

# Exercise in Pregnancy: Weighing Up the Long-Term Impact on the Next Generation

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HOPKINS, S.A. and W.S. CUTFIELD. Exercise in pregnancy: weighing up the long-term impact on the next generation. *Exerc. Sport Sci. Rev.*, Vol. 39, No. 3, pp. 120–127, 2011. *There is now a large body of evidence demonstrating the influence of the in utero environment on postnatal growth. Regular aerobic exercise during pregnancy elicits maternal and fetal adaptations that seem specific to the period of gestation in which training is initiated and maintained. This review considers the evidence for both positive and negative long-term health outcomes for offspring.* **Key Words:** fetal growth, postnatal growth, obesity, large for gestational age, small for gestational age

## INTRODUCTION

The prenatal period now is recognized as a unique physiological window in which maternal and fetal adaptations can have major consequences for the long-term health and well-being of offspring. The effects of exercise during pregnancy have been studied extensively. However, a dearth of well-controlled randomized studies has led to conflicting evidence as to the impact of exercise on fetal growth. Furthermore, there is a lack of consensus regarding the potential long-term risks or benefits for the offspring of exercising mothers. We recently have reported a modest reduction in offspring birth size in response to regular nonweight-bearing aerobic exercise in the second half of pregnancy (21). This was accompanied by lower cord blood insulin-like growth factor I (IGF-I) and IGF-II in exercise offspring, suggesting that maternal exercise elicited adaptations in nutrient partitioning to the fetus, leading to decreased endocrine stimulation of fetal growth. We hypothesize that regular exercise during pregnancy elicits maternal and fetal adaptations, and that these adaptations have the potential for both positive and negative long-term outcomes for offspring. Based on current evidence, these adaptations seem to be dependent on the gestational period in which exercise training is initiated and maintained. In view of current observations, the potential exists for a link

between maternal exercise and the developmental origins of obesity, such that a small reduction in birth weight in the upper normal range or in large for gestational age (LGA) offspring may reduce their overall risk for childhood obesity. In contrast, a significant reduction in offspring size across the entire birth weight range could increase the incidence of small for gestational age (SGA) infants, leading to long-term metabolic sequelae that previously have been defined by our group (18,20) and an increased risk of later adult diseases, including coronary heart disease and type 2 diabetes mellitus. This review will present the current evidence for an exercise-associated impact on fetal growth, explore the potential mechanisms by which this occurs, and evaluate the current evidence for positive and negative long-term effects for offspring.

## THE EFFECT OF EXERCISE TRAINING ON FETAL GROWTH

Despite extensive literature on the relationship between maternal physical activity and pregnancy outcomes, the evidence for a consistent and meaningful impact of regular aerobic exercise on fetal growth is lacking. Studies examining the effects of maternal exercise on fetal growth primarily have focused on offspring birth weight. There have been few randomized controlled trials, and those in existence generally have involved a small number of predominantly lean, physically active participants. Furthermore, most of the studies have provided minimal quantification of exercise performance and have given only limited consideration to factors confounding the relationship between exercise and fetal growth (25). For example, many studies have provided little or no information on nutritional status and caloric intake during pregnancy

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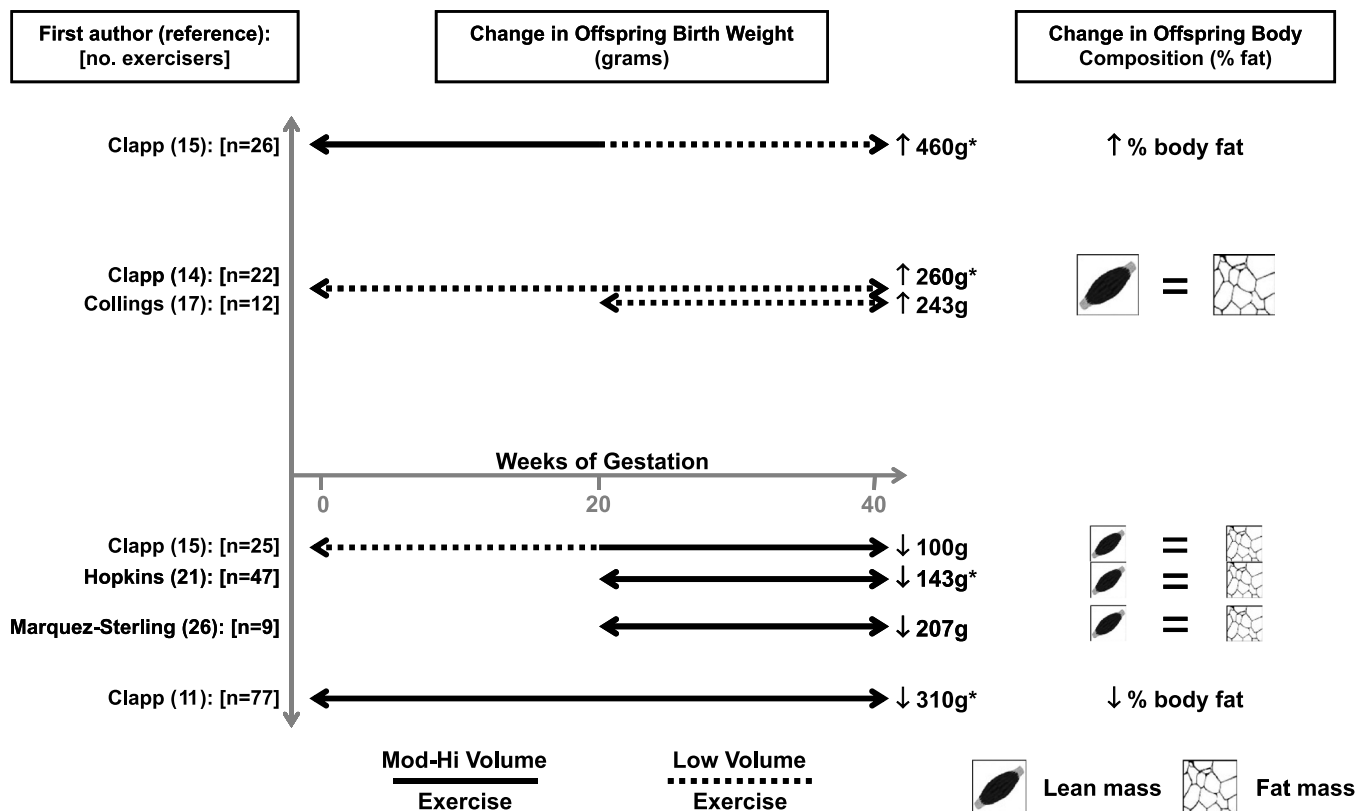
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nor have they controlled for gestational age at birth, socioeconomic status, or environmental factors likely to have confounding effects on offspring size. This has been an important limitation in the current literature because recent evidence suggests that maternal diet, and in particular dietary carbohydrate composition, has a large moderating effect on the exercise-induced change in offspring birth size and may provide one explanation for the large variability demonstrated in the previous literature (reviewed in (10)). As a result of these methodological limitations, randomized controlled studies examining the effects of regular aerobic exercise during pregnancy have reported contrasting effects on offspring birth weights (14,15,17,21,26). There also is substantial evidence that different patterns of exercise during pregnancy have diverse effects on the relationship between exercise and pregnancy outcomes (15) (Fig. 1). This has led to the suggestion that existing data are not sufficient to “infer important risks or benefits for the mother or infant” (25).

The most comprehensive examinations of antenatal exercise on pregnancy outcomes have been carried out by Clapp and colleagues in predominantly lean, physically active women. In one of the earliest cohort studies, well-conditioned recreational athletes continuing a program of vigorous weight-bearing aerobic exercise into late gestation led to a 310-g

reduction in birth weight compared with the offspring of matched controls (11). The reduction in birth weight in these neonates was caused entirely by a reduction in fat mass, with no difference in lean mass between groups. In contrast, Clapp *et al.* (14) reported that, in previously sedentary women, beginning a program of moderate weight-bearing exercise in early pregnancy was associated with a 260-g increase in birth weight corrected for gestational age, gender, and race. The increase in birth weight was caused by a proportional increase in lean body mass and fat mass. The differential effects seen in sedentary compared with previously active women may be caused by the ability of physically fit women to maintain a more intense exercise regimen during pregnancy. Alternatively, they may relate to long-term effects of an active lifestyle on body composition because maternal adiposity is linked strongly to offspring birth weight (7). In support of this hypothesis, two recent articles based on large (40,000–80,000 women) population-based cohorts have suggested that the independent effect of exercise in pregnancy on birth weight is minimal and may be explained largely by the confounding effects of maternal body composition (19,24).

For a number of pregnant women, particularly if they are overweight, nonweight-bearing exercise may be more appropriate to minimize joint and musculoskeletal stress, especially



**Figure 1.** Depiction of timing- and volume-dependent effects of aerobic exercise training on fetal growth, as assessed by offspring birth weight and body composition. A lower volume of regular exercise or a reduction in volume in the second half of pregnancy (*dotted lines*) seems to stimulate fetal growth, leading to increased birth weight compared with the offspring of control women who do not exercise in pregnancy. Increased birth size has been associated with a balanced increase in fat and lean mass (14) or a preferential increase in fat mass (increased percent fat) (15). It is likely that these outcomes occur through adaptations in placental structure and transport capacity in response to exercise in early gestation. In contrast, maintaining a vigorous weight-bearing exercise program (*solid lines*) for the duration of pregnancy leads to reduced birth weight and neonatal fat mass (decreased percent fat) (11). Regular moderate or vigorous exercise in the second half of pregnancy consistently has been associated with decreased birth weight, with no effect on body composition (no change in percent fat) (15,21,26). \*Significant change in offspring birth weight ( $P < 0.05$ ).

in late gestation. An effect of nonweight-bearing exercise (such as cycling or swimming) on birth size has not been documented clearly (17). However, studies using cycling protocols have suffered from small sample sizes, suggesting a lack of statistical power to detect potential effects, or have prescribed only a light volume of exercise (either short duration or low intensity). We recently have sought to clarify these issues by examining the impact of a home-based nonweight-bearing exercise programs on maternal and neonatal outcomes in a larger randomized controlled study (21). Healthy nulliparous women with a variety of prepregnancy exercise levels and a range of body mass index (BMI) in midpregnancy ( $18\text{--}32\text{ kg}\cdot\text{m}^{-2}$ ) were randomized to home-based exercise or a control group who were asked to maintain their normal daily activities. Exercisers took part in an individually prescribed stationary cycling program from gestation at 20 wk to term and were asked to maintain five sessions of 40 min moderate-intensity (65% of predicted aerobic capacity or  $\text{VO}_{2\text{max}}$ ) exercise per week, in line with The American College of Obstetricians and Gynecologists (ACOG) recommendations for exercise in pregnancy (3). Exercise training was associated with lower IGF-I and IGF-II concentrations in cord venous blood and a significant reduction in offspring birth weight. However, exercise had no effect on offspring body composition, with a proportional decrease in both lean and fat mass. The results of this study suggest that a predominantly nonweight-bearing exercise program may be sufficient to elicit adaptations leading to alterations in fetal growth.

## **EXERCISE TRAINING HAS TIME-SPECIFIC EFFECTS ON FETAL GROWTH**

The time-specific effects of exercise during pregnancy have been illustrated elegantly by Clapp and colleagues (15). A prospective randomized study was designed to examine the effects of vigorous weight-bearing aerobic exercise in physically fit women when the volume of exercise was altered midpregnancy (15). Women who performed a high volume of exercise in early pregnancy and then cut back their exercise in late pregnancy delivered offspring who significantly were heavier (mean increase in birth weight of 460 g vs moderate group;  $P < 0.001$ ) and taller at birth. The increase in birth weight was caused by a balanced increase in fat and lean tissue, resulting in a significantly higher percentage of body fat (12.1% vs 7.9%;  $P < 0.001$  vs moderate group). The latter finding is logical, considering that fetal body fat is laid down primarily in the last third of gestation. In contrast, the offspring of women who increased from a low volume of exercise in early pregnancy to a high volume in the second half of gestation were on average 100 g lighter than the offspring of a comparison group who maintained a moderate volume of exercise for the entire duration of pregnancy ( $P$  value not significant). When compared with the offspring of the moderate-volume group, these neonates had a similar percentage of fat mass, suggesting a symmetrical decrease in body size. Although they were not compared with a nonexercise control group, these outcomes are similar to those reported in our study in women who began a structured exercise program at 20 wk gestation. These findings strongly suggest that fetoplacental adaptations are dependent on the period of gestation in which

exercise training is initiated and maintained, as well as the intensity or volume of exercise performed.

## **MECHANISMS INVOLVED IN TIME-SPECIFIC EFFECTS OF EXERCISE ON FETAL GROWTH**

### **Effects of Exercise Training on Maternal Insulin Sensitivity and Glucose Metabolism**

It has been proposed that the impact of exercise on fetal growth involves an effect on maternal insulin sensitivity, a major determinant of fetal nutrient supply. However, we recently have reported that 15 wk of moderate-intensity cycling training had no impact on changes in insulin sensitivity in late pregnancy (21). This suggests that within a normal healthy pregnancy, maternal insulin sensitivity is regulated persistently to achieve optimal fetal growth and is not sensitive to the chronic training adaptations previously described in nonpregnant individuals. Further studies are required to confirm whether exercise training impacts insulin sensitivity in overweight and obese mothers, who likely are to be more insulin resistant before pregnancy and in late gestation.

In the absence of a persistent effect on maternal insulin sensitivity, transient changes in glucose regulation in response to regular bouts of aerobic exercise may drive the fluctuations in nutrient supply responsible for the reduction in offspring birth size observed in our study. Previous studies have demonstrated a reduction in glucose and insulin for a sustained period after an exercise session, particularly in late pregnancy (12). Furthermore, a single bout of moderate-intensity cycling exercise in late gestation has been shown to increase insulin sensitivity for at least 30 min after exercise (35). Therefore, intermittent reductions in maternal glucose levels after exercise may contribute to subtle adaptations within the placenta, leading to reduced nutrient transfer to the fetus.

### **Effects of Exercise Training on Placental Development**

The availability and rate of delivery of oxygen and nutrients via the placenta are major regulators of fetoplacental growth. Placental trophoblasts seem to be able to sense substrate and oxygen availability and respond by modulating the vascular and tissue growth within the placenta and by altering the local production of multiple growth-regulatory peptides (8,23). Regular aerobic exercise has been shown to influence the volume of functional placental tissue with or without an overall increase in placental mass, as well as indices of placental function (14,15,22). It is likely that acute exercise produces intermittent fluctuations in substrate and oxygen delivery, consistent with a redistribution of maternal cardiac output toward the exercising muscle and skin. These recurrent acute stimuli seem to elicit a different pattern of growth compared with chronic depressions in oxygen delivery such as those seen with maternal anemia or exposure to high altitudes (8); however, further studies are required to confirm if this hypothesis is correct. The insulin-like growth factors (IGF) and their binding proteins are thought to be the dominant peptides regulating tissue growth in the fetal compartment. Direct correlations have been observed with both placental mass and parameters of offspring size at birth (birth weight and neonatal fat mass) (8,21). Furthermore, the IGFs, together with tumor

necrosis factor- $\alpha$  (TNF- $\alpha$ ) and leptin, have been shown to regulate placental nutrient transporters, thereby affecting substrate delivery to the fetus (23).

Similar to the effect of maternal exercise on offspring birth size, there seem to be time-specific effects on placental development and growth. Placental growth and morphometrics with altered volumes of maternal exercise were assessed in the 2002 randomized study of Clapp *et al.* (15). Midtrimester placental growth rates significantly were greater in those women who took part in a moderate- or high-volume exercise program during early pregnancy (Mod group and Hi-Lo group) in comparison with a group who reduced their exercise volumes to a low level during early pregnancy (Lo-Hi). These observations suggest that aerobic exercise initiated in early pregnancy has volume-dependent stimulatory effects on placental growth. At term, however, placental volumes remained greater only in the Hi-Lo group, suggesting that vigorous exercise in late gestation has suppressive effects on further placental growth. We did not examine changes in placental structure and function in our randomized trial. However, based on the evidence presented, it likely seems that the reduction in birth weight reported in our exercise offspring may be caused by the timing of the intervention period. Our exercise program began at 20 wk gestation and only reached maximal exercise volume at 28 wk. It is possible that initiation of a moderate to vigorous exercise program after the first 20 wk would lead to more distinct fetoplacental adaptations as the stimulus occurred without the buffer of early placental adaptations.

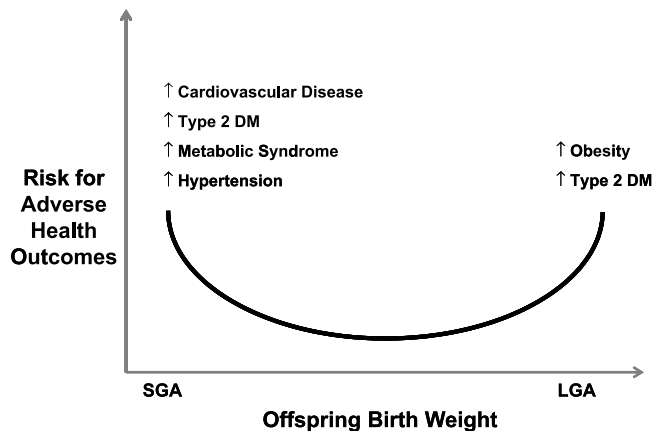
## WHAT ARE THE LONG-TERM HEALTH CONSEQUENCES FOR THE OFFSPRING?

### The Long-Term Impact of the *In Utero* Environment

The role of the *in utero* environment and early infancy period on the risk of adult disease is established clearly now. The long-term effects of fetal growth on adult health are thought to be the consequences of programming. A programming effect describes a stimulus occurring at a critical sensitive period of early life that has permanent effects on the growth and metabolism of the organism. These stimuli may lead to alterations in birth size and metabolic changes, such as reduced insulin sensitivity and increased susceptibility to disease in later life (6). Birth weight, although a crude estimation of overall fetal growth, is the most easily measured outcome of the impact of the *in utero* environment on fetal growth and has been used to investigate associations with later postnatal health outcomes. The earliest observations linked size at birth with type 2 diabetes mellitus, the metabolic syndrome, hypertension, cerebrovascular disease, and coronary heart disease. These observations subsequently have been replicated by many other research groups across different ethnicities and nations (reviewed in (6)).

#### *The consequences of being small at birth*

Epidemiological observations have linked small size or relative thinness at birth with increased rates of cardiovascular disease, type 2 diabetes mellitus, and the metabolic syndrome in adult life (6) (Fig. 2). Metabolic abnormalities also have been demonstrated in asymptomatic children and young adults



**Figure 2.** Hypothesized U-shaped relationship between offspring birth weight, the most commonly reported marker of the effects of the *in utero* environment, and risk for postnatal adverse health outcomes. Based on early observations and the hypothesis of Barker (6), with more recent evidence of the association between being born large for gestational age (LGA) and obesity in postnatal life (28,34). SGA indicates small for gestational age; DM, diabetes mellitus.

born SGA (<10th percentile for birth weight corrected for gender and gestational age) (18,20). In a study from our group, short prepubertal children born SGA demonstrated markedly reduced insulin sensitivity and increased insulin response to glucose infusion in comparison with short appropriate for gestational age (AGA) children (20). These metabolic changes were not the result of differences in height or body composition and were not complicated by catch-up growth in children born SGA.

In addition to the long-term consequences of being born SGA, there is evidence for a continuous relationship between birth weight and future health risk. That is, increased risk for adverse postnatal health outcomes does not occur only at extreme birth weights, but also through a reduction in size within the normal range of birth weights (6). However, many of these studies examine cohorts born almost a century ago and are based on comparisons of relative risk in birth weight brackets with increments of 1 lb (454 g) or 500 g. Therefore, it is likely that a reduction in mean birth weight of 500 g or more may confer increased risk of adverse postnatal health outcomes. However, our current knowledge does not allow conclusions to be made with regard to relative risk conferred by a smaller reduction in mean birth weight within the normal range.

#### *The consequences of being large at birth*

A major focus of past research has been on growth restriction and prematurity; however, there is now increasing evidence that the effect of the *in utero* environment on the development of obesity and risk factors for adult disease is U shaped (28,30) (Fig. 2). That is, children born at both the lower and upper ends of the birth weight spectrum are at risk of obesity and, subsequently, a range of adult diseases in later life. These studies have suggested an alternative developmental pathway for adult disease based on a link between large size at birth and later obesity (28). The prevalence of large birth weight (>4.5 kg) and LGA (>90th percentile for birth weight corrected for gender and gestational age) babies continues to increase (4,32). Consequently, the implications

of being born at the upper end of the birth weight spectrum and the potential role that *in utero* events may have in the obesity epidemic have become important considerations for ongoing research.

Infants weighing greater than 4500 g at birth have an elevated risk of infant mortality and birth trauma (31). Adverse consequences persist into later life because being born LGA may predispose to overweight and obesity in childhood and adolescence (28,34) as well as subsequent comorbidities, such as type 2 diabetes mellitus and cardiovascular disease (28). LGA babies have increased fat mass at birth compared with AGA infants and already display signs of metabolic dysregulation, including increased lipolysis and reduced insulin sensitivity, shortly after delivery (2). Recent evidence also suggests that females born LGA reach their child-bearing years with an increased risk of themselves delivering an LGA infant, independent of adult BMI, suggesting a cycle that may further contribute to the rising obesity epidemic (1).

### **Current Evidence for Health Risks in Offspring of Exercisers**

#### *The potential health risks of exercise during pregnancy*

Sustained moderate- or high-intensity aerobic exercise initiates a redistribution of blood flow to working muscles and results in an increase in core temperature and reduced maternal substrate levels. Consequently, there have been considerable concerns that exercise of this nature during pregnancy could result in transient fetal hypoxia and lead to fetal growth restriction. Documenting the safety of physical activity during pregnancy has proven a challenge for investigators largely because of issues of study design, small sample sizes, and inconsistent assessments of physical activity. Recent investigations have suggested that regular exercise does not result in an increase in early pregnancy loss, stillbirth, or neonatal death (reviewed in (25)); however, data on these outcomes are limited to small numbers in intervention studies. Maternal physiological adaptations to pregnancy may reduce markedly the impact of potentially harmful exercise effects (8). Furthermore, exercise training during pregnancy may enhance pregnancy adaptations in a manner beneficial for fetoplacental growth (8). Our observation that physiological changes in maternal insulin sensitivity in healthy pregnancy are regulated strongly to achieve optimal fetal growth and are not sensitive to modest increases in energy expenditure through exercise supports the safety of maternal exercise for fetal well-being.

#### *The potential postnatal health risks to offspring of exercisers*

The primary concerns for the offspring of exercisers involve the hypothesized long-term risks associated with a reduction in size at birth. In our cohort, participation in a nonweight-bearing exercise program in the second half of pregnancy was associated with an average birth weight reduction of 143 g compared with offspring of women who did not take part in a structured exercise program (21). In contrast, prospective observational cohort studies have found larger (300–600 g) reductions in birth weight in the offspring of heavy exercisers compared with the offspring of nonexercising mothers (11,13). Clapp and Capeless (11) reported that continuing a program of vigorous weight-bearing exercise for the duration

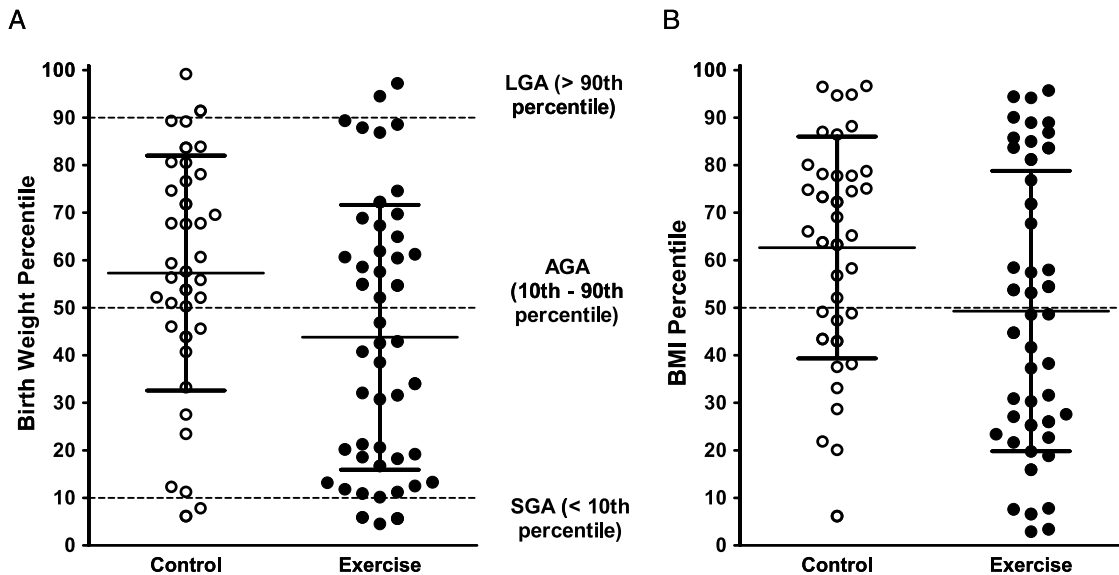
of pregnancy led to a mean reduction in birth weight of 310 g compared with the offspring of closely matched women who reduced their physical activity levels from early gestation. A large proportion of these offspring were then reassessed at 5 yr of age. Exercise offspring remained lighter and leaner than controls, but there were no differences in height or head circumference, suggesting no evidence of adverse early postnatal health outcomes (9).

#### *The potential risks of “catch-up” growth in early childhood*

Childhood obesity and adult disease may be further programmed by rapid or “catch-up” growth in infancy. Epidemiological studies have suggested that associations between small size at birth and increased risk for some adult diseases are amplified or even depend on the development of obesity in adulthood (6). Adult BMI tends to track from around 6 yr of age; therefore, early postnatal growth rates may have a profound influence on risk factors for disease in later life. During this period, postnatal growth seems to be influenced by a desire to compensate for undesirable intrauterine conditions (either growth restraint or enhanced fetal growth) through compensatory “catch-up” or “catch-down” growth. Therefore, the potential long-term consequences of maternal exercise may not be caused by a reduction in fetal growth *per se*, but increased risk of postnatal catch-up growth, predisposing to adult obesity and subsequent comorbidities. No catch-up growth was observed at 5 yr of age in offspring of women who continued vigorous exercise in pregnancy (9). We await postnatal growth assessment in our cohort to determine whether this is an ongoing consideration when evaluating the long-term consequences of exercise in pregnancy.

### **Current Evidence for Postnatal Health Benefits in the Offspring of Exercisers**

The concept that intrauterine overnutrition may affect lifelong risk of obesity now is well supported (28). Increased availability of maternal fuels, in particular glucose and amino acids, leads to the stimulation of fetal insulin and, in turn, IGF-I, which assumes a primary role in the stimulation of fetal growth (8). In fact, even a moderate increase in maternal glucose levels has been shown to increase the incidence of macrosomia (33). We previously have speculated that a modest reduction in birth weight in the offspring of women who exercise during their pregnancy may lead to a long-term reduction in the risk of obesity in childhood (21). In our cohort of healthy first-time mothers, we observed a significant reduction in standardized mean birth weight and BMI in exercise offspring compared with controls. We further speculated that this alteration was, in part, due to a normalization, rather than a reduction, of nutrient supply for fetal growth, given that our control offspring displayed newborn size parameters greater than the mean for our reference population (Fig. 3). As our reference cohort included children who were born from 1980 to 1989, our observations are consistent with the general trend of increasing mean birth weight in recent generations (4), a phenomenon that may be contributing to the ever-increasing global obesity epidemic. Studies reporting a positive linear relationship between size at birth and either obesity risk or BMI in childhood suggest that a reduction in mean birth weight may confer positive health benefits. However, the magnitude of a



**Figure 3.** Distribution of (A) birth weight and (B) body mass index (BMI) percentile in the offspring of women randomized to a control group ( $n = 37$ ) or a home-based stationary cycling program from 20 wk gestation to term (exercise;  $n = 47$ ). Mean birth weight and BMI percentile were reduced significantly at birth ( $P < 0.05$ ). However, there were no significant differences in the proportion of children born small for gestational age (SGA), appropriate for gestational age (AGA), or large for gestational age (LGA). Percentiles calculated from population standards, corrected for gender and gestational age at delivery. Data from Hopkins *et al.* (21).

birth weight reduction required for a meaningful long-term outcome yet has to be determined and should be a focus of continued research. Ongoing postnatal growth assessments continue in our cohort to assess whether the reduction in size at birth translates into a leaner phenotype in childhood and reduced risk for obesity and subsequent disease.

There are very little long-term longitudinal data on postnatal growth in the offspring of women who exercised during their pregnancy. Two studies have come out of Clapp's laboratory, examining morphometric and developmental outcomes in different cohorts at ages 1 and 5 yr (9,16). In their initial follow-up study, the offspring of exercisers were significantly lighter and leaner than control offspring at birth (9). When reassessed at age 5 yr, exercise offspring had the same morphometric profile (reduced weight and fat mass) but remained within normal population limits (45th–60th percentile for age- and gender-adjusted body weight). Exercise offspring also demonstrated slightly advanced neurodevelopmental scores compared with their control counterparts. However, these neurodevelopmental outcomes should be interpreted with caution because they did not control for maternal IQ.

A subsequent study by the same authors then was designed to follow-up a second cohort of exercise offspring earlier in life (16). The offspring of women who began a less intensive weight-bearing exercise program in early pregnancy were again lighter and leaner at birth. At age 1 yr, exercise offspring were not different in height, weight, or body composition compared with the offspring of control mothers. The results of these two studies suggest that maternal exercise may continue to promote health benefits into childhood but, like neonatal outcomes, these may depend on the volume of exercise performed in pregnancy.

The impact of maternal exercise on postnatal growth in the offspring of women who are overweight or obese during pregnancy has not been investigated. It is our hypothesis that

the potential health benefits of maternal exercise for offspring could be greater in overweight and obese mothers, who have an increased risk of delivering a large baby (32). There is a paucity of robust intervention studies examining the effects of regular aerobic exercise on fetal growth in overweight and obese mothers. The emphasis of exercise studies in these populations has been placed on maternal outcomes in response to combined dietary and exercise interventions, particularly on the prevention of excessive weight gain or other pregnancy complications such as gestational diabetes mellitus (GDM) and preeclampsia. Studies that have documented success in preventing excessive weight gain have not shown accompanying effects on mean offspring birth weight (5,27). However, a small reduction in the risk of delivering a macrosomic infant (birth weight  $>4000$  g) has been shown in overweight women taking part in a structured diet and exercise intervention (27). In a second study, obese GDM women who did not gain weight during their pregnancy delivered a lower proportion of macrosomic infants than those who gained weight, suggesting that a successful intervention to prevent excessive weight gain may help reduce the risk of fetal overgrowth in overweight and obese women (5).

## DOES EXERCISE AFFECT THE BIRTH WEIGHT DISTRIBUTION?

The long-term consequences for offspring of mothers who regularly exercise in pregnancy may depend on whether exercise results in a global reduction in birth weight or a shift in the birth weight distribution as a result of asymmetric effects at either or both extremes of the birth weight range. A shift in mean birth weight may be of less relevance if the primary concern is directed toward the two extremes of birth weight, where the risks of adverse outcomes for offspring are increased. Furthermore, a shift in mean birth weight could be caused by

exercise adaptations exerting more influence at one extreme and little or none at the other. In this situation, extrapolating effects on mean values to all parts of the distribution may be misleading. In contrast, if antenatal exercise affects the spread of the birth weight distribution, there may be no difference in mean birth weight but a possible increase or decrease in the proportion at both extremes (*i.e.*, SGA, LGA). Figure 3 shows the distribution of standardized birth weight and BMI obtained from offspring of women randomized to exercise or control for the second half of their pregnancy (21). These data are an example of a reduction in mean birth weight in exercise offspring without significant alterations in the birth weight distribution. However, this study was powered to detect an exercise effect on mean birth weight and may have had reduced statistical power to detect a change in birth weight distribution.

The prevention or reduction in the incidence of LGA may be the most meaningful outcome with regard to long-term health benefits because these infants are at the greatest risk for later obesity. In our cohort, exercise training in pregnancy did not reduce the risk of delivering an LGA baby. However, there were low rates of LGA infants in both exercise and control groups, suggesting a generally low-risk population. In contrast, large population-based studies have suggested that women who continue regular physical activity during the second and third trimesters of pregnancy have a reduced risk of delivering LGA infants (24,29). To our knowledge, there have been no randomized controlled trials specifically evaluating the effect of regular exercise on excessive neonatal birth weight in overweight or obese pregnancy.

Similarly, an increase in the proportion of infants born SGA may be the most meaningful outcome with regard to potential long-term health risks for offspring because these infants also are at an increased risk of adverse long-term health consequences. In our cohort, the significant reduction in birth weight in offspring of exercisers was not associated with an increase in the incidence of SGA (Fig. 3). However, there are inconsistent reports in the literature with regard to altered risk of SGA in exercising women. An early observational cohort study reported an increased incidence of SGA in lean active women who continued vigorous weight-bearing exercise during pregnancy (13). In contrast, a recent report based on data from almost 80,000 women and newborns in the Danish National Birth Cohort indicated that exposure to any amount of exercise compared with no exercise in pregnancy was associated with a slightly decreased risk of SGA (adjusted hazards ratio, 0.88; 95% confidence interval, 0.83–0.93) (24). Taken together, these data clearly do not indicate an elevated risk for fetal growth restriction after regular moderate exercise in pregnancy.

In summary, there is gathering evidence that the effects of regular aerobic exercise in pregnancy on offspring birth weight preferentially may affect the upper end of the birth weight distribution. That is, exercise may reduce the risk of delivering an LGA infant without significantly increasing the risk of delivering an SGA baby. These effects may occur with or without a concomitant reduction in mean birth weight; therefore, it is important to consider both mean birth weight and LGA/SGA incidence when evaluating the neonatal outcomes of exercise training in pregnancy.

## WEIGHING UP THE LONG-TERM HEALTH CONSEQUENCES FOR OFFSPRING

Physical activity is unlikely to be uniformly beneficial or detrimental for all populations. In this review, we have interpreted evidence from previous and ongoing studies to make the following conclusions and recommendations:

1. The most favorable long-term health outcomes for offspring likely are to result from a modest reduction in mean birth weight or a reduction in the incidence of LGA, without a significant increase in the incidence of SGA.
2. The potential health benefits of maternal exercise for offspring may be the greatest in overweight and obese mothers, who have an increased risk of delivering a large baby with an increased risk of obesity in childhood. Future studies are required to determine the effects of regular aerobic exercise in pregnancy on excessive fetal growth. It is likely that for long-term health benefits to occur in this population, prevention or restriction of excessive gestation weight gain is required. Therefore, dietary intervention may be an essential part of a lifestyle modification program during pregnancy in overweight and obese women.
3. There have been few studies examining postnatal growth outcomes in exercise offspring. The two studies to date provide no evidence of adverse outcomes in childhood. There have been no studies examining postnatal growth in the offspring of overweight or obese women who engage in regular aerobic exercise in pregnancy. Postnatal follow-up, particularly in the first 2 yr of life, should be included as an important part of ongoing research in this area.
4. To achieve a modest reduction in mean birth weight or a reduction in the incidence of LGA, the following recommendations can be made with respect to the design of future exercise programs in pregnancy:
  - a) Type of exercise. Weight-bearing aerobic exercise training largely has been favored in previous studies. Recent evidence suggests that nonweight-bearing exercise, which may be more tolerable for overweight and obese women, also may be sufficient to elicit fetoplacental adaptations. There currently is scant evidence on the efficacy and safety of resistance training in pregnancy. However, the addition of modified resistance training to aerobic exercise programs in pregnancy may have different or additional benefits for both mothers and offspring.
  - b) Timing of exercise training. Exercise in early pregnancy has stimulatory effects on placental growth and function. Although this may be a beneficial adaptation in physically active women, it may have the less desirable effect of promoting excess fetal growth in overweight and obese mothers. Initiating a moderate exercise program during gestation at 20 wk may be more beneficial in preventing excess fetal growth. Further studies in overweight and obese women are required to determine whether this hypothesis is correct.
  - c) Volume of exercise. If fetoplacental adaptations are caused by the repeated acute stimulus of each exercise

session, exercise should be frequent. Based on the current evidence, the ACOG recommendation of “30 min of moderate exercise on most, if not all, days of the week” seems safe and achievable for healthy pregnant women in the absence of medical or obstetric complications.

## CONCLUSIONS

There is now a large body of evidence demonstrating the influence of the *in utero* environment on growth trajectory in postnatal life. Regular aerobic exercise during pregnancy elicits maternal and fetal adaptations that have the potential for both positive and negative health outcomes for offspring. Based on current evidence, the effect of exercise on offspring size at birth may occur primarily at the upper end of the birth weight range. Therefore, engaging in regular exercise may be important particularly for overweight and obese women, whose infants have increased risk for later obesity. Future studies should continue to examine the effects of regular exercise in obese pregnancy to determine whether the risk of adverse postnatal health outcomes can be reduced in their offspring and provide a healthy start to life.

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