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Impact of Maternal Exercise during Pregnancy on Offspring Chronic Disease Susceptibility

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Abstract

Maternal behaviors during pregnancy have been reported to impact offspring health in adulthood. In this review we explore the novel hypothesis that exercise during pregnancy can protect against chronic disease susceptibility in offspring. To date research has demonstrated that improvements in metabolic outcomes, cardiovascular risk, and cancer can occur in response to maternal exercise during pregnancy.

Keywords

Exercise; Pregnancy; Offspring; Cardiovascular disease; Diabetes; Cancer

INTRODUCTION

It is well established that maternal exercise during pregnancy has many beneficial health outcomes for mothers, such as improved fitness, a reduction of excessive weight gain, reduced risk for gestational diabetes, and better post-partum recovery (5). Based partly on these data the American College of Obstetricians and Gynecologist (ACOG) recommend pregnant women, free of complications, participate in at least 30 minutes of moderate intensity exercise on most, if not all, days of the week (1). Similarly, the 2008 Physical Activity Guidelines from the Department of Health and Human Services (DHHS) recommend healthy pregnant mothers perform at least 150 minutes of moderate-intensity aerobic activity per week during pregnancy and the postpartum period (32). Surprisingly, even in light of the countless data that support the safety and health benefits of maternal

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exercise during pregnancy, it has been reported that only 15% of women adhere to this recommendation (5).

Studies have indicated that pregnant mothers have the perception that physical activity during pregnancy can help control weight gain and glucose levels, maintain fitness, improve their energy and mood, ease labor, and improve infant health (19, 26, 31). However, women may not reach the recommendations for exercising during pregnancy because of feelings of discomfort, fatigue, illness, and lack of enjoyment while exercising (11, 26). Additionally, while women recognize the benefits of exercising during pregnancy, they rate rest and relaxation as more important than exercise during pregnancy (11, 26). While this review does not address ways to improve adherence to the exercise recommendations during pregnancy, it does provide more in depth information on the impact exercise can have on offspring health during adolescence and adulthood. The goal is to explore the published literature examining the central hypothesis that maternal exercise during pregnancy can protect against chronic disease susceptibility in offspring (Figure 1). Health care providers can use these data to encourage women to exercise during pregnancy. The influence of maternal exercise on offspring birth weight has been reported extensively elsewhere (26) so this review article will not cover birth weight or morphometric outcomes in the offspring.

METABOLIC DISORDERS

Prior work has established a link between offspring low birth weight and T2DM risk later in life (17, 33). While considerable research has been conducted to examine the influence of negative maternal behaviors and offspring risk for T2DM, little attention has been given to positive maternal behaviors, such as exercise, during pregnancy. We hypothesized that maternal exercise during pregnancy would decrease offspring risk for T2DM later in life. Consistent with this hypothesis, our laboratory recently reported that maternal voluntary exercise in mice fed a normal diet positively influenced both male and female offspring glucose and insulin tolerance during adulthood (7). We also found that female rat offspring born to exercising dams had significantly improved insulin sensitivity (measured by hyperinsulinemic-euglycemic clamp) compared to those born to sedentary dams (8). Beneficial effects on insulin sensitivity are observed in mice and rats, which suggest that the findings are not species specific. Maternal exercise may also affect obesity outcomes in a sex-specific manner as body composition was significantly affected in male mouse offspring only. Male offspring from exercised dams showed decreased fat and increased lean mass percentages compared to offspring born to sedentary dams, but maternal exercise did not significantly affect body composition in female mouse or rat offspring (7, 8). Unfortunately, male offspring insulin sensitivity and body composition were not measured as part of the rat study (8). We have also found that maternal exercise during pregnancy can increase insulin-stimulated glucose uptake in offspring skeletal muscle (mice and rats) and adipose tissue (mice) and decrease uptake in heart tissue (rats) compared to offspring born to sedentary dams (7, 8). These data suggest that maternal exercise in normal chow fed dams can improve metabolic outcomes in normal chow fed offspring.

Adverse health outcomes and birth weight are now thought to form a U-shaped relationship, with offspring born at low birth weights from under-nourished mothers and high birth weights from over-nourished mothers having the greatest risk for disease later in life (15). Recently, it was reported that rodent offspring from mothers who consumed a low-protein diet and exercised during pregnancy displayed improved growth and development and glucose homeostasis compared to offspring from mothers who consumed the low-protein diet and did not exercise (13). Data such as these suggest that maternal exercise can attenuate the effects of a maternal low-protein diet on metabolic outcomes in the offspring. With the growing incidence of women of reproductive age being classified as overweight or obese it is important to determine if this has an impact on the metabolic changes observed in offspring from mothers who exercise. Research utilizing obese pregnant rats has shown that maternal exercise helps improve offspring metabolism by increasing lean mass and decreasing fat mass percentage in male offspring (34). In addition, work in mice has shown that offspring from mothers who consume a high fat diet, a known factor that can lead to obesity, demonstrated hypermethylation of a metabolic master regulator, peroxisome proliferator-activated receptor γ covactivator-1α (PGC-1α) (21). While the offspring of mothers who consumed a high fat diet and did not exercise had hypermethylation of PGC-1α, those exercising mothers fed the same diet produced offspring that did not have hypermethylated PGC-1α (21). These data indicate that maternal exercise during pregnancy can prevent metabolic dysregulation in the offspring that is associated with maternal high fat diet consumption (21). Likewise, a recent study examined the impact of exercise during pregnancy on mouse offspring from dams that were either fed a standard chow or high fat diet and further divided into four experimental subgroups: exercise trained before gestation only, exercise trained before and during gestation, exercise trained only during gestation, or sedentary before and during gestation (30). The offspring from mothers who were sedentary and consumed a high fat diet had impaired glucose tolerance, increased serum insulin, and increased % body fat compared to the offspring from mothers who exercised before and during gestation (30). This indicates that maternal exercise before and during pregnancy can protect the offspring’s metabolic profile from the detrimental effects of a maternal high fat diet. Therefore, maternal exercise during pregnancy helps break the endocrine cycle that promotes obesity and, instead, improves the offspring’s metabolic profile.

Not all studies have shown beneficial effects of maternal exercise on offspring glucose homeostasis. A recent report examined the effects of exercise ancestry on metabolic phenotypes in multiple generations of mouse offspring (16). The F0 generation mothers exercised during pregnancy producing the F1 generation. Breeding of the F1 generation produced F2 offspring with either a sedentary or exercise ancestry (16). Glucose tolerance test results indicated that only the female F2 generation offspring were affected, with those offspring from an exercise ancestry displaying impaired glucose tolerance compared to their sedentary counterpart (16). While these data do not fall in line with previous reports (7, 8), it is important to note that the mouse offspring from Guth et al. (16) were only 8 weeks old at the time of testing, whereas the offspring from other studies were 31–32 weeks (7) or 10 months of age (8). This discrepancy in whether or not maternal exercise during pregnancy produces an improved glucose profile in the offspring points to a further need to examine offspring at varying ages and under additional metabolic stressors.

Most data suggest that maternal exercise during pregnancy positively impacts offspring metabolic health. Offspring from exercised mothers have improved insulin sensitivity, glucose handling, increased lean mass, and decreased fat mass compared to offspring from sedentary mothers (7, 8). Interestingly, the improved metabolic profile of the offspring is not limited to those born to normal weight, healthy mothers, but rather these improvements can also be seen in offspring from mothers who are undernourished or obese and exercise during pregnancy (13, 21, 30, 33). These data can be further used to support the claim that exercise during pregnancy is safe for both mothers and their offspring.

CARDIOVASCULAR DISEASE

Cardiovascular disease was once thought of as a disease of the aging, however, reports have shown that atherosclerotic lesions can occur in infants which suggests that the intrauterine environment may play a role in atherosclerosis disease risk (15). Therefore, we hypothesized that maternal exercise during pregnancy would result in decreased CVD risk in the offspring later in life. To this effect, recent research supports the notion that the prenatal environment can impact human fetal cardiovascular health (23, 24, 25). Data have shown that at 36 weeks of gestation, maternal exercise during pregnancy lowers fetal heart rate (HR) and increases HR variability (HRV) (23). These results indicate a maturation of the central nervous system, specifically the autonomic nervous system (ANS) and brainstem, in response to maternal exercise during pregnancy. This is important because, abnormalities in the autonomic nervous system have been reported to promote the progression of atherosclerosis (20). The results from these initial studies suggested a protective effect of exercise during pregnancy against CVD development in the offspring. In the past, reports on the impact of maternal exercise during pregnancy listed that fetal hypoxia could occur due to the decreased blood flow to the placenta during the time course of exercise (29). However, the results of May et al. (23) suggest this is not the case, as fetal hypoxia would lead to lower HRV. In addition, there is evidence for a dose-response relationship between maternal exercise and fetal cardiac ANS control, with women who engage in higher intensity exercise having fetuses with lower resting HRs and higher HRV (25). Regular moderate to vigorous intensity exercise is known to lower HR and improve cardiovascular well-being in adults. Therefore, these data suggest that exercise during pregnancy may be the earliest intervention to improve offspring cardiovascular health (23).

In response to the above data indicating that maternal exercise during pregnancy can positively impact cardiac ANS control in offspring, our lab has used various animal models to examine the impact of exercise during pregnancy on offspring arterial function and atherosclerosis risk (2, 3, 4, 27). Our first report on the impact of maternal exercise on swine offspring vascular health revealed phenotypic changes to the offspring's vasculature (27). At birth, female offspring from treadmill exercise-trained mothers had significantly greater thoracic aorta endothelial cell function, as assessed through in-vitro wire myography and cumulative doses of bradykinin, when compared to the male and female offspring from sedentary gilts (27). We speculated that these improvements in offspring vascular function might be linked to increases in fetal HR and subsequent increases in blood flow through the thoracic aorta, which could have increased the availability of nitric oxide (NO). It is well documented that increases in exercise blood flow cause frictional changes across the endothelium that increase NO bioavailability through the upregulation of endothelial nitric oxide synthase (eNOS) (22). These increases in fetal blood flow may be linked to reported increases in fetal HR during maternal exercise (10). The fact that the endothelium-dependent relaxation response was abolished when an NO inhibitor (L-NAME) was administered suggests that the increased relaxation response to bradykinin in the female exercise offspring was likely a result of increased NO bioavailability (27). These data indicate that maternal exercise during pregnancy may have an atheroprotective effect on the offspring at birth.

While our first report indicated improvements in offspring vascular function in response to maternal exercise during pregnancy, we recently revealed that maternal exercise during pregnancy decreased endothelium-independent vascular function in adult swine offspring (2). This study utilized offspring from the same mothers and *in vitro* methodology as the Newcomer et al. (27) study but examined offspring's femoral artery at ages 3, 5, and 9 months. The differences in vascular smooth muscle function between the exercise and sedentary offspring decreased with advancing age (2). Differential expression of genes and proteins associated with vascular tone between the exercise and sedentary offspring could explain the observed functional phenotype (2). To date, this is the first report to demonstrate that maternal exercise during pregnancy can induce vascular programming in adult offspring. However, it is important to note that the observed decreases in vascular smooth muscle function could potentially be interpreted as a negative phenotype for the offspring.

The disconnect between our labs observed increase in endothelium-dependent vasorelaxation function at 48 hours after birth in the swine exercise offspring to decreased endothelium-independent vasorelaxation function at 3, 5, and 9 months warranted further examination. Our previous studies were limited by the fact that we used healthy offspring who did not develop atherosclerosis. In addition, it was unclear if the vascular function differences seen in the thoracic and femoral arteries from our previous studies would be observed in the coronary arteries. Therefore, we recently examined the impact of maternal exercise during pregnancy on vascular function in swine offspring fed an atherogenic high fat diet (4). We followed the same maternal exercise and *in vitro* vascular function procedures as our previous studies (2, 27) and examined the left anterior descending (LAD) and femoral arteries of 4 and 8 months old high fat fed swine offspring. Contrary to our previous reports, there were no significant differences in offspring coronary or femoral artery endothelium-dependent or -independent vascular function at 4 or 8 months of age.

The majority of research conducted by our laboratory to examine the impact of maternal exercise during pregnancy on offspring vascular health has used a swine model due to the similarities of the swine cardiovascular system to a human (2, 4, 27). While swine may be an ideal model for studying cardiovascular disease, fetal programming work has commonly used rodents due to placental similarities to humans, short gestation lengths, and their ability to generate large sample sizes (3). Therefore, we examined the impact of maternal voluntary wheel running during pregnancy on rodent offspring vascular function at 4- and 8-months of age (3). Similar to our last study using a swine model, we found no differences in endothelium-independent or –dependent relaxation between offspring from exercise and sedentary mothers. One can speculate that the differences observed between our rat and pig studies may be influenced by dissimilarities in species and experimental design. Specifically, the two models have dramatically different types of placentation, gestational lengths, and different states of offspring maturity at birth. Furthermore, the lack of differences in vascular function between the offspring from exercise and sedentary mothers in the rodent study could also be a result of the exercise intensity. In our previous study using swine, the mothers were forced to exercise on a treadmill 5 days a week at an intensity of 65–85% of maximal heart rate (2, 27). In the rodent study, the exercise dams had free access to a running wheel throughout gestation. Since the exercise protocol was voluntary, it is possible that the dams did not meet the exercise intensity required for vascular programming.

While the existing data has some variation, the majority of the literature supports the notion that exercise during pregnancy positively influences or does not impact offspring cardiovascular health. Mothers who exercise during pregnancy have produced offspring with lower resting HR, higher HRV, and improved vascular health. In line with previous areas of research this work mostly supports the view that exercise during pregnancy is beneficial for both mother and offspring.

CANCER

Physical activity in adolescents and adults plays a pivotal role in the prevention of chronic diseases, including cancer (14). While there has been an increasing body of literature examining maternal exercise during pregnancy and risk for chronic diseases in the offspring in recent years, only one study to date has explored the risk of cancer development. Maternal high-methyl diet consumption during pregnancy has been shown to suppress mammary carcinogenesis in female rat offspring (9). This evidence indicates a link between maternal behaviors and reduced mammary cancer risk in the offspring. Data such as these led us to investigate the impact that maternal exercise during pregnancy had on offspring mammary cancer development. We hypothesized that maternal exercise during pregnancy would decrease the risk of breast cancer in offspring. Female rats were divided into two experimental groups during pregnancy: sedentary and voluntary wheel exercise (6). After weaning, female offspring from both sedentary and exercise mothers were fed a high fat diet. All of the pups were injected with the carcinogen N-methyl-N-nitrosourea at 6 weeks of age. Mammary tumor development in all pups was monitored for 15 weeks. These data revealed that offspring from mothers who exercise during pregnancy have a mammary tumor incidence of 42.9% compared to 100% incidence in sedentary offspring (6). Therefore, these preliminary data suggest that maternal exercise can reduce offspring mammary cancer risk (6). Camarillo et al. hypothesized that maternal metabolism or factors released into the maternal circulation in response to exercise could lead to programming changes in the offspring, however, there is currently no data to support this claim (6). Future work should seek to elucidate the mechanisms by which protective effects of maternal exercise during pregnancy on offspring breast cancer risk occurs.

FUTURE DIRECTIONS

The area of research examining maternal exercise during pregnancy and offspring chronic disease progression is growing. While the work that has been completed to date has answered a number of questions there are still areas that need to be explored. Specifically, there are many unknowns about how the type, timing, intensity, and dose (i.e., amount) of maternal exercise during pregnancy may impact offspring chronic disease risk later in life. Additionally, future work should explore mechanisms by which any changes in the offspring are occurring in response to maternal exercise during pregnancy.

The majority of research to date examining the impact of maternal exercise during pregnancy on long-term health outcomes in offspring has utilized some form of aerobic exercise (i.e., human aerobic trained, rodent treadmill or wheel running, or swine treadmill exercise). Future work needs to utilize different modes of exercise to determine whether the metabolic, cancer, and cardiovascular disease risk in the offspring are changed based on varying types of exercise. A number of studies exploring the impact of maternal resistance training during pregnancy have been published in recent years; however, they have focused on maternal and/or perinatal outcomes (28, 35). Therefore, randomized control trial studies examining the impact of maternal resistance training on chronic disease outcomes in the offspring would be an important addition to the literature. Additionally, the research to date has not utilized a specific intensity or amount of exercise during pregnancy when examining offspring chronic disease risk outcomes (Table 1). For instance, some studies utilize animal models that allow dams to exercise voluntarily on a running wheel, whereas others force dams to exercise, thus controlling the amount, duration, and intensity of exercise. Future research should continue to explore the impact of exercise modality, intensity, and dose of exercise during pregnancy on offspring chronic disease risk.

Timing of exercise during pregnancy also needs to be considered in future fetal programming studies. The published work to date has not exposed pregnant mothers to exercise for the same duration during pregnancy. While some studies have only allowed mothers to exercise during the finite duration of gestation, other studies have allowed exercise before, during, after pregnancy. For example, recent work examining the impact of exercise during pregnancy on offspring metabolic health utilized an experimental design where pregnant dams exercised during different time points of pregnancy (30). The data indicated that there were differences in metabolic health outcomes between offspring from the various experimental maternal exercise groups. Therefore, future work should explore the impact of the timing of exercise during pregnancy on offspring chronic disease risk.

As stated previously, the use of animal models is common in fetal programming research. The data from studies utilizing animal models have provided an important foundation for the field. However, future work needs to focus on experimental designs that can determine if the offspring health changes that have been identified in animal models are translated to humans. Particularly, researchers should explore the mechanisms by which these phenotypes occur in the offspring in response to maternal exercise during pregnancy. Researchers have predicted that these phenotypes are attributed to epigenetic alterations but there has only been one study demonstrating that maternal exercise during pregnancy causes DNA methylation or histone modifications in the offspring (18). Furthermore, if these phenotypes are produced by epigenetic alterations, studies should explore what specifically about maternal exercise causes these changes.

CONCLUSIONS

In general, research examining the impact of maternal exercise during pregnancy on offspring chronic disease risk has been positive. While there have been a few reports of detrimental health outcomes in the offspring in response to maternal exercise during pregnancy, the vast majority has shown improvements in offspring metabolic, cancer, and cardiovascular health and disease risk. Thus, overall the current body of work supports the recommendations for exercise during pregnancy set by the ACOG and DHHS. Health care providers can utilize these data to educate pregnant mothers on the benefits of exercise during pregnancy for their offspring and encourage them to incorporate exercise into their prenatal care.

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Summary

This manuscript explores the novel hypothesis that maternal exercise during pregnancy can protect against chronic disease susceptibility in offspring.

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Figure 1.

Graphic depicting the known offspring phenotypes in response to maternal exercise during pregnancy, as well as the unknown factors of maternal exercise that may impact offspring heath outcome. Photo credit to gettyimages.com.

Table 1

Summary of species and exercise protocol and duration for articles examining the impact of exercise during pregnancy on offspring health outcomes.

Reference	Species	Sex	Exercise Protocol	Exercise Duration[a]
			Metabolic Disorders	
Carter et al., 2012	ICR Mice	M/F	Wheel Running, available 24 h/day, 7 d/week	Gestation and Lactation
Carter et al., 2013	Sprague Dawley Rats	F	Wheel Running, available 24 h/day, 7 d/week	Gestation and lactation
Fidalgo et al., 2013	Wistar Rats	M	Treadmill, 20–50 min/day, 5 d/week	Gestation
Guth et al., 2013	C57BL/6 Mice	M/F	[d] Wheel Running, available 24 h/day, 7 d/week	Gestation
Laker et al., 2014	C57BL/6 Mice	F	Wheel Running, available 24 h/day, 7 d/week	Gestation
Stanford et al., 2014	C57BL/6 Mice	M	Wheel Running, available 24 h/day, 7 d/week	Before or Gestation
Vega et al., 2013	Wistar Rats	M/F	Wheel Running, 15 min/day, 7 d/week	Before and Gestation
			Cardiovascular Disease	
Bahls et al., 2014	Duroc × Landrace Crossbred Swine	M/F	Treadmill, 20–45 min/day, 5 d/week	Gestation
Blaize et al., 2015	Crossbred Swine	M/F	Treadmill, 20–45 min/day, 5 d/week	Gestation
Blaize et al., 2015	Sprague Dawley Rat	M/F	Voluntary Wheel, available 24 h/day, 7d/week	Gestation
May et al., 2010	Human	–	[b] Aerobic exercise, ≥ 30 min/day, 3 d/week	Before and Gestation
May et al., 2012	Human	–	[c] Report of leisure time activity	Before and Gestation
May et al., 2014	Human	–	[c] Report of leisure time activity	Gestation
Newcomer et al., 2011	Duroc × Landrace Crossbred Swine	M/F	Treadmill, 20–45 min/day, 5 d/week	Gestation
			Cancer	
Camarillo et al., 2014	Sprague Dawley Rat	F	Voluntary Wheel, available 24 h/day, 7 d/week	Gestation

[a] Exercise duration is the reviewers interpretation of the time period for the mothers exercise during pregnancy that was used for analysis; exercise is listed as before pregnancy (before), during (gestation), and/or after pregnancy (lactation)

[b] Ran, performed aerobics, skied cross-country

[c] General physical activity report of time spent engaging in aerobic, anaerobic, or lifestyle activities

[d] Voluntary wheel exercise was only in the F0 generation mother