

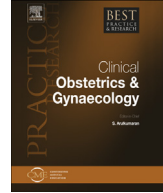


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# The role of exercise in reducing the risks of gestational diabetes mellitus in obese women



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The global obesity epidemic continues unabated, now rapidly expanding to developing countries. Multiple comorbidities and premature mortality are associated with obesity, most frequently diabetes. The associated financial and economical burden is escalating as well. The sedentary lifestyle adopted by many pregnant women because of traditional practices and the current recommendation for gestational weight gain are contributing factors to the obesity and diabetes epidemic.

Physical inactivity is recognized as an independent risk factor for obesity insulin resistance and type 2 diabetes; the physiological and hormonal changes associated with pregnancy magnify this risk. Conversely, evidence and accumulated experience indicate that antenatal lifestyle interventions that include physical activity and judicious dieting could improve the pregnancy outcome and reduce the risk of gestational diabetes and is effective as an adjunctive therapy for diabetes in pregnancy. All major professional organizations, among them American Congress of Obstetricians and Gynecologists (ACOG), American Diabetes Association (ADA), Royal College of Obstetricians and Gynaecologists (RCOG), and Society of Obstetricians and Gynaecologists of Canada (SOGC), recommend lifestyle interventions that include diet and exercise to prevent or manage gestational diabetes or diabetes mellitus.

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## Introduction

In 2008, it was estimated that 508 million people worldwide were obese and another 1.46 billion people overweight and many more currently [2]. The associated comorbidities, particularly diabetes, and consequences are staggering and the financial burden is of historic proportions. Globally, 366 million are affected by diabetes; it is expected that there will be 552 million diabetics by 2030 [3]. For many women, gestational diabetes is the first manifestation of diabetes.

Several large trials conducted in nonpregnant subjects in China, Finland and United States have proven that adopting a healthy lifestyle that includes weight reduction and physical activity can prevent, manage, or reverse diabetes [4–6]. Recommendations for the prevention of type 2 diabetes in nonpregnant women include pharmacotherapy, surgery, diet, aerobic exercise, and, most recently, also resistance training [7]. For pregnant women, the only management option available is lifestyle modification. The most effective interventions have combined judicious weight gain and physical activity.

### *Pathophysiology*

Normal pregnancy has been characterized as a diabetogenic event due to hormonal changes, and the progressive insulin resistance begins near mid-pregnancy and progresses through the third trimester to levels that approximate the insulin resistance of patients with type 2 diabetes [8]. Insulin resistance develops at the level of skeletal muscles; thus, exercise is a logical intervention that can counteract this activity. However, caloric restriction also plays an important role in improving insulin sensitivity.

In overweight and obese patients, insulin resistance is further increased in pregnancy. Obese women are at an increased risk of gestational diabetes mellitus (GDM) (odds ratio (OR) 2.6; 95% confidence interval (CI) 2.1–3.4). The increased fat deposition leads to an increase in adiponectin and leptin. Adiponectin regulates insulin sensitivity and glucose homeostasis. Additional weight gain leads to a further increase in adipokines and leptin, which increase the oxidative stress that contributes to the insulin resistance. Adiponectin regulates insulin sensitivity; low levels of adiponectin are associated with beta cell dysfunction. Muscles bind adiponectin which translocates glucose transporter 4 (GLUT4). GLUT4 is responsible for the transport of glucose into cells. Exercise enhances glucose uptake by GLUT4 translocation. Exercise regulates glycemia through two mechanisms:

- 1) Insulin-stimulated muscle glucose uptake
- 2) Insulin-independent glucose transport (GLUT4)

The production of leptin by the adipose tissue is also a major cause for insulin resistance. Exercise reduces the leptin levels.

Judicious caloric restriction also plays an important role in enhancing insulin sensitivity. It was demonstrated that obese subjects with impaired glucose tolerance or mild type 2 diabetes after 10 days of caloric restriction and a body weight reduction of only 3.5% will experience a 35% increase in glucose disposal rate during a hyperglycemic clamp, thus enhancing significantly the insulin sensitivity [9].

The glucose homeostasis is also altered by placental hormones such as human placental lactogen, progesterone, cytokines (tumor necrosis factor alpha (TNF $\alpha$ )), and others. It is also worth noting that fat deposits in the body are used as an energy source, and when large amounts of energy are required the duration and intensity of physical activity can make a difference. The utilization of fat involves several processes: lipolysis, mobilization, transportation, uptake, activation, translocation, and  $\beta$ -oxidation. The process is stimulated by multiple hormones, among them catecholamines. In our previous studies, we have demonstrated that in sedentary pregnant diabetic patients who exercised at 50–70% of their maximal aerobic capacity they elicit a proper catecholamine response, predominantly norepinephrine, sufficient to facilitate the skeletal muscle uptake of glucose [10].

Adopting the 2009 Institute of Medicine (IOM) recommendations [11] for additional gestational weight gain for overweight and obese pregnant women could further amplify the risk of gestational diabetes as insulin resistance worsens with additional weight gain.

The IOM guidelines for gestational weight gain are based on prepregnancy body mass index (BMI). In the IOM report, it is specified that basing the recommendation on prepregnancy BMI is an approach

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