protocol analysis, a total of 110 women met ≥75% adherence to exercise training throughout the ~24 weeks (AE=31, RE=19, AERE=32, CON=28). There were no differences between groups for age, gravida, parity, race, or length of gestation.

Pre-pregnancy BMI is higher in the Control group, and GWG was higher in the Resistance group (Table 3.2).

Table 3.1: Intention to Treat Maternal Descriptors

	Full Sample	Control	Aerobic	Resistance	Combination	p-value
Maternal Measures	(N=150)	(N=28)	(N=47)	(N=32)	(N=43)	
Age (years)	30.28 ± 4.18	30.04 ± 3.83	30.33 ± 4.77	30.47 ± 3.71	30.20 ± 4.19	.82
Prepregnancy BMI	25.17 ± 4.50	27.66 ± 5.35	23.92 ± 4.21**	24.95 ± 4.71	25.25 ± 3.58	**.01**
Gravida†	2.0 (1, 7)	2.0 (1, 4)	2.0 (1, 4)	2.0 (1, 5)	2.0 (1, 7)	.59
Parity†	1.0 (0, 4)	1.0 (0, 3)	1.0 (0, 2)	1.0 (0, 4)	1.0 (0, 3)	.60
Attendance	85.48 ± 17.84	--	83.40 ± 16.41	78.19 ± 19.57	85.20 ± 17.53	.19
Race						.14
White	125 (83.3%)	19 (67.9%)	37 (78.7%)	29 (90.6%)	40 (93%)	
Black	15 (10%)	6 (21.4%)	5 (10.6%)	2 (6.3%)	2 (4.7%)	
Other	10 (6.7%)	3 (10.7%)	5 (10.6%)	1 (3.1%)	1 (2.3%)	
GWG	33.11 ± 10.66	29.59 ± 10.71	30.30 ± 9.89	36.44 ± 11.27*	35.48 ± 9.87	**.01**
Length of Gestation (wks)	39.72 ± 1.03	39.42 ± 1.18	39.74 ± 1.11	39.94 ± 0.90	39.69 ± 0.95	.28
Infant Measures						
Infant Sex (Male)	79 (52.7%)	18 (64.3%)	26 (55.3%)	18 (56.2%)	17 (39.5%)	.19
Birth Length (m)	0.50 ± 0.02	0.50 ± 0.03	0.49 ± 0.03	0.50 ± 0.02	0.50 ± 0.02	.45
Birth Weight (kg)	3.55 ± 0.44	3.53 ± 0.59	3.54 ± 0.39	3.60 ± 0.48	3.52 ± 0.37	.87

Presented as mean +/- SD. † due to non-normal distribution, Mann Whitney U test performed and expressed as median (minimum, maximum). GWG - Gestational Weight Gain. *p < 0.05, **p < 0.01, p*** < 0.001 compared to control

Table 3.2: Per Protocol - Maternal Descriptors

	Full Sample	Control	Aerobic	Resistance	Combination	p-value
	(N=110)	(n=28)	(N=31)	(N=19)	(N=32)	
Age (years)	30.59 ± 3.88	30.04 ± 3.83	30.66 ± 4.29	31.37 ± 3.43	30.45 ± 3.87	.47
Prepregnancy BMI	25.12 ± 4.66	27.66 ± 5.35	23.70 ± 4.36 **	23.79 ± 4.17 *	25.41 ± 3.90	**.004**
Gravida†	2.00 (1, 4)	2.00 (1, 4)	2.00 (1, 4)	2.00 (1,4)	2.00 (1,3)	.54

Parity†	1.00 (0, 3)	1.00 (0,3)	1.00 (0, 2)	1.00 (0, 3)	1.00 (0, 1)	.62
Attendance	0.94 ± 0.07	--	0.93 ± 0.06	0.90 ± 0.05	0.93 ± 0.07	.26
Race						.18
White	89 (80.9%)	19 (67.9%)	23 (74.2%)	16 (84.2%)	31 (96.9%)	
Black	11 (10%)	6 (21.4%)	4 (12.9%)	2 (10.5%)	0 (0%)	
Other	9 (8.2%)	3 (10.7%)	4 (12.9%)	1 (5.3%)	1 (3.1%)	
GWG	32.86 ± 9.79	29.59 ± 10.71	31.88 ± 8.83	38.25 ± 8.56 *	32.92 ± 9.72	**.04**
Length of Gestation (wks)	39.72 ± 1.01	39.42 ± 1.18	39.37 ± 1.07	40.07 ± 0.81	39.66 ± 0.87	.18
Infant Measures						
Infant Sex (Male)	60 (54.5%)	17 (63%)	18 (58.1%)	12 (63.2%)	12 (37.5%)	.14
Birth Weight (kg)	3.51 ± 0.44	3.53 ± 0.59	3.48 ± 0.38	3.57 ± 0.44	3.51 ± 0.38	.71
Birth Length (m)	0.50 ± 0.02	0.50 ± 0.03	0.49 ± 0.03	0.50 ± 0.02	0.50 ± 0.02	.07
Presented as mean +/- SD. † due to non-normal distribution, Mann Whitney U test performed and expressed as median (minimum, maximum). GWG - Gestational Weight Gain. *p < 0.05, **p < 0.01, p*** < 0.001 compared to control						

1-month Infant Descriptors. For the per protocol, there were between-group differences in pre-pregnancy BMI (p=.004; CON to AE and RE) and GWG (p-value=.04; RE to CON) (Table 3.3).

Table 3.3: Intention to Treat 1-mo Infant Outcomes

	Control (N=28)	Aerobic (N=47)	Resistance (N=32)	Combination (N=43)	p-value
Length (cm)	54.24 ± 3.12	54.11 ± 3.38	54.79 ± 2.86	54.62 ± 3.16	.78
Weight (kg)	4.46 ± 0.70	4.46 ± 0.60	4.72 ± 0.71	4.63 ± 0.71	.30
Lean Mass (%)	10.77 ± 0.65	10.64 ± 0.46	10.48 ± 0.52	10.56 ± 0.49	.18
Triceps Skinfold (mm)	7.66 ± 1.87	7.39 ± 1.82	7.88 ± 1.95	7.90 ± 2.50	.63
Bicep Skinfold (mm)	5.97 ± 1.56	5.83 ± 1.86	5.91 ± 1.89	6.33 ± 2.03	.62
Subscapular Skinfold (mm)	6.56 ± 1.37	6.66 ± 1.88	6.70 ± 1.91	7.46 ± 2.04	.12
Skinfold Sum (mm)	20.19 ± 3.95	19.88 ± 4.55	20.48 ± 4.98	21.70 ± 5.94	.36
Abdominal Circumference (cm)	38.08 ± 2.64	38.91 ± 2.94	38.53 ± 2.76	39.27 ± 2.63	.32
Head Circumference (cm)	37.74 ± 2.50	38.00 ± 1.71	37.84 ± 2.84	37.87 ± 1.66	.96
Head/Abd Circumference	0.99 ± 0.08	0.98 ± 0.08	0.99 ± 0.09	0.98 ± 0.05	.79
Body Fat (%)	13.82 ± 2.92	13.64 ± 3.21	14.14 ± 3.47	14.87 ± 3.85	.37
BMI	15.15 ± 2.03	15.19 ± 1.84	15.72 ± 2.06	15.55 ± 2.11	.59
Total Cholesterol (mg/dL)	139.25 ± 35.46	134.25 ± 20.51	134.00 ± 19.94	123.69 ± 19.92	.45
HDL (mg/dL)	42.38 ± 13.13	57.37 ± 17.63*	44.62 ± 9.22	47.00 ± 15.31	**.05**
LDL (mg/dL)	82.43 ± 27.78	61.47 ± 13.60	63.45 ± 2.30	59.80 ± 15.16	.07
Triglycerides (mg/dL)	139.25 ± 35.46	134.25 ± 20.51	134.00 ± 19.94	123.69 ± 19.92	.08

NonHDL-C (mg/dL)	101.86 ± 26.23	78.60 ± 12.82 *	92.75 ± 18.24	82.00 ± 20.69	**.04**
Glucose (mg/dL)	77.38 ± 12.00	80.08 ± 6.76	74.85 ± 8.24	79.46 ± 9.23	.44
Lactate (mmol/L)	2.16 ± 1.54	1.73 ± 0.44	2.69 ± 1.81	2.05 ± 0.94	.32
Presented as mean +/- SD. SBP - Systolic Blood Pressure; HDL - High Density Lipoprotein; LDL - Low Density Lipoprotein; GDM - Gestational Diabetes Mellitus *p < 0.05, **p < 0.01, p*** < 0.001 relative to control group					

1-month Infant Outcomes. For the per-protocol analysis, we found significant differences for NonHDL-C and trending towards significance in LDL cholesterol (Table 4). Specifically, NonHDL-C is lower in infants from women who did AE compared to infants from CON women (Table 3.4).

Table 3.4: Per Protocol 1-mo Infant Outcomes

Infant Measures	Control	Aerobic	Resistance	Combination	p-value
	(N=28)	(N=31)	(N=19)	(N=32)	
Length (cm)	54.00 ± 3.65	54.06 ± 3.14	54.72 ± 2.93	54.51 ± 3.30	.80
Weight (kg)	4.45 ± 0.66	4.46 ± 0.59	4.51 ± 0.61	4.66 ± 0.72	.49
Fat-Free Mass %	85.91 ± 2.92	86.90 ± 3.42	86.51 ± 3.79	85.45 ± 4.10	.33
BMI	15.34 ± 2.49	15.19 ± 1.64	15.15 ± 1.74	15.71 ± 2.09	.65
Triceps Skinfold (mm)	7.79 ± 1.83	7.17 ± 1.90	7.69 ± 2.19	7.78 ± 2.66	.56
Bicep Skinfold (mm)	6.17 ± 1.91	5.87 ± 2.04	5.77 ± 1.55	6.21 ± 2.12	.76
Subscapular Skinfold (mm)	6.70 ± 1.39	6.35 ± 1.97	6.25 ± 1.96	7.27 ± 2.16	.12
Skin Fold Sum (mm)	20.66 ± 4.29	19.38 ± 5.03	19.71 ± 5.07	21.26 ± 6.30	.42
Abdominal Circumference (cm)	37.95 ± 2.56	38.69 ± 2.63	38.17 ± 2.70	39.16 ± 2.81	.26
Head Circumference (cm)	37.75 ± 2.37	37.93 ± 1.71	37.59 ± 1.75	37.39 ± 4.16	.86
Head/Abd Circumference	1.00 ± 0.08	0.98 ± 0.08	0.96 ± 0.21	0.96 ± 0.12	.51
Body Fat %	14.09 ± 2.92	13.10 ± 3.42	13.49 ± 3.79	14.55 ± 4.10	.33
Total Cholesterol (mg/dL)	139.25 ± 35.46	132. 23 ± 21.38	129.70 ± 44.83	126.33 ± 19.13	.84
HDL (mg/dL)	42.38 ± 13.13	56.92 ± 19.37	48.53 ± 13.00	49.00 ± 16.15	.24
LDL (mg/dL)	82.43 ± 27.77	59.50 ± 13.25	64.64 ± 20.14	59.33 ± 16.00	.07
Triglycerides (mg/dL)	102.00 ± 34.34	87.46 ± 30.36	138.67 ± 56.68	120.09 ± 79.45	.13
NonHDL-C (mg/dL)	101.86 ± 26.21	77.33 ± 11.29*	92.67 ± 17.90	79.70 ± 22.17	**.03**
Glucose	77.38 ± 12.00	79.00 ± 6.28	75.83 ± 6.53	79.75 ± 9.20	.69
Lactate	2.16 ± 1.54	1.80 ± 0.41	2.85 ± 1.83	2.05 ± 1.00	.30
Presented as mean +/- SD. HDL - High Density Lipoprotein; LDL - Low Density Lipoprotein *p < 0.05, **p < 0.01, p*** < 0.001 compared to control					

Discussion

The purpose of this research paper is to examine the effects of maternal exercise during pregnancy on infant body composition and blood biomarkers associated with obesity risk. We hypothesize that prenatal exercise, any type, will decrease infant body composition and blood biomarkers, with aerobic exercise having the greatest change, relative to non-exercise-exposed infants. We found significant differences between maternal exercise groups regarding infant lipid profiles.

In this study, participants engaged in AE, RE, or AERE programs, with varying impacts on metabolic biomarkers and body composition outcomes. We observed that aerobic exercise significantly reduced non-HDL cholesterol (nonHDL-C) in 1-month-old infants, while resistance exercise is trending towards significance in lowering LDL cholesterol levels at the same age compared to the control group. This suggests that different types of maternal exercise during pregnancy may have distinct effects on infant lipid profiles. Similarly, in other research, the type of exercise in non-gravid adults can significantly influence blood cholesterol levels, with aerobic and resistance exercises offering distinct benefits[107-108]. Resistance exercise complements these effects by improving insulin sensitivity and glucose control. Combination exercise has shown improvements in glucose metabolism in non-gravid adults[119].

In terms of body composition, surprisingly there were no significant differences between maternal exercise groups. In non-gravid adults' aerobic exercise has been associated with lower body fat percent and BMI[102]. This highlights the critical role of understanding how exercise type during pregnancy shapes infant metabolic health outcomes.

Strengths & Limitations

The current analysis is the first to demonstrate a direct effect of maternal exercise type on infant body composition and blood biomarker outcomes at one-month age. The study is strengthened by its large sample size, derived from rigorous supervised RCT exercise intervention trials. The diverse range of outcomes measured provides a comprehensive picture of the effects of maternal exercise type on infant body composition and blood biomarker outcomes. However, the study has some limitations. Some body composition measures were obtained as standard procedures; skinfold measurements may have interindividual differences between study staff. These are standard processes in the research team and, thus, likely have a small margin of error. Overall, these initial findings highlight the need for further research in this area and support continued work in understanding the differences in maternal exercise groups on infant body composition and blood lipids.

Conclusion

The study revealed significant associations between maternal exercise type and infant blood biomarkers. Aerobic-only exercisers showed significant decreases in NonHDL-C.

In contrast, none of the exercise groups provided significant improvements in body composition compared to non-exercisers. These results underscore the need for further research to refine optimal exercise strategies for pregnant women, ensuring safety while maximizing health benefits for both mothers and their developing children. Structured exercise during pregnancy can significantly enhance infant metabolic and body composition outcomes, contributing to long-term child health.

Chapter 4

Effects of Maternal Exercise Metrics on 1- and 6-month-old Infant Body Composition and Blood Biomarkers

ABSTRACT:

Purpose: To assess the effects of prenatal exercise metrics on infant 1- and 6-month body composition measures and lipid profile. We hypothesized that increased prenatal exercise metrics would improve infant body composition and blood lipids relative to infants exposed to decreased exercise metrics.

Study Design: Participants were randomly assigned to 150 minutes/week of either aerobic, muscular strength, or combined (aerobic+strength) moderate-intensity exercise, or a non-exercising stretching/breathing (control) group for ~24 weeks. At 4 and 24 weeks post-delivery, 1- and 6- month skinfold, length, and circumference measures and a blood draw were performed to assess infant body composition and lipid profile. Data were analyzed using analysis-of-variance and regression.

Results: Data from 180 participants were analyzed. Maternal descriptors were similar between groups, except prepregnancy (p=.008). Groups were similar in gestational age at birth. Controlling for GDM presence, GWG, and maternal 36 wk body fat%, infant sex (p<.001) and exercise type (.09) predict infant lean mass (R2=.13, p=.004). Controlling for infant sex, prepregnancy BMI, exercise type, and GDM presence, exercise attendance (p=.003) predicts infant LDL (R2=.35, p=.006). Controlling for exercise attendance, GDM presence, maternal BMI and 36 wk body fat, exercise type (p=.04) predicts infant triglycerides (R2=.31, p=.0497). Controlling for infant sex, exercise attendance, 36 wk fat %, exercise type (p=<.001) predicts infant nonHDL. Controlling for gravida, GWG, and

weekly exercise duration, 6-month lactate is predicted (R^2=0.79, p=.02) by exercise group [combination] and [control], maternal race [Asian], prepregnancy BMI, and average weekly METs.

Conclusions: These findings demonstrate that prenatal exercise may improve infant body composition and blood lipids measures. Further research is needed to determine if these differences persist past 1 month of age.

Introduction

Childhood obesity begins to take root early in life, even as young as 1 to 6 months of age, and is closely linked to metabolic challenges such as dyslipidemia and impaired glucose regulation. Infants who develop early signs of obesity often exhibit higher levels of low-density lipoprotein (LDL) cholesterol, lower levels of high-density lipoprotein (HDL) cholesterol, and insulin resistance[1-5]. These factors significantly increase their risk for cardiovascular disease, obesity, and type 2 diabetes later in life. The foundation for these metabolic challenges is often laid during critical early developmental periods, shaped by both genetic predispositions and environmental influences[115].

Maternal health and behaviors during pregnancy have a profound impact on an infant's metabolic health in the first months of life. Among these, maternal exercise has emerged as a vital intervention to reduce the risk of early obesity and related metabolic disorders[79]. Regular physical activity during pregnancy improves maternal glucose regulation and reduces the risk of gestational diabetes—a condition strongly associated with adverse

metabolic outcomes in infants. Studies suggest that infants born to mothers who exercised during pregnancy tend to have healthier lipid profiles, better insulin sensitivity, and improved early cardiovascular markers[82-85,88-89].

Maternal exercise also plays a role in shaping the metabolic programming of infants through its influence on fetal epigenetics. These changes are particularly evident in pathways related to lipid and glucose metabolism and body composition[79]. Exercise-induced improvements in maternal insulin signaling and lipid regulation may result in healthier metabolic trajectories for infants, helping to protect against early adiposity and elevated blood lipid levels—key precursors to lifelong cardiometabolic diseases.

Furthermore, maternal exercise is linked to reduced inflammation during pregnancy and optimal fetal growth, which are critical for preventing early metabolic disorders in infants, such as dyslipidemia and hyperglycemia. These protective effects can be seen as early as the first month of life and remain evident at 6 months, promoting better health outcomes during a crucial period of growth and development. Early attention to maternal health and activity levels can have lasting benefits for the metabolic health of children[80]. Therefore, understanding how maternal exercise enhances infant body composition, blood biomarkers, and overall health in the postpartum period empowers clinicians, exercise professionals, and expectant mothers to make informed decisions.

Methods

Participants and Recruitment

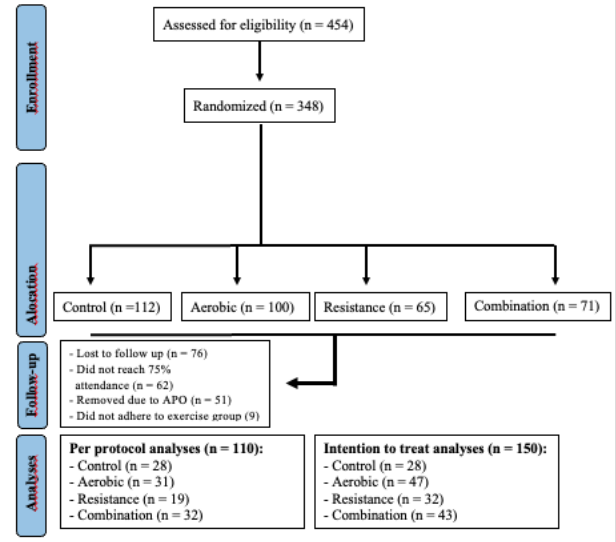

Fig. S2 Flow chart of the study participants.

This is a post-hoc secondary analysis of a randomized, blinded, prospective study focused on determining the influence of maternal exercise types on maternal and offspring health outcomes. All protocols were approved by the East Carolina University Institutional Review Board. Recruitment was focused on low-risk, healthy women with a singleton pregnancy. Women enrolled in the study met the following criteria: clearance from an obstetric provider to participate in exercise; between ages 18 and 40; pre-pregnancy BMI of 18.5–39.9; gestational age ≤ 16 weeks; no current alcohol, tobacco, or medication use. Criteria for exclusion were pre-existing diabetes mellitus, hypertension, cardiovascular disease, and medications known to affect fetal growth and well-being. Written informed consent was obtained from each participant; each participant subsequently completed a treadmill test and 1-repetition maximum strength tests. Using concealed randomization methods (GraphPad Prism Software), participants were randomly assigned to aerobic, resistance, combination (aerobic + resistance) exercise, or stretching-breathing attention control. Due to the nature of an exercise intervention, the participants and trainers knew the group assignment.

Pre-Intervention Exercise Testing

Participants recruited before March 2020, the COVID-19 pandemic, completed a submaximal modified Balke treadmill test following previously described protocols[92] to determine the target heart rate (HR) zone for exercise training. To minimize exposure and potential risk associated with COVID-19, women recruited following March 2020 did not complete the treadmill protocol, and target HR zones for exercise were determined based on their pre-pregnancy physical activity level and age, using published guidelines for pregnant women[92]. Target HR zones for exercise corresponded to maternal HR at 60 to 80% of maximal oxygen consumption, reflecting moderate intensity[92].

Exercise Intervention

Participants trained according to American College of Obstetricians and Gynecologists guidelines for approximately 24 weeks, beginning at 16 weeks gestation and continuing until delivery (~40 weeks gestation)[93]. Each exercise training session included a 5-minute warmup, a 50-minute exercise period, and a 5-minute cooldown. Every training session, regardless of training type, was supervised; participants exercised within their target HR zone corresponding to moderate intensity (60%-80% maximal oxygen consumption and 12-14 rated perceived exertion) throughout the exercise session. HR was monitored (Polar FS2C HR Monitor) throughout each exercise session to ensure that participants remained within their target HR ranges for the duration of the session. All sessions were performed three times per week until delivery. The aerobic exercise (AE) group completed moderate-intensity training for 50 minutes on the treadmill, elliptical,

recumbent bicycle, rowing, and/or stair-stepping equipment. To maintain the appropriate HR zone, speed, grade, and/or resistance were adjusted. The resistance exercise (RE) group completed sessions consisting of two to three sets of 15 repetitions of each exercise at moderate intensity[94]. Seated Cybex machines (leg extension, leg curl, shoulder press, chest press, triceps extension, latissimus dorsi pull-down), dumbbells (biceps curls, lateral shoulder raises, front shoulder raises), resistance bands, dumbbells, exercise balls, benches, and/or mats were used. The combination exercise (CE) group performed five 4.5-5 minutes long alternating bouts of aerobic and resistance exercise, each for 50 minutes per workout. The CE group performed aerobic and resistance exercises on the same equipment as the AE and RE groups. The control group (CON) performed supervised stretching, breathing, and flexibility exercises for the duration of the session. The breathing exercises and stretches of CON participants targeted major muscle groups[94] and maintained a low intensity (<40% VO_{2peak}) throughout the session. Maternal exercise intensity in METs was determined based on the published compendium of physical activity for the exercise performed in each session[95]. The ability of this protocol to elicit maternal physiological adaptations to exercise has been previously reported[96].

Maternal Exercise Metrics

Exercise metrics such as frequency, intensity, duration, and type were recorded from the training logs across pregnancy. Exercising women aimed to complete aerobic, resistance, or combination exercise 3 times per week, for 50 minutes per session, at moderate intensity (3 – 6 METs). To control for differences in the duration of pregnancy and the start

of the intervention, exercise metrics were analyzed from 16 to 36 weeks for all participants.

Frequency was calculated as the number of times the participant attended supervised exercise each week and is expressed as sessions per week. Intensity was calculated using the published compendium of physical activity (19, 20) for each specific exercise performed. The average intensity (METs) was calculated via an average of these METs through all exercise sessions. Time was determined by exercise duration, in minutes within each week, with average duration reported as minutes/week. Lastly, prenatal exercise volume was calculated by 1) multiplying average intensity (METs) by the average weekly duration for volume in MET*min/wk (weekly exercise volume), and 2) multiplying weekly MET*min by the total number of weeks prenatal exercise was performed through pregnancy for total MET*min (total pregnancy volume).

Maternal Measurements

Maternal age, gravida, parity, pre-pregnancy weight and height, gestational weight gain (GWG), length of gestation, infant sex, birth length, and birth weight were abstracted from various sources, including pre-screening eligibility, postpartum questionnaires, as well as maternal and neonatal electronic health records. Gestational weight gain was calculated by subtracting maternal pre-pregnancy weight from weight at delivery and was extracted from electronic health records, while weight before pregnancy (lbs.) was collected via the pre-screening eligibility questionnaires. At 16 and 36 weeks of gestation, maternal height and weight were obtained using a stadiometer and calibrated medical grade scale; BMI

was calculated as [weight (kg)] / [height (m) ^{2}] (healthy BMI 18.5-24.9; overweight 25-29.9, obese $\geq$30).

Birth Outcomes

At birth, delivery type (spontaneous vaginal delivery, C-section), neonate sex, gestational age, and neonatal measures (heart rate, chest, head, and abdominal circumferences, birth weight, length) were measured by labor & delivery nurses, using standard protocols, and entered into the electronic health record. Head circumference-abdominal circumference (HCAC) and weight-length ratios were calculated.

One- and Six-month Infant Measurements

Infant percent body fat was determined using a previously validated skinfold technique and age-adjusted equations[122-123]; sites include triceps, subscapular, and biceps. Circumferences were measured at the head and abdomen. Further, via venipuncture, blood biomarkers, glucose, lactate, and lipid profiles were completed. Two drops of blood from the sample were used to measure glucose, lipids (NonHDL, TC, TG, HDL, LDL), and lactate with Cholestech (Abbott, Orlando FL) and lactate analyzer (Nova Biomedical, Waltham MA), respectively.

Statistical Analysis

ANOVA was used to determine statistical significance between groups where appropriate. Kruskal-Wallis (4-group comparison) and Mann-Whitney (2-group comparison; exercise vs. control) tests were used to assess the difference between non-normally distributed variables (gravida and parity). Linear Regressions were completed to determine predictors of infant body composition and blood biomarker outcomes. Based on preliminary infant blood biomarker data, power analysis indicated a sample of 9 per group was needed to detect group differences in 1-month NonHDL cholesterol. Statistical significance was set at $p \leq 0.05$. Statistical analyses were performed using IBM SPSS Statistics (IBM Corp., Armonk, NY) and JMP Statistical Discovery (Cary, NC) for Mac.

Results

Maternal and infant birth descriptors. We were contacted by 454 pregnant women interested in the study (Figure 3.1). Our final analysis included 150 participants (CON=28, AE=47, RE=32, AERE=43). The intervention is conducted from September 1, 2018, to January 30, 2023. For the intention-to-treat (ITT) analysis, participants were similar in age, gravida, parity, race, and gestational length. Pre-pregnancy BMI is higher in the Control group (Table 4.1). For the per-protocol analysis, a total of 110 women met ≥75% adherence to exercise training throughout the ~24 weeks (CON=28, AE=31, RE=19, AERE=32). There were no differences between groups for age, gravida, parity, race, or length of gestation. Pre-pregnancy BMI is higher in the Control group, and GWG was higher in the Resistance group (Table 4.2).

Table 4.1: Intention to Treat Maternal Descriptors

	Full Sample	Control	Aerobic	Resistance	Combination	p-value
Maternal Measures	(N=150)	(N=28)	(N=47)	(N=32)	(N=43)	
Age (years)	30.28 ± 4.18	30.04 ± 3.83	30.33 ± 4.77	30.47 ± 3.71	30.20 ± 4.19	.82
Prepregnancy BMI	25.17 ± 4.50	27.66 ± 5.35	23.92 ± 4.21**	24.95 ± 4.71	25.25 ± 3.58	**.01**
Gravida†	2.0 (1, 7)	2.0 (1, 4)	2.0 (1, 4)	2.0 (1, 5)	2.0 (1, 7)	.59
Parity†	1.0 (0, 4)	1.0 (0, 3)	1.0 (0, 2)	1.0 (0, 4)	1.0 (0, 3)	.60
Attendance	85.48 ± 17.84	--	83.40 ± 16.41	78.19 ± 19.57	85.20 ± 17.53	.19
Race						.14
White	125 (83.3%)	19 (67.9%)	37 (78.7%)	29 (90.6%)	40 (93%)	
Black	15 (10%)	6 (21.4%)	5 (10.6%)	2 (6.3%)	2 (4.7%)	
Other	10 (6.7%)	3 (10.7%)	5 (10.6%)	1 (3.1%)	1 (2.3%)	
GWG	33.11 ± 10.66	29.59 ± 10.71**	30.30 ± 9.89	36.44 ± 11.27	35.48 ± 9.87	**.01**
Length of Gestation (wks)	39.72 ± 1.03	39.42 ± 1.18	39.74 ± 1.11	39.94 ± 0.90	39.69 ± 0.95	.28
Infant Measures						
Infant Sex (Male)	79 (52.7%)	18 (64.3%)	26 (55.3%)	18 (56.2%)	17 (39.5%)	.19
Birth Length (m)	0.50 ± 0.02	0.50 ± 0.03	0.49 ± 0.03	0.50 ± 0.02	0.50 ± 0.02	.45
Birth Weight (kg)	3.55 ± 0.44	3.53 ± 0.59	3.54 ± 0.39	3.60 ± 0.48	3.52 ± 0.37	.87

Presented as mean +/- SD. † due to non-normal distribution, Mann Whitney U test performed and expressed as median (minimum, maximum). GWG - Gestational Weight Gain. *p < 0.05, **p < 0.01, p*** < 0.001 compared to control

Table 4.2: Per Protocol Maternal Descriptors

	Full Sample	Control	Aerobic	Resistance	Combination	p-value
	(N=110)	(n=28)	(N=31)	(N=19)	(N=32)	
Age (years)	30.59 ± 3.88	30.04 ± 3.83	30.66 ± 4.29	31.37 ± 3.43	30.45 ± 3.87	.47
Prepregnancy BMI	25.12 ± 4.66	27.66 ± 5.35	23.70 ± 4.36 **	23.79 ± 4.17 *	25.41 ± 3.90	**.004**
Gravida†	2.00 (1, 4)	2.00 (1, 4)	2.00 (1, 4)	2.00 (1,4)	2.00 (1,3)	.54
Parity†	1.00 (0, 3)	1.00 (0,3)	1.00 (0, 2)	1.00 (0, 3)	1.00 (0, 1)	.62
Attendance	0.94 ± 0.07	--	0.93 ± 0.06	0.90 ± 0.05	0.93 ± 0.07	.26
Race						.18
White	89 (80.9%)	19 (67.9%)	23 (74.2%)	16 (84.2%)	31 (96.9%)	

Black	11 (10%)	6 (21.4%)	4 (12.9%)	2 (10.5%)	0 (0%)	
Other	9 (8.2%)	3 (10.7%)	4 (12.9%)	1 (5.3%)	1 (3.1%)	
GWG	32.86 ± 9.79	29.59 ± 10.71	31.88 ± 8.83	38.25 ± 8.56 *	32.92 ± 9.72	**.04**
Length of Gestation (wks)	39.72 ± 1.01	39.42 ± 1.18	39.37 ± 1.07	40.07 ± 0.81	39.66 ± 0.87	.18
Infant Measures						
Infant Sex (Male)	60 (54.5%)	17 (63%)	18 (58.1%)	12 (63.2%)	12 (37.5%)	.14
Birth Weight (kg)	3.51 ± 0.44	3.53 ± 0.59	3.48 ± 0.38	3.57 ± 0.44	3.51 ± 0.38	.71
Birth Length (m)	0.50 ± 0.02	0.50 ± 0.03	0.49 ± 0.03	0.50 ± 0.02	0.50 ± 0.02	.07
Presented as mean +/- SD. † due to non-normal distribution, Mann Whitney U test performed and expressed as median (minimum, maximum). GWG - Gestational Weight Gain. *p < 0.05, **p < 0.01, p*** < 0.001 compared to control						

Maternal Descriptors. For the per protocol, there were between-group differences in pre-pregnancy BMI (p=.004; CON to AE and RE) and GWG (p-value=.04; RE to CON) (Table 4.2).

1-month Infant Outcomes. Controlling for prepregnancy BMI, exercise attendance, and maternal race, 1-month infant length is predicted (R^2=0.23, p=<.0001) by average weekly METs, gravida, and parity. Controlling parity and maternal race, 1-month length is predicted (R^2=0.15, p=.0005) by exercise attendance, gravida, and total exercise volume. 1-month infant weight is predicted (R^2=0.07, p=.01) by GWG, gravida, and prepregnancy BMI. Controlling for exercise attendance, weekly exercise duration, gravida, and weekly exercise frequency, 1-month infant fat-free mass percent, and body fat percent is predicted (R^2=0.20, p=.005) by exercise group [aerobic] and [control], GWG, and maternal race [Asian]. Controlling for GWG and maternal race, 1-month infant fat-free mass percent and body fat percent are predicted (R^2=0.15, p=.005) by exercise group [aerobic] and [control], gravida, and exercise attendance. Controlling for GWG and prepregnancy BMI, 1-month BMI is predicted (R^2=0.06, p=.02) by average weekly METs.

Controlling for GWG and maternal age, 1-month infant total cholesterol is predicted (R^2=0.29, p=.004) by weekly exercise duration and maternal race [Asian] and [Black]. Controlling for gravida and maternal race, 1-month infant LDL cholesterol is predicted (R^2=0.37, p=.0002) by weekly exercise frequency. Controlling for exercise attendance, 1-month-old infant HDL cholesterol is predicted (R^2=0.18, p=.03) by parity and exercise group [aerobic]. Controlling for maternal race, gravida, GWG, and exercise attendance, 1-month-old infant NonHDL cholesterol is predicted (R^2=0.43, p=.0007) by weekly exercise frequency (Table 4.3).

Table 4.3. 1mo Infant Body Composition and Blood Biomarker Regression Models

	Estimate	STD Error	t Ratio	Lower 95%	Upper 95%	p-value
Length[1] R^2 = 0.23 p = <0.0001						
Average Weekly METs	2.31	0.50	4.63	1.32	3.31	<.0001
Gravida	1.20	0.50	2.38	0.20	2.20	0.02
Parity	-1.34	0.62	-2.15	-2.57	-0.11	0.03
Length[2] R^2 = 0.15 p = 0.005						
Attendance	-6.04	1.86	-3.26	-9.72	-2.37	0.002
Gravida	1.20	0.52	2.29	0.0.16	2.34	0.02
Total Exercise Volume	0.0002	0.00001	2.26	-0.00002	0.0003	0.03
Weight[3] R^2 = 0.07 p = 0.01						
GWG	0.01	0.005	2.54	0.003	0.02	0.01
Maternal Race [Black]	-0.27	0.12	-2.15	-0.51	-0.02	0.03
Prepregnancy BMI	0.02	0.01	2.06	0.001	0.04	0.04
Fat free Mass Percent[4] R^2 = 0.20 p = 0.005						
Exercise Group [Aerobic]	1.42	0.55	2.61	0.34	2.51	0.01
Exercise Group [Combination]	-1.33	0.61	-2.19	-2.53	-0.13	0.03
GWG	0.07	0.03	2.13	0.005	0.13	0.04
Maternal Race [Asian]	-1.85	0.87	-2.11	-3.58	-0.12	0.04

Fat free Mass Percent[5]	**R^2 = 0.15**	**p = 0.002**				
Exercise Group [Aerobic]	1.19	0.46	2.60	0.29	2.09	0.01
Gravida	-0.77	0.30	-2.60	-1.36	-0.18	0.01
Attendance	4.24	1.74	2.43	0.80	7.68	0.02
Exercise Group [Combination]	-0.99	0.49	-2.03	-1.95	-0.03	0.04
Body Fat Percent[5]	**R^2 = 0.15**	**p = 0.002**				
Exercise Group [Aerobic]	-1.19	0.46	-2.60	-2.09	-0.29	0.01
Gravida	0.77	0.30	2.60	0.18	1.36	0.01
Attendance	-4.24	1.74	-2.43	-7.68	-0.80	0.02
Exercise Group [Combination]	0.99	0.49	2.03	0.03	1.95	0.04
Body Fat Percent[4]	**R^2 = 0.20**	**p = 0.005**				
Exercise Group [Aerobic]	-1.42	0.55	-2.61	-2.51	-0.34	0.01
Exercise Group [Combination]	1.33	0.61	2.19	0.13	2.53	0.03
GWG	-0.07	0.03	-2.13	-0.13	-0.005	0.04
Maternal Race [Asian]	1.85	0.87	2.11	0.12	3.58	0.04
Body Mass Index[6]	**R^2 = 0.06**	**p = 0.02**				
Average Weekly METs	-0.78	0.34	-2.29	-1.45	-0.11	0.02
Total Cholesterol[7]	**R^2 = 0.29**	**p = 0.004**				
Weekly Exercise Duration	-0.31	0.11	-2.80	-0.54	-0.09	0.007
Maternal Race [Asian]	21.64	8.11	2.67	5.35	37.93	0.01
Maternal Race [Black]	-16.06	7.90	-2.03	-31.92	-0.20	0.04
LDL Cholesterol[8]	**R^2 = 0.37**	**p = 0.0002**				
Weekly Exercise Frequency	-25.40	5.25	-4.84	-35.97	-14.83	<.0001
HDL Cholesterol[9]	**R^2 = 0.18**	**p = 0.03**				
Parity	7.27	2.49	2.92	2.28	12.26	0.005
Exercise Group [Aerobic]	7.42	3.18	2.33	1.05	13.79	0.02
NonHDL Cholesterol[10]	**R^2 = 0.43**	**p = 0.0007**				
Weekly Exercise Frequency	-25.59	6.17	-4.15	-38.07	-13.11	0.0002

Model 1: Average Weekly METs, Gravida, Parity, Prepregnancy BMI, Attendance, Maternal Race
Model 2: Attendance, Gravida, Total Exercise Volume, Parity, Maternal Race
Model 3: GWG, Prepregnancy BMI, Maternal Race
Model 4: Exercise Group, Maternal Race, GWG, Attendance, Weekly Exercise Duration, Gravida, Weekly Exercise Frequency
Model 5: Gravida, Attendance, Exercise Group, GWG, Maternal Race
Model 6: Average Weekly METs, GWG, Prepregnancy BMI
Model 7: Weekly Exercise Duration, Maternal Race, GWG, Age
Model 8: Weekly Exercise Frequency, Gravida, Maternal Race
Model 9: Parity, Exercise Group, Attendance
Model 10: Weekly Exercise Frequency, Maternal Race, Gravida, GWG, Attendance
*p<0.05, **p<0.01, ***p<0.001

6-month Infant Outcomes. Controlling for maternal age, exercise group, weekly exercise frequency, and maternal race, 6-month infant length is predicted (R^2=0.27, p=.005) by gravida and prepregnancy BMI. Controlling for gravida, 6-month infant weight is predicted (R^2=0.16, p=.006) by maternal age, and exercise group [aerobic] and [control]. Controlling for gravida, prepregnancy BMI, average weekly METs, weekly exercise duration, GWG, weekly exercise frequency, and total exercise training, 6-month infant fat-free mass percent is predicted (R^2=0.22, p=.003) by maternal race [Asian] and maternal age. Controlling for gravida, prepregnancy BMI, total exercise volume, and GWG, 6-month body fat percent is predicted (R^2=0.30, p=.0009) by maternal race [Asian] and maternal age. Controlling for maternal race, prepregnancy BMI, and maternal age, 6-month BMI is predicted (R^2=0.13, p=.05) by weekly exercise duration. Controlling for the exercise group, parity, GWG, and maternal age, the 6-month head/abdominal circumference ratio is predicted (R^2=0.41, p=.0003) by gravida, weekly exercise duration, weekly exercise frequency, and maternal race [Asian] and [Black]. Controlling for gravida, GWG, and weekly exercise duration, 6-month lactate is predicted (R^2=0.79, p=.02) by exercise group [combination] and [control], maternal race [Asian], prepregnancy BMI, and average weekly METs (Table 4.4).

Table 4.4. 6mo Infant Body Composition and Blood Biomarker Regression Models

		Estimate	STD Error	t Ratio	Lower 95%	Upper 95%	p-value
Length[1]	**R^2 = 0.27**	**p = 0.005**					
Gravida		1.01	0.33	3.10	0.36	1.66	0.003
Prepregnancy BMI		-0.17	0.07	-2.37	-0.32	-0.03	0.02
Weight[2]	**R^2 = 0.16**	**p = 0.006**					
Maternal Age		0.98	0.04	25.31	0.91	1.06	<0.0001

Exercise Group [Aerobic]	-0.28	0.10	-2.68	-0.49	-0.07	0.009
Exercise Group [Combination]	12.73	5.35	2.38	2.09	23.37	0.02
Fat free Mass Percent[3] R^2 = 0.33 p = 0.003						
Maternal Race [Asian]	-3.22	1.15	-2.80	-5.51	-0.92	0.007
Maternal Age	0.28	0.10	2.70	0.07	0.49	0.009
Body Fat Percent[4] R^2 = 0.30 p = 0.0009						
Maternal Race [Asian]	3.18	1.12	2.84	0.95	5.42	0.006
Maternal Age	-0.27	0.10	-2.66	-0.47	-0.07	0.01
Body Mass Index[5] R^2 = 0.13 p = 0.05						
Weekly Exercise Duration	0.03	0.02	2.17	0.003	0.07	0.03
HCAC Ratio[6] R^2 = 0.41 p = 0.0003						
Gravida	-0.04	0.01	-3.40	-0.06	-0.02	0.001
Maternal Race [Black]	0.05	0.02	2.74	0.01	0.09	0.008
Weekly Exercise Duration	-0.002	0.0008	-2.62	-0.004	-0.0005	0.01
Maternal Race [Asian]	-0.05	0.02	-2.62	-0.08	-0.02	0.01
Weekly Exercise Frequency	0.10	0.05	2.14	0.007	0.19	0.04
Lactate[7] R^2 = 0.79 p = 0.02						
Exercise Group [Combination]	-1.32	0.37	-3.62	-2.13	-0.51	0.005
Maternal Race [Asian]	-1.17	0.37	-3.14	-2.01	-0.34	0.01
Exercise Group [Control]	1.74	0.64	2.71	0.31	3.18	0.02
Prepregnancy BMI	-0.17	0.07	-2.59	-0.32	-0.02	0.03
Average Weekly METs	0.96	0.41	2.38	0.060	1.87	0.04

Model 1: Gravida, Prepregnancy BMI, Age, Exercise Group, Weekly Exercise Frequency, Maternal Race
Model 2: Maternal Age, Exercise Group, Gravida
Model 3: Maternal Age, Maternal Race, Gravida, Prepregnancy BMI, Average Weekly METs, Weekly Exercise Duration, GWG, Weekly Exercise Frequency, Total Exercise Training
Model 4: Maternal Age, Maternal Race, Gravida, Prepregnancy BMI, Total Exercise Volume, GWG
Model 5: Weekly Exercise Duration, Maternal Race, Prepregnancy BMI, Maternal Age
Model 6: Gravida, Weekly Exercise Duration, Maternal Race, Exercise Group, Weekly Exercise Frequency, Parity, GWG, Maternal Age
Model 7: Maternal Race, Prepregnancy BMI, Exercise Group, Average Weekly METs, Gravida, GWG, Weekly Exercise Duration
*p<0.05, **p<0.01, ***p<0.001

Discussion

The current study aimed to determine the influence of maternal exercise FITT-V on infant body composition and blood biomarker outcomes. We hypothesized that maternal

exercise FITT-V would improve infant body composition and blood biomarker outcomes, such as decreasing body fat percent, cholesterol levels, and lactate while increasing fat-free mass percent, and HDL-C. We found a plethora of significant associations between exercise FITT-V and infant body composition and blood biomarkers. Relationships were found for many specific FITT-V metrics. In brief, weekly exercise volume is related to increased 1-month length; weekly exercise duration decreases 1-month total cholesterol; weekly exercise frequency is associated with decreased 1-month nonHDL cholesterol and LDL cholesterol and increased 6-month head circumference-to-abdominal circumference ratio; while exercise intensity decreases 1-month BMI. These effects were specific to exercise type, with AE showing more associations than RE or AERE, especially in body weight, body fat percent, and HDL cholesterol. The findings from this study align with existing research on adult populations, particularly regarding the benefits of exercise for metabolic health and body composition. However, the study adds a novel perspective by examining how maternal exercise FITT-V influences infant body composition and blood biomarkers outcomes.

Maternal Exercise Volume

Increased exercise volume was associated with increased infant length at 1 month and no other body composition or blood biomarker effects. This is contrary to what is found in research in adults, that higher total exercise volume leads to greater improvements in body composition, cardiovascular health, and metabolic markers[99,116]. Studies have demonstrated that individuals who accumulate greater weekly exercise volume experience more pronounced reductions in body fat, greater improvements in insulin

sensitivity, and enhanced lipid metabolism compared to those with lower weekly exercise volume[99,116]. While the specific mechanisms linking maternal exercise volume to infant growth remain unclear, the findings highlight the potential benefits of maintaining a consistent moderate level of physical activity throughout pregnancy.

Maternal Exercise Duration

Increased exercise duration was associated with lower total cholesterol at 1 month. This is similar to what is seen in research on adults, which indicates that longer exercise sessions contribute to improved cardiovascular health, lower total cholesterol, and greater fat loss. Studies have found that adults who engage in longer bouts of moderate-to-vigorous physical activity experience significant reductions in total cholesterol and triglycerides[118]. This suggests that prolonged maternal physical activity may support early lipid regulation in offspring, potentially setting the stage for long-term metabolic benefits. Research in adults emphasizes that sustained exercise reduces systemic inflammation, increases fat oxidation, and improves lipid clearance, mechanisms that may also play a role in fetal development by influencing placental function and nutrient transport[118-119].

Maternal Exercise Frequency

Increased exercise frequency was associated with lower LDL and non-HDL cholesterol at 1 month and a higher head circumference-to-abdominal circumference ratio at 6 months. This is similar to what is found in research in adults that consistently shows that more

frequent exercise is associated with improved lipid profiles, reduced body fat, and better cardiovascular health. Studies indicate that engaging in physical activity multiple times per week lowers LDL and non-HDL cholesterol while increasing HDL cholesterol, reducing the risk of cardiovascular disease. The decrease in LDL and non-HDL cholesterol aligns with adult research showing that regular exercise enhances lipid metabolism and reduces cardiovascular risk factors by upregulating enzymes involved in cholesterol transport and clearance[111-113-115]. The increase in the head-to-abdominal circumference ratio suggests a potential influence on early growth patterns, though this specific relationship has not been widely explored in adult populations.

Maternal Exercise Intensity

Increased exercise intensity was associated with decreased infant BMI at 1 month. This is similar to what is found in research in adults demonstrating that high-intensity exercise is particularly effective for reducing body fat, improving insulin sensitivity, and enhancing cardiovascular function. High-intensity interval training (HIIT), for example, has been shown to decrease BMI, improve glucose regulation, and increase fat oxidation[116-117]. This supports the idea that maternal exercise intensity could influence fetal energy metabolism and growth regulation, much like how high-intensity exercise in adults promotes fat oxidation and weight control through increased mitochondrial efficiency and hormonal adaptations[124]. However, while higher-intensity exercise in adults often leads to greater improvements in HDL cholesterol, this study did not highlight strong associations between maternal exercise intensity and infant lipid markers, suggesting that other factors may mediate lipid metabolism in early infancy.

Maternal Exercise Type

The current study utilized data from participants engaged in aerobic (AE), resistance (RE), and combination exercise (AERE). This study found that AE had the strongest associations with infant body weight, body fat percentage, and HDL cholesterol, suggesting a prenatal advantage of aerobic activity. The relatively weaker associations for RE and AERE (combined aerobic and resistance exercise) contrast with adult studies, where combined training is often the most effective for overall fitness and metabolic health[119]. In adult research, aerobic exercise (AE) is widely regarded as the most effective form of exercise for reducing body fat and improving lipid profiles, whereas resistance exercise (RE) is more strongly associated with increases in lean muscle mass and strength. Long-term studies show that regular aerobic exercise leads to significant reductions in body fat percentage, lower LDL cholesterol, and higher HDL cholesterol[120].

Strengths & Limitations

The current analysis is the first to demonstrate a direct effect of maternal exercise FITT-V on infant body composition and blood biomarker outcomes at one-month age. The study is strengthened by its large sample size, derived from rigorous supervised RCT exercise intervention trials. The diverse range of outcomes measured provides a comprehensive picture of the effects of maternal exercise FIIT-V on infant body composition and blood biomarker outcomes. However, the study has some limitations. Some body composition measures were obtained as standard procedures; skinfold measurements may have

interindividual differences between study staff. These are standard processes in the research team and, thus, likely have a small margin of error. Overall, these initial findings highlight the need for further research in this area and support continued work on this topic.

Conclusion

These findings reinforce established adult research on exercise benefits while extending them to a prenatal context, showing intergenerational effects. The strong influence of exercise frequency and duration on lipid profiles suggests that consistent maternal physical activity could help regulate early metabolic health in offspring, much like regular exercise in adults improves long-term cardiovascular health. The dominant role of aerobic exercise in shaping infant outcomes further highlights its potential advantages during pregnancy compared to other forms of exercise, mirroring adult studies that emphasize AE as the most effective exercise type for fat loss and cardiovascular benefits. Additionally, the positive association between maternal exercise volume and infant growth suggests that a comprehensive approach to physical activity—including high frequency, sufficient duration, and adequate intensity—may be most beneficial. These results support the idea that maternal exercise FITT-V influences infant health in ways that mirror adult exercise research, emphasizing the importance of structured physical activity during pregnancy as a means of promoting optimal early-life health outcomes.

These results underscore the need for further research to refine optimal exercise strategies for pregnant women, ensuring safety while maximizing health benefits for both mothers and their developing children. Structured exercise during pregnancy can significantly enhance infant metabolic and body composition outcomes, contributing to long-term child health.

Chapter 5

Summary and Conclusion

Obesity is a significant global health issue, particularly among reproductive-aged women, posing risks during pregnancy and long-term consequences for both mother and child. Excess adiposity during pregnancy is associated with complications such as gestational diabetes, hypertension, and preeclampsia, which negatively affect maternal and fetal health[7-11]. Obesity also disrupts metabolic processes, including lipid and glucose regulation, potentially leading to insulin resistance and increased risks of gestational diabetes[22,43,69-70,83]. Interventions like regular exercise can improve lipid profiles, insulin sensitivity, and maternal body composition, mitigating these risks and promoting healthier pregnancy outcomes[89-91]. Maternal exercise plays a pivotal role in reducing childhood obesity and its associated metabolic challenges. Regular physical activity during pregnancy enhances maternal glucose regulation, reduces systemic inflammation, and supports healthier fetal growth. It also influences fetal gene expression related to lipid and glucose metabolism, fostering beneficial metabolic programming[79]. These adaptations contribute to long-term improvements in offspring's metabolic health, reducing risks for obesity, cardiovascular disease, and type 2 diabetes[90]. Research highlights that maternal exercise, combined with balanced nutrition, offers a comprehensive strategy to promote healthier body composition and metabolic outcomes in both mothers and their children. This dual approach helps address early signs of obesity in infants, improving lipid profiles, insulin sensitivity, and cardiovascular health markers. Encouraging maternal exercise during pregnancy empowers clinicians and expectant mothers to optimize health outcomes for future generations.

In aim one of this dissertation, we show the impact of maternal exercise, using the FITT-V (Frequency, Intensity, Time, Type, and Volume) model, on maternal body composition and blood biomarkers during pregnancy. The results showed that exercise positively influenced various metabolic health markers and body composition. Specifically, increased weekly exercise volume was associated with decreased body weight, reduced body fat, and improved body composition, including lower skinfold thickness. Longer exercise durations (45-60 minutes) contributed to better body composition, particularly by lowering fat percentage, waist circumference, and triceps skinfold thickness, though it did not consistently affect metabolic markers like glucose or cholesterol. Increased exercise frequency led to improvements in metabolic health, including reductions in insulin resistance and lactate levels, while promoting fat loss and lean muscle gain. Moderate-intensity exercise, such as walking or jogging, proved effective for maintaining metabolic health during pregnancy and was linked to gradual fat loss and muscle retention. However, higher exercise intensity was associated with increased cholesterol levels and did not show the same benefits for metabolic regulation as in non-pregnant individuals, highlighting the need for tailored recommendations during pregnancy. Regarding exercise type, aerobic exercise (AE) was the most beneficial for improving metabolic outcomes, such as reducing lactate and insulin levels, while resistance exercise (RE) helped maintain lean mass and improved waist-to-hip ratio. Surprisingly, the combination of aerobic and resistance exercises (AERE) did not show the expected benefits, potentially due to the diluted effects of combining both types. Overall, aerobic exercise was found to be especially advantageous for metabolic health and body composition during pregnancy,

while resistance exercise provided complementary benefits, particularly in maintaining muscle mass and alleviating pregnancy-related discomforts like back pain. These findings suggest that a balanced exercise program, with a focus on aerobic activity and supplemented by resistance training, is optimal for promoting maternal and fetal health during pregnancy and supporting postpartum recovery.

In aim 2, maternal exercise during pregnancy significantly influenced infant body composition and cholesterol levels, with aerobic and resistance exercises offering distinct benefits. These maternal changes positively influence the fetal lipid environment, potentially reducing the infant's risk of high cholesterol and future metabolic disorders. The combined use of aerobic and resistance exercises theoretically may amplify these benefits by optimizing the intrauterine environment and influencing fetal gene expression related to lipid metabolism, setting the stage for better long-term cholesterol regulation in the offspring. In this study, aerobic exercise significantly reduced non-HDL cholesterol and increased HDL cholesterol in 1-month-old infants. This suggests that different types of maternal exercise during pregnancy can have distinct effects on infant cholesterol profiles. Overall, maternal exercise, particularly aerobic and resistance exercises, plays a crucial role in shaping infant metabolic health outcomes.

In aim 3, maternal exercise during pregnancy maternal exercise frequency and duration were strongly associated with improved infant lipid profiles, mirroring adult studies that link consistent physical activity to better cardiovascular health. Exercise intensity was

linked to lower infant BMI, reflecting similar fat-loss and metabolic benefits observed in adults. Aerobic exercise (AE) showed the most significant associations with improved infant body composition and cholesterol levels, paralleling findings in adults where AE is the most effective for fat loss and lipid regulation. Additionally, higher maternal exercise volume was associated with increased infant length, suggesting that greater prenatal exercise exposure may contribute to early growth patterns, much like how higher exercise volume in adults promotes muscle development and metabolic efficiency[119].

Overall, these results highlight the intergenerational impact of maternal physical activity, demonstrating that structured prenatal exercise can influence infant health in ways that resemble adult exercise adaptations. Given these parallels, promoting maternal exercise—particularly aerobic activity with adequate frequency, duration, and intensity—may serve as a valuable strategy for optimizing maternal and early-life health outcomes and establishing a foundation for long-term metabolic well-being.

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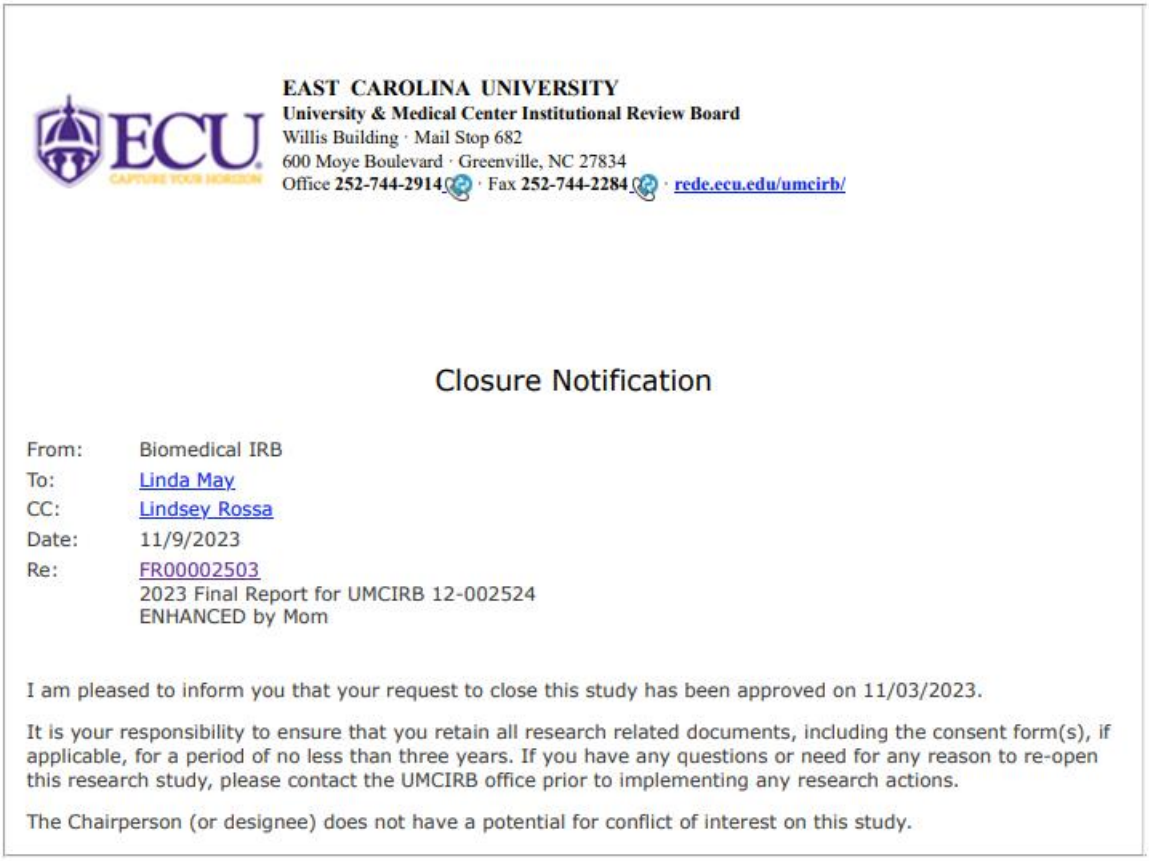
Appendix A: University and Medical Center Institutional Review Board (UMCIRB) Approval



EAST CAROLINA UNIVERSITY
University & Medical Center Institutional Review Board
Willis Building · Mail Stop 682
600 Moye Boulevard · Greenville, NC 27834
Office **252-744-2914** · Fax **252-744-2284** · rede.ecu.edu/umcirb/

Closure Notification

From: Biomedical IRB
To: Linda May
CC: Lindsey Rossa
Date: 11/9/2023
Re: FR00002503
2023 Final Report for UMCIRB 12-002524
ENHANCED by Mom

I am pleased to inform you that your request to close this study has been approved on 11/03/2023.

It is your responsibility to ensure that you retain all research related documents, including the consent form(s), if applicable, for a period of no less than three years. If you have any questions or need for any reason to re-open this research study, please contact the UMCIRB office prior to implementing any research actions.

The Chairperson (or designee) does not have a potential for conflict of interest on this study.