Among the many factors that contribute to childhood obesity (i.e. maternal smoking, nutrition/sugar intake, air pollution, endocrine disruptors, sleep disturbance, lack of breastfeeding), there are two powerful maternal determinants: high maternal body mass index (BMI) and exceeding the Institute of Medicine (IOM) gestational weight gain (GWG) guidelines [1]. Furthermore, pregnancy weight-related issues increase the likelihood of adverse cardiovascular risk factors in offspring [2,3]. Excessive GWG is directly linked to giving birth to a large-for-gestational-age (LGA) neonate [4], which is predictive of downstream obesity and chronic conditions, including Type 2 diabetes and cardiovascular disease [reviewed in 5,6]. Recently, high birth weight and parental overweight/obesity were associated with lower levels of both physical activity (PA) and cardiorespiratory fitness in adolescence [7], further supporting the need for prenatal strategies that optimize fetal growth for long-term health. To the surprise of many, excessive GWG in normal-weight women is associated with higher neonatal fat mass and less favorable body composition (i.e. greater percentage of body fat, less muscle mass) [8]. This dysregulation in body composition suggests that an energy surplus in utero acts independent of parental genetics with respect to predisposition for excess weight. In fact, according to recent systematic reviews and meta-analyses, excessive GWG increases the risk of childhood overweight/obesity by 30-40% [9,10], thereby propagating the intergenerational cycle of obesity and the proliferation of chronic disease [5]. This may be due to a host of sociopolitical and physiological factors that promote maternal resource storage, decrements in PA, a loss of metabolic control, and a partitioning of excess energy reserves to the fetus [5,11].

Physical activity remains one of nature’s best medicines for prevention and management of chronic disease [12]. However, it is seldom recommended in pregnancy [13]. Furthermore, population PA levels are at an all-time low [14] and reach a nadir during the prenatal period. The reasons for these observations are not well-established, but likely involve a patient-provider knowledge translation discrepancy [15]. Despite the historical dogma and the ensuing clinical recommendations, a pregnant
Commentary

In addition to healthy eating, regular PA is a critical mediator of weight gain and a vital component of weight maintenance strategies at all ages. Activity levels during pregnancy are also a predictor of maternal obesity and excessive GWG [17]. Given the importance of PA, the American College of Obstetrics and Gynecology, the Society of Obstetrics and Gynecology Canada/Canadian Society for Exercise Physiology, and the International Olympic Committee have issued specific guidelines that encourage all pregnant women to engage in routine PA in the absence of contraindications [18-20]. Regular moderate intensity PA during pregnancy has consistently been shown to reduce the incidence of GDM [21-24] and pre-eclampsia [23, 25-29], two pregnancy-related complications implicated in poor neonatal outcomes and downstream child health. Systematic reviews and meta-analyses looking exclusively at PA interventions during pregnancy have shown success in restricting GWG (-0.36 kg, 95% CI: -0.64 to -0.09 kg [30]; -0.61 kg, 95% CI: -1.17, -0.06 [31]; -0.91 kg, 95% CI: -1.76, -0.06 [32]) but few studies have been designed to examine the effects on longer-term child growth or body composition [33-35]. Data that are available from population-level surveillance, randomized controlled trials, and prospective birth cohorts suggest that regular, moderate amounts of PA can protect against birth weight extremes (i.e., small- and large-for-gestational age), and increase the likelihood of delivering an infant whose birth weight is appropriate for their gestational age [28-36]. Research demonstrating a reduction in fetal growth without an increased incidence of small-for-gestational-age infants suggests that sensible prenatal exercise may help normalize nutrient supply to the fetus, thus helping regulate fetal growth [37]. For instance, first and second trimester GWG are directly associated with cord blood hormone levels (e.g., insulin, c-peptide, insulin-like growth factor-I (IGF-I) and IGF-II) at delivery. These growth-promoting hormones are vital for glycemic control and somatic growth and, when in excess, have been implicated in obesity predisposition and metabolic dysregulation [38]. On the other hand, a reduction in growth-promoting peptides has been noted in offspring cord blood of maternal exercisers [39]. This suggests that an active pregnancy may alter nutrient partitioning to the fetus without any demonstrated effect on maternal insulin sensitivity or changes in GWG. Studies by Clapp et al. report that the reduction in birth weight of exercising mothers was entirely accounted for by a reduction of neonatal fat mass with no changes in lean mass compared to the offspring of matched controls [40]. Most striking is the finding that 5 years later the offspring of exercising mothers remained lighter and leaner than their comparators with no difference in other anthropometrics or health outcomes [41]. More recent work has shown that women with a higher total energy expenditure during pregnancy delivered babies with less fat mass, similar lean mass, and thus an improved body composition [42]. A recently published randomized controlled trial concluded that exercise may attenuate adverse pregnancy outcomes including infant size at birth (e.g., macrosomia) when complicated by overweight or obesity [43]. Taken together, PA during pregnancy helps optimize development by preventing overgrowth and inhibiting fetal growth restriction.

### Table 1. Sample exercise prescription for pregnant women without contraindications (adapted from [13,19]).

<table>
<thead>
<tr>
<th>Program Frequency</th>
<th>Previously Sedentary</th>
<th>Active</th>
</tr>
</thead>
<tbody>
<tr>
<td></td>
<td>3 d/wk</td>
<td>4 d/wk</td>
</tr>
<tr>
<td>Program Intensity\†</td>
<td>Low-moderate</td>
<td>Moderate-vigorous</td>
</tr>
<tr>
<td>Program Duration</td>
<td>15 min, gradually increase to 30 min sessions</td>
<td>30 min per session</td>
</tr>
<tr>
<td>Program Type\‡</td>
<td>Low impact aerobic (swim, walk, cycle)</td>
<td>Low impact aerobic (swim, walk, cycle)</td>
</tr>
<tr>
<td></td>
<td>Resistance/strength training</td>
<td>Resistance/strength training</td>
</tr>
</tbody>
</table>

* Brief warm-up and cool-down should be incorporated with each bout of activity
\† The “talk test” may also confirm that women are not over-exerting
\‡ Avoid exercise in the supine position after approximately 16 weeks’ gestation
The optimization of infant birth weight in women who engage in regular PA is thought to result from an increased functional capacity of the placenta to appropriately shuttle nutrients across the maternal-placental-fetal interface. This involves mechanisms that increase placental surface area, improve blood flow, and enhance perfusion [44,45]. Evidence from animal and human studies suggests that fetal growth rate is matched with maternal nutrient availability via altered expression of placental transporters, receptors, and signaling pathways involved in nutrient sensing and delivery. This acts to restrict growth when maternal nutrition is limited and accelerates growth in nutrient excess conditions [46-49]. Even though the placenta is a pivotal regulatory organ [50], few groups have explored placental function and cellular mechanisms in pregnancies exposed to maternal exercise [51]. New data suggest that meeting PA guidelines (30 minutes of aerobic activity, 3-4 days/week) during the second trimester is associated with altered expression of genes involved in fatty acid and amino acid transport across the placenta [49,52], which may contribute to altered nutrient delivery to the fetus and subsequent changes in fetal body composition.

Although considerable animal research has illustrated that maternal diet alters developmental pathways and offspring body composition through epigenetic changes in metabolic control genes [52], there is a burgeoning body of literature investigating the effect of maternal PA on these processes. Controlled experiments of maternal PA in animal models has shown beneficial impact on many offspring variables; hippocampal neurons and angiogenesis [53], insulin sensitivity [54,55] and metabolism [56], expression of molecules known to attenuate placental dysfunction [57], high fat diet induced changes in metabolic regulator genes [58], as well as hippocampal neurogenesis, learning, and memory [59]. Thus, it is not unrealistic to presume that PA behaviors, affecting maternal metabolism and the metabolic milieu, could affect fetal body composition and downstream health. As such, PA should be considered alongside dietary factors as keystones to childhood obesity prevention [60].

In summary, it is advisable that trainees and physicians be aware of the tremendous physiological benefits of active living during pregnancy. Every little bit counts and the physiological benefits precede phenotypical change. This is an important talking point to address with patients. With respect to physically active pregnancy, some is better than none, and more is better than some in the absence of contraindications. Patients should listen to their bodies and be open with their physicians about the activities they are involved in and the symptoms that present. For the latter to occur, doctors must engage patients in non-judgmental dialogue and provide encouragement to support patients to live the healthiest life that they can enjoy, while maintaining balance and well-being. After all, small changes during pregnancy have the potential to improve public health on a population level and minimize intergenerational disease risk.

REFERENCES


