

We are IntechOpen, the world's leading publisher of Open Access books Built by scientists, for scientists

5,600

Open access books available

137,000

International authors and editors

170M

Downloads

Our authors are among the

154

Countries delivered to

TOP 1%

most cited scientists

12.2%

Contributors from top 500 universities



WEB OF SCIENCE™

Selection of our books indexed in the Book Citation Index
in Web of Science™ Core Collection (BKCI)

Interested in publishing with us?
Contact book.department@intechopen.com

Numbers displayed above are based on latest data collected.
For more information visit www.intechopen.com



Using Resistance Training in Women with Gestational Diabetes Mellitus to Improve Glucose Regulation

Brittany R. Allman, Samantha McDonald, Linda May, Amber W. Kinsey and Elisabet Børsheim

Abstract

Gestational diabetes mellitus (GDM) poses a significant threat to the short- and long-term health of the mother and baby. Pharmacological treatments for GDM do not fully correct the underlying problem of the disease; however, non-pharmacological treatments such as exercise are increasingly recognized as foundational to glycemic management in other populations with disordered glucose regulation, such as non-gravid women with type II diabetes mellitus (T2DM). Much of the research regarding the impact of exercise on glycemic control in T2DM leverages aerobic training as the primary modality; yet research has demonstrated the effectiveness of resistance training on improving glycemic control in T2DM. This chapter will review the rationale for resistance training in the management of GDM using evidence from individuals with T2DM; then the chapter will review available studies on the effectiveness of resistance training on glucose control in women with GDM.

Keywords: physical activity, pregnancy, aerobic training, resistance training, strength training, insulin, glucose, insulin resistance, insulin sensitivity

1. Introduction

Gestational diabetes mellitus (GDM) is glucose intolerance diagnosed during pregnancy [1] and occurs in approximately 10% of all pregnancies [2]. The prevalence of GDM is increasing in the United States [3, 4] and once diagnosed, the odds of GDM in subsequent pregnancies [5, 6] and postpartum type II diabetes mellitus (T2DM) [7, 8] are significantly increased. GDM poses significant health threats to mothers and their offspring, including, but not limited to, placental dysfunction, preterm birth, neural tube defects, macrosomia [9, 10], and increased cardiometabolic disease risk (e.g., obesity, insulin resistance) later in life [11–14]. Consequently, the threat of declining, preventable health outcomes of future generations is imminent, prompting the need for cost-effective therapeutic strategies for the treatment of GDM. Exercise is an effective lifestyle intervention for GDM; however, the precise design of such interventions first requires an understanding of the metabolic changes that occur during pregnancy and the development of GDM.

2. Metabolic changes in pregnancy and the development of GDM

From conception to birth, the female human body undergoes several structural and physiological changes to optimize fetal growth and development; these changes related to normal gestation have been extensively reviewed by others [15]. In uncomplicated pregnancies, maternal metabolism adjusts to the nutrient and energy needs of the growing fetus. In the first half of pregnancy, the fetal nutrient and energy demand is rather low. Thus, maternal metabolism is in an anabolic state favoring nutrient storage, demonstrated by enhanced appetite and tissue-specific insulin sensitivity, specifically of adipose tissue (i.e., fat tissue), and consequently increases in stored triglycerides [15].

Conversely, from the mid-2nd trimester until birth, there is a rapid acceleration in fetal nutrient and energy demands paralleling the augmented growth and development, requiring another shift in maternal metabolism [15]. In this phase, maternal metabolism shifts from an anabolic state to a catabolic state characterized by marked increases in maternal insulin resistance and the shunting of maternal glucose to the fetus, which is the most critical energy substrate for optimal fetal growth and development [15]. Maternal insulin resistance primarily occurs within the skeletal muscle, resulting in progressive and substantial reductions (~55–75%) in maternal glucose uptake relative to pre-pregnancy [15]. Subsequently, meeting the energy demands of the mother requires a dramatic increase in lipolysis, specifically of the triglyceride stores deposited in early pregnancy [16, 17]. Paralleling the increase in maternal insulin resistance, maternal serum lipid concentrations increase by 200–300% compared to pre-pregnancy [16, 17]. The natural increases in maternal insulin resistance must occur or its absence leads to severe fetal growth restriction and permanent, lifelong adverse health outcomes.

The onset of maternal insulin resistance prompts the maternal pancreas to upregulate insulin production and secretion, promoting adequate, yet still reduced, maternal glucose uptake. This response maintains optimal fetal glucose supply, protecting it from an oversupply. However, a failed or insufficient pancreatic response and increased maternal glucose concentrations may lead to a persistent state of maternal hyperglycemia, yielding a continuous oversupply of glucose to the fetus. Consequently, the maternal pancreas either (1) continues to respond to the hyperglycemia via further increases in insulin production and secretion resulting in maternal hyperinsulinemia potentially worsening the progressing maternal insulin resistance and ensuing hyperglycemia via reduced insulin receptor sensitivity or (2) fails to produce and secrete a sufficient amount of maternal insulin, yielding worsened hyperglycemia, without hyperinsulinemia. These alterations in maternal metabolic responses can lead to the development and diagnosis of GDM.

Given the grave maternal and fetal health consequences of glucose intolerance and GDM, all pregnant women are screened for glucose intolerance or GDM in the mid-to-late 2nd trimester via glucose challenge tests by consuming a beverage containing a 50-g load of glucose. Following intake, maternal blood is drawn via venipuncture and serum glucose levels measured. If maternal fasting glucose levels exceed 95 mg dL^{-1} , or if glucose levels at 1-h post-dose exceed 180 mg dL^{-1} , the pregnant women 'fails' and subsequently undergoes a 3-h glucose oral glucose tolerance test (OGTT) to confirm a GDM diagnosis. To confirm a GDM diagnosis, maternal glucose levels must exceed two of the following three glucose thresholds: 180 mg dL^{-1} at 1 h, 155 mg dL^{-1} at 2 h, or 140 mg dL^{-1} at 3 h post OGTT [18]. A confirmed GDM diagnosis requires immediate treatment intervention.

3. Current treatment interventions for GDM

The first line of treatment for GDM includes medical nutrition therapy (e.g., complex carbohydrate-rich diabetic diet), capillary blood glucose monitoring, and recommendations of at least 150 min of aerobic exercise per week [18]. If clinicians render the behavioral strategies ineffective, pharmacological therapy (insulin, metformin, or glyburide) is prescribed [18]. Pharmacological therapy effectively manages maternal hyperglycemia via stimulation of peripheral glucose uptake by skeletal muscle and fat cells, and by inhibiting hepatic glucose production. While effective, pharmacological therapies fail to address the underlying mechanisms that cause insulin resistance in GDM, including a reduction in peripheral and hepatic insulin sensitivity, pancreatic β -cell failure or damage, and dysfunctional insulin action at the post-receptor level in skeletal muscle [19]. Furthermore, pharmacological therapy is associated with adverse health outcomes such as small-for-gestational-age offspring [20] and maternal vascular damage [21], and comes with a significant medical financial burden.

In contrast, exercise has been shown to improve peripheral (e.g., muscle) glucose tolerance through both insulin-dependent and insulin-independent mechanisms [22], and pancreatic β -cell function [23, 24] in T2DM populations. With this general understanding of the benefits of exercise for glucose management, several professional organizations such as the American College of Obstetrics and Gynecology [25], the American College of Sports Medicine [26], the American Diabetes Association [27] advocate for the use of prenatal exercise as an adjunctive therapy to improve glycemia in GDM.

4. Exercise and GDM

4.1 Exercise and aerobic exercise: definitions

Exercise training is defined as a structured, goal-oriented, progressive behavioral regimen, whereby individuals repeatedly perform bodily movements aimed to improve health, locomotion, ease of daily physical activities, sports performance etc. Two common types of exercise training are aerobic training and resistance training. Aerobic training involves performing exercises that rhythmically and continuously move large muscle groups for sustained periods of time such as walking, cycling, rowing, swimming, running etc. Aerobic training typically focuses on improving an individual's cardiorespiratory fitness.

4.2 Resistance training: definition

Resistance training is a form of exercise characterized by repetitive voluntary skeletal muscle contractions working against an external resistance (e.g., gravity during body weight exercises, free weights) and is designed to improve muscular fitness [28]. Resistance training programs typically focus on improving muscular strength. One form of resistance training, called strength training, typically involves higher loads (e.g., heavier weight), lower repetitions, more recovery time between sets, and isolates specific muscle groups (e.g., legs, back). For example, a person might perform a barbell squat at 75% of their maximal effort for three sets of 8 repetitions, with 2 min of rest between sets. Circuit training is a form of body conditioning involving full-body exercises performed in a series with minimal rest between each exercise. Although it is predominately a form a resistance training, circuit training often includes a combination of resistance training and moderate-to-high

intensity aerobic training. Circuit resistance training typically involves lighter loads or body weight, a higher number of repetitions (e.g., 10–15), and little to no rest periods. One example of CRT might be performing the following eight exercises for 10 repetitions each, as many times as possible in a given amount of time (e.g., 10 min), and taking breaks as needed: chest press, low row, squat, lunge, shoulder press, latissimus dorsi pull-down, biceps curl, and triceps extension.

4.3 Effectiveness of aerobic training in women with GDM

Growing evidence demonstrates that participating in aerobic training during pregnancy elicits profound positive effects on maternal glucose tolerance. Previous studies showed that exercising during the first 20 weeks of pregnancy significantly reduces (up to 50%) a pregnant woman's risk of developing GDM [29]. Moreover, studies have shown that prenatal aerobic exercise effectively manages maternal glucose levels and may replace pharmacological therapies in pregnant women diagnosed with GDM [29–31]. For these reasons, several worldwide private and governmental agencies endorse pregnant women engaging in prenatal aerobic exercise for the prevention and management of GDM [24–26], along with a plethora of other health-related benefits. Aerobic training is a promising modality to optimize maternal and offspring outcomes considering this type of exercise encompasses a wide range of activities (e.g., walking, cycling, and swimming).

4.4 Prevalence of resistance training and recommendations

Currently, it is unknown what percentage of pregnant women with GDM participate in resistance training. However, despite being the third most commonly reported activity during pregnancy, resistance training is performed in only 10% of pregnant women overall [32]. These statistics are slightly outdated, however, there have been no other more recent reports over the past several years. Nevertheless, resistance training has gained significant popularity among non-gravid women [33, 34], indicating that women, in general, are becoming more interested in the benefits gleaned from resistance training. However, the lack of resistance training participation while pregnant is likely driven by many factors. Misconceptions about resistance training during pregnancy, in particular, may be a major contributor. For example, anecdotally, common misconceptions include e.g., resistance training being dangerous for the mother and baby, core training causing separation of the abdominal muscles (diastasis recti), an increase in pregnancy pains when resistance training, you cannot perform resistance training during pregnancy if you have never resistance trained before, you cannot lay on your back during exercise after 16 weeks gestation, and others. Although many of these misconceptions are likely rooted in cultural ideologies, the lack of rigorous research regarding the impact of resistance training during pregnancy, especially GDM, is likely the reason that American governing bodies have just recently (year 2020) added resistance training guidelines for all pregnant women [25, 35], and have not yet added resistance training as part of the first line of glucose management upon GDM diagnoses [18, 27]. As a result, the breadth of exercise recommendations at the practice level (e.g., OB/GYNs) is limited. Thus, more research on resistance training in GDM populations is needed to inform the public and in turn impact the participation of pregnant women in resistance training.

4.5 Effectiveness of resistance training in T2DM

Despite a dearth of resistance training research in pregnant women, there is evidence demonstrating the effectiveness of resistance training in individuals with

T2DM, who have similar peripheral impairments in insulin resistance as GDM. For instance, both resistance training and aerobic training individually elicit similar improvements in glycemic control in T2DM in non-gravid adults [36–39], indicating that resistance training may be a novel approach to achieving the same outcome in GDM women. A meta-analysis of studies in GDM [38] determined that as long as the exercise training (either aerobic training or resistance training) is performed at a sufficient frequency (3–4 times per week), intensity (moderate to vigorous), and duration (20–30 min), similar glycemic outcomes will occur in response to aerobic training vs. resistance training. These findings confirm evidence demonstrating mechanical contraction of muscle, in general, is a potent physiological stimulator of skeletal muscle glucose uptake [40], and suggest that the type of exercise (e.g., resistance training or aerobic training) may not be as important given that bodily movement produces muscle contractions. However, glucose uptake into muscle is contraction-intensity dependent in both fast- and slow-twitch skeletal muscle fibers [40]. Thus, although any type of physical activity will increase glucose uptake due to its respective contractile nature, the magnitude of blood glucose uptake depends on the intensity with which the activity is performed.

Although aerobic exercises is often prescribed for glucose management in T2DM, sustaining continuous activity for 30–60 min at a time may be difficult for these individuals for a number of reasons (e.g., reduced aerobic capacity and exercise tolerance, orthopedic issues, excess weight [41, 42]). These barriers to aerobic exercise may encourage exercise participation at lower than recommended intensities or lead to exercise dropout. In general some exercise is better than none, however, there is a positive relationship between the intensity at which aerobic exercise is performed and glycemic control in T2DM [43]. Aerobic exercise may need to be performed at a higher intensity than is feasible for many adults with T2DM to sustain. Fortunately, resistance training may address aforementioned barriers associated with aerobic exercise as it can be performed with lower aerobic effort, intensity can be modified in a variety of ways (e.g., load, tempo, exercise progressions and regressions), and the extent to which activities are weight bearing can be adjusted (e.g., free weights vs. machines). Because these aspects are relevant to T2DM and pregnant women, resistance training may be an effective exercise option for GDM populations.

On a practical level, it may not be prudent to simply recommend an increase in physical activity (e.g., walk more throughout the day) in patients with glucose regulatory disorders, such as GDM. Nevertheless, if the exercise dose (frequency, intensity, and duration) is at or above recommended levels, the type of exercise may not be as important for glucose regulation in GDM. These findings are encouraging for both practitioners and pregnant women since it moves the focus of an exercise program to the preferences of the pregnant woman, allowing the program to be individually tailored. The ability to adjust exercise prescription to the needs and preferences of the individual will ultimately help increase adherence to an exercise program and lifestyle modification.

5. Mechanisms of the improvement in insulin sensitivity with resistance training in T2DM

The mechanisms by which resistance training may improve glycemia in GDM has not yet been elucidated in the literature. Therefore, this section will review the mechanisms of resistance training-induced improvements in glycemia in T2DM. The improvements in glycemia with resistance training can occur independent

of the addition of aerobic training into a resistance training program [44], and without changes in maximal oxygen uptake [45]. In other words, improved insulin sensitivity with resistance training can occur without improved aerobic capacity suggesting that resistance training alone may be a sufficient stimulus to improve glycemia independent of traditional aerobic exercise training recommendations for the management of glycemia. In fact, studies have reported that the impact of resistance training on insulin sensitivity and glucose control is greater than aerobic training [46, 47], or at a minimum, elicits the same glycemic effect [48], when matched for training units or time. Therefore, it may be that the higher intensity contractile nature of resistance training compared to aerobic training results in greater glucose uptake during exercise, and this physiological stimulus may supersede the benefit of improved aerobic capacity on glycemia.

There are a variety of reported mechanisms by which resistance training improves glucose regulation in T2DM. First, resistance training increases muscular glucose disposal and insulin sensitivity [49, 50], which can occur acutely after a singular resistance training session [51]. However, resistance training should be maintained as a part of a regular exercise routine because the effect of resistance training on glycemic control and insulin sensitivity is not sustained when resistance training is discontinued [52]. Second, although it may be assumed that hypertrophy is one of the mechanisms by which glucose control is achieved with chronic resistance training in T2DM, an increase in muscle mass, *per se*, may not be the direct catalyst of change [53]. Instead, an array of intrinsic metabolic changes within the muscle may be the driver of improvements in glucose control in T2DM. For instance, resistance training increases insulin receptor concentration [54] and enhances the activation of the insulin signaling cascade [55, 56]. Upon activation of insulin receptors by insulin, several intracellular cascades are stimulated, including glucose transporter type 4 (GLUT4) translocation that ultimately increases glucose uptake into the cell. GLUT4 permits facilitated diffusion of glucose into skeletal muscles, and therefore, a larger concentration of GLUT4 and faster movement of GLUT4 to the cell surface with resistance training will enhance glucose flux into the cell, and therefore better regulate blood glucose levels. Resistance training also directly increases the content and rate of GLUT4 translocation within the muscle cell [57]. Importantly, these changes occur independent of significant increases in muscle mass [58], and even after only one resistance training session or single set of exercises [51], suggesting that repeated mechanical muscular contractions, rather than muscle growth, may be the most important for glucose control in T2DM. These findings, however, should not discount the importance of muscle mass, because it is known that low relative muscle mass is related to an increased risk of developing T2DM [59]. However, these findings may be particularly important for pregnant women, considering that (1) there is a stigma around resistance training and becoming “bulky” in female populations, and (2) resistance training programs may not have to be built on high intensity regimens (i.e., it does not have to be straining) characteristic of muscle hypertrophy programs to achieve glycemic benefits. Considering there is a substantial body of evidence to suggest that resistance training is beneficial for glycemic control in T2DM, and the peripheral insulin resistance effects of T2DM and GDM are similar, it may be assumed that many of the mechanisms of change as a result of resistance training in GDM would be similar to T2DM. However, mechanistic data in women with GDM is not available in the current literature. Therefore, the next section will discuss available research on the effect of resistance training on several clinical outcomes related to glucose control. Future research describing the mechanisms by which these changes occur is needed.

6. The effect of resistance training on glucose regulation in GDM

6.1 Risk of GDM

It is important to determine the impact of resistance training during pregnancy on the risk of developing GDM to evaluate resistance training as preventative therapy, rather than solely for treatment upon diagnosis. However, the only reported study that assessed this relationship found that a moderate intensity resistance training intervention during pregnancy did not reduce the risk of developing GDM in sedentary, normal weight Spanish women after adjusting for maternal age and body weight pre-pregnancy [60]. Therefore, it may be that light-to-moderate intensity resistance training exercises cannot “override” the predisposition that women with higher BMIs (even though the ones in the study were normal weight) have for the risk of GDM. This study was limited because it assessed healthy women with normal BMIs, and not overweight or obese women who are known to have a significantly higher risk of developing GDM [61]. In addition, the resistance training protocol (3×/wk., 25–30 min per session at moderate intensity) included “toning and joint mobilization,” which consisted of isolation movements of small muscles or muscle groups using very light loads (3 kg barbells and 1–3 kg elastic resistance bands). The movements included shoulder shrugs and rotations, arm elevations, leg lateral elevations, and pelvic tilts and rocks. Women who are experienced weightlifters would consider this protocol to be more of a mobility and activation routine characteristic of a warm-up, rather than a workout routine that properly stresses the muscle. Depending on an individual’s experience with resistance training, the light-to-moderate intensity exercises described in the study may not provide a sufficient mechanical stimulus to evoke changes at the level of the muscle. The women in the study mentioned above were sedentary; therefore, they may have initially gleaned strength benefits from the program, but likely would have quickly plateaued. Even so, this particular study did not assess muscular strength gains as a result of the resistance training intervention. The goal of the study may not have been to use traditional resistance training with the goal of improving strength considering it was designed for toning and mobilization. Overall, more research is needed to determine if a resistance training program providing a sufficient stimulus reduces the risk of GDM in at-risk women, such as women with overweight and obesity or those with a history of GDM.

6.2 Insulin therapy

It may not be viable to use resistance training as a preventative therapy against the diagnosis of GDM in all women because there may be a low likelihood of starting a resistance training exercise routine prior to conception in women with no prior experience in resistance training. Therefore, determining how resistance training can attenuate the pharmacological requirement for the regulation of glucose in women with GDM upon diagnosis is important. Insulin therapy is the first line antihyperglycemic drug therapy recommended for treatment of GDM [62] when initial lifestyle changes (medical nutrition therapy, physical activity) are ineffective. One study demonstrated that fewer women in the resistance training group required insulin therapy compared to the control group [63]; while another study found no differences between resistance training-plus-diet vs. diet alone (standard diabetic diet) groups [64]. However, all women in the resistance training-plus-diet group were prescribed less insulin (diet: 0.48 ± 0.3 units/kg; resistance training-plus-diet: 0.22 ± 0.2 units/kg, $P < 0.05$) and commenced insulin therapy later after diagnosis (diet: 1.1 ± 0.8 weeks;

resistance training-plus-diet: 3.71 ± 3.1 weeks, $P < 0.05$) [64]. Furthermore, overweight women in the resistance training-plus-diet group had a significantly lower incidence of insulin therapy use [64]. Therefore, the effect of diet therapy on insulin use may be complemented by the addition of resistance training overall, and the metabolic effects of resistance training are likely to be greater in women with higher BMIs compared to women with healthy weight BMI. These findings are of no surprise considering it is likely that the diabetic diet consisting of less daily carbohydrates (40% of total energy intake) and the contractile nature of resistance training have a synergistic effect on the maintenance of blood glucose levels. Although both diet and exercise are the first line of treatment for GDM, this study was the only one to combine exercise and nutrition therapy. Therefore, more research that truly reflects the overall treatment strategies for women with GDM is required.

6.3 Fasting glucose and insulin concentrations

Being one of the most widely used clinical measures of glycemia, fasting glucose and insulin concentrations must be examined with a resistance training intervention during GDM. The American Diabetes Association recommends that fasting glucose concentrations during pregnancy should be $<95 \text{ mg dL}^{-1}$ [27]. After chronic resistance training in women with GDM, fasting glucose concentration tends to decrease more from pre- to post-intervention compared to aerobic training [63, 65–67]. However there are rarely differences between resistance training and aerobic training groups [63, 65–68], indicating that exercise in general (e.g., muscular contraction) may be the most important factor in the regulation of fasting glucose concentrations. Importantly, although the women in each of these studies were diagnosed with GDM, they had well-managed glucose levels represented by fasting glucose concentrations below recommended levels even before the exercise intervention. Thus, perhaps women with GDM with less control over circulating glucose concentrations may be more responsive to exercise training. In regard to fasting insulin concentrations, most work has demonstrated that there is no effect of resistance training [65, 69], however, one study showed a significant difference between resistance training and aerobic training groups whereby fasting insulin levels increased with resistance training and decreased with aerobic training [66]. Nevertheless, fasting insulin levels after the resistance training intervention ($10.22 \pm 2.76 \text{ mIU/mL}$) were still within normal limits ($<20 \text{ mIU/L}$ [70]). Therefore, it seems that there are minimal to no effects of resistance training on fasting insulin concentrations in GDM.

6.4 Markers of insulin resistance and β -cell function

A more significant indicator of the potential impact of resistance training on glucose regulation in GDM may be indirect measures of insulin resistance and pancreatic beta cell function. For example, measures such as the homeostatic model assessment (HOMA) of insulin resistance (HOMA-IR) and HOMA- β , respectively, use fasting insulin and glucose concentrations. The only reference values for HOMA-IR during pregnancy are in Mexican women (first trimester: <1.6 ; second trimester: <2.9 , third trimester: <2.6) [71], however, in general, the higher the HOMA-IR values, the more insulin resistant the individual. Changes in HOMA-IR tends to not differ between resistance training and aerobic training protocols in GDM [65, 69]; however, one study found there was a significant difference between resistance training, aerobic training, and control groups, with HOMA-IR decreasing to a greater extent in the aerobic training (-7.1%) compared to resistance training (-3.54%) groups. Nonetheless, HOMA-IR was reduced in both exercise groups and increased in the non-exercise control group ($+9.06\%$), indicating that, much

like fasting glucose concentrations, exercise in general (and not exercise type) may be the most important factor regulating indirect measures of insulin resistance in GDM. On the other hand, in the few studies using HOMA- β , an estimate of steady-state beta-cell function, no differences have been found between resistance training, aerobic training, and control groups [65, 66]. Therefore, more research is needed to assess the impact of resistance training on β -cell function.

The impact of resistance training in women with GDM on dynamic measures of glycemia, such as post-meal and post-exercise glucose concentrations, are promising. Chronic resistance training in women with GDM is associated with a greater percentage of weeks spent within a healthy target glucose range throughout the day (e.g. after an overnight fast, and after meals) compared to no exercise [63]. In addition, women with GDM using insulin therapy and exercise also spent more weeks within a healthy target glucose range throughout the day compared to women using insulin therapy that do not exercise [63]. Another study confirmed that after chronic resistance training in women with GDM, there is a greater reduction in postprandial glucose levels compared to aerobic training [68]; these findings indicating that resistance training may improve nutrient handling after a meal to a greater extent than aerobic training. Lastly, there are no differences in the reduction in blood glucose levels from baseline between an acute bout of resistance training vs. aerobic training [67], indicating that resistance training is a safe exercise modality to use in women with GDM, especially as it pertains to post-exercise glucose levels. Therefore, overall, resistance training in women with GDM improves glycemia throughout the day, and specifically after a meal, indicating that it may have therapeutic potential for women with GDM.

7. Conclusions

In conclusion, because of the potent effects of resistance training on glucose control in T2DM, it may be surmised that resistance training would also benefit women with GDM, who share similar impairments in peripheral insulin resistance. However, the studies of resistance training in women with GDM are minimal. Based on the work available, there seems to be initial promise for the use of resistance training in women with GDM to reduce the need for pharmacological insulin and improve glucose control throughout the day and after meals. Future work should assess the impact of a resistance training program on the risk of GDM in women with obesity; additionally, future research should provide more knowledge about potential effects of resistance training on clinical outcomes such as glucose and markers of insulin resistance. As more research becomes available, exercise guidelines can be properly tailored to pregnant women in a way that includes not only AT, but also resistance training.

Acknowledgements

The Arkansas Children's Research Institute and Arkansas Biosciences Institute Postgraduate Grant (B.R.A.).

Conflict of interest

B.R.A. has a podcast about exercise and health-related outcomes ("BENT by Knowledge") and is also the Senior Innovation Scientist for Breakout Lifestyle

Fitness, Little Rock, a gym emphasizing resistance training and health-related outcomes. The other authors report no conflicts of interest or competing interests.

Appendices and nomenclature

BMI	body mass index
GDM	gestational diabetes mellitus
GLUT4	glucose transporter protein type 4
HOMA- β	homeostatic model assessment of beta cell function
HOMA-IR	homeostatic model assessment of insulin resistance
T2DM	Type II diabetes mellitus

Author details

Brittany R. Allman^{1,2,3*}, Samantha McDonald⁴, Linda May^{5,6,7}, Amber W. Kinsey⁸
and Elisabet Børsheim^{1,2,3,9}

1 Arkansas Children's Nutrition Center, Little Rock, AR, USA

2 Arkansas Children's Research Institute, Little Rock, AR, USA

3 Department of Pediatrics, University of Arkansas for Medical Sciences, Little Rock, AR, USA

4 School of Kinesiology and Recreation, Illinois State University, Normal, IL, USA

5 Department of Obstetrics and Gynecology, East Carolina University (ECU), Greenville, NC, USA

6 Department of Kinesiology, ECU, Greenville, NC, USA

7 Department of Foundational Sciences and Research, ECU, Greenville, NC, USA

8 Department of Medicine, Division of Preventive Medicine, University of Alabama at Birmingham, Birmingham, AL, USA

9 Departments of Geriatrics, University of Arkansas for Medical Sciences, Little Rock, AR, USA

*Address all correspondence to: ballman@uams.edu

IntechOpen

© 2021 The Author(s). Licensee IntechOpen. This chapter is distributed under the terms of the Creative Commons Attribution License (<http://creativecommons.org/licenses/by/3.0>), which permits unrestricted use, distribution, and reproduction in any medium, provided the original work is properly cited. 

References

- [1] Proceedings of the 4th International Workshop-Conference on Gestational Diabetes Mellitus. Chicago, Illinois, USA. 14-16. March 1997. *Diabetes Care*. 1998;**21 Suppl 2**:B1-B167
- [2] DeSisto CL, Kim SY, Sharma AJ. Prevalence estimates of gestational diabetes mellitus in the United States, Pregnancy Risk Assessment Monitoring System (PRAMS), 2007-2010. *Preventing Chronic Disease*. 2014; **11**:E104. DOI: 10.5888/pcd11.130415
- [3] American Diabetes Association. Standards of medical care in diabetes—2015. *Diabetes Care*. 2015;**38**: S1-S2. DOI: 10.2337/dc15-S001
- [4] Practice Bulletin No. 137: Gestational diabetes mellitus. *Obstetrics Gynecology*. 2013;**122**:406-416. DOI: 10.1097/01.AOG.0000433006.09219.f1
- [5] Gaudier FL, Hauth JC, Poist M, Corbett D, Cliver SP. Recurrence of gestational diabetes mellitus. *Obstetrics and Gynecology*. 1992;**80**:755-758
- [6] Moses RG. The recurrence rate of gestational diabetes in subsequent pregnancies. *Diabetes Care*. 1996;**19**: 1348-1350. DOI: 10.2337/diacare.19.12.1348
- [7] O'Sullivan JB. Diabetes mellitus after GDM. *Diabetes*. 1991;**40**(Suppl 2): 131-135. DOI: 10.2337/diab.40.2.s131
- [8] Henry OA, Beischer NA. Long-term implications of gestational diabetes for the mother. *Baillière's Clinical Obstetrics and Gynaecology*. 1991;**5**:461-483. DOI: 10.1016/s0950-3552(05)80107-5
- [9] Leddy MA, Power ML, Schulkin J. The impact of maternal obesity on maternal and fetal health. *Reviews in Obstetrics and Gynecology*. 2008; **1**:170-178
- [10] Lynch CM, Sexton DJ, Hession M, Morrison JJ. Obesity and mode of delivery in primigravid and multigravid women. *American Journal of Perinatology*. 2008;**25**:163-167. DOI: 10.1055/s-2008-1061496
- [11] Freeman DJ. Effects of maternal obesity on fetal growth and body composition: Implications for programming and future health. *Seminars in Fetal and Neonatal Medicine*. 2010;**15**:113-118. DOI: 10.1016/j.siny.2009.09.001
- [12] Catalano PM, Presley L, Minium J, Hauguel-de MS. Fetuses of obese mothers develop insulin resistance in utero. *Diabetes Care*. 2009;**32**:1076-1080. DOI: 10.2337/dc08-2077
- [13] Whitaker RC. Predicting preschooler obesity at birth: The role of maternal obesity in early pregnancy. *Pediatrics*. 2004;**114**:e29-e36. DOI: 10.1542/peds.114.1.e29
- [14] Schmatz M, Madan J, Marino T, Davis J. Maternal obesity: The interplay between inflammation, mother and fetus. *Journal of Perinatology*. 2010;**30**: 441-446. DOI: 10.1038/jp.2009.182
- [15] Hytten F, Chamberlain G. *Clinical Physiology in Obstetrics*. Oxford, United Kingdom: Blackwell Scientific Publications; 1980
- [16] Knopp RH, Herrera E, Freinkel N. Carbohydrate metabolism in pregnancy. 8. Metabolism of adipose tissue isolated from fed and fasted pregnant rats during late gestation. *The Journal of Clinical Investigation*. 1970;**49**:1438-1446. DOI: 10.1172/JCI106361
- [17] Grimes SB, Wild R. Effect of pregnancy on lipid metabolism and lipoprotein levels [Online]. In: Feingold KR, Anawalt B, Boyce A, Chrousos G, de Herder WW,

- Dhatariya K, et al. editors. Endotext. MDText.com, Inc; 2021. Available from: <http://www.ncbi.nlm.nih.gov/books/NBK498654/>
- [18] Practice Bulletin No ACOG. 190: Gestational diabetes mellitus. *Obstetrics and Gynecology*. 2018;**131**:e49-e64. DOI: 10.1097/AOG.0000000000002501
- [19] Coustan DR. Pharmacological management of gestational diabetes: An overview. *Diabetes Care*. 2007;**30**: S206-S208. DOI: 10.2337/dc07-s217
- [20] Tarry-Adkins JL, Aiken CE, Ozanne SE. Neonatal, infant, and childhood growth following metformin versus insulin treatment for gestational diabetes: A systematic review and meta-analysis. *PLoS Medicine*. 2019;**16**: e1002848. DOI: 10.1371/journal.pmed.1002848
- [21] Meigs JB, Mittleman MA, Nathan DM, Tofler GH, Singer DE, Murphy-Sheehy PM, et al. Hyperinsulinemia, hyperglycemia, and impaired hemostasis: The Framingham Offspring Study. *JAMA*. 2000;**283**:221-228. DOI: 10.1001/jama.283.2.221
- [22] Romeres D, Schiavon M, Basu A, Cobelli C, Basu R, Dalla MC. Exercise effect on insulin-dependent and insulin-independent glucose utilization in healthy individuals and individuals with type 1 diabetes: A modeling study. *The American Journal of Physiology—Endocrinology and Metabolism*. 2021;**321**:E122-E129. DOI: 10.1152/ajpendo.00084.2021
- [23] Dela F, von Linstow ME, Mikines KJ, Galbo H. Physical training may enhance β -cell function in type 2 diabetes. *The American Journal of Physiology—Endocrinology and Metabolism*. 2004;**287**:E1024-E1031. DOI: 10.1152/ajpendo.00056.2004
- [24] Heiskanen MA, Motiani KK, Mari A, Saunavaara V, Eskelinen J-J, Virtanen KA, et al. Exercise training decreases pancreatic fat content and improves beta cell function regardless of baseline glucose tolerance: A randomised controlled trial. *Diabetologia*. 2018;**61**:1817-1828. DOI: 10.1007/s00125-018-4627-x
- [25] Physical Activity and Exercise During Pregnancy and the Postpartum Period [Online]. [date unknown]. Available from: <https://www.acog.org/en/clinical/clinical-guidance/committee-opinion/articles/2020/04/physical-activity-and-exercise-during-pregnancy-and-the-postpartum-period> [Accessed: 22 June 2021]
- [26] Pate R, Pratt M, Blair S, Macera C, Bouchard C, Buchner D, et al. A recommendation from The Centers for Disease Control and Prevention and the American College of Sports Medicine. *JAMA*. 1995;**273**:402-407. DOI: 10.1001/jama.273.5.402
- [27] Management of diabetes in pregnancy: Standards of medical care in diabetes—2019, American Diabetes Association. *Diabetes Care*. 2019;**42**: S165-S172. DOI: 10.2337/dc19-S014
- [28] Fleck S, Kraemer W. *Designing Resistance Training Programs*. 3rd ed. Champaign, IL: Human Kinetics; 2003
- [29] Oteng-Ntim E, Varma R, Croker H, Poston L, Doyle P. Lifestyle interventions for overweight and obese pregnant women to improve pregnancy outcome: Systematic review and meta-analysis. *BMC Medicine*. 2012;**10**:47. DOI: 10.1186/1741-7015-10-47
- [30] Davenport MH, Mottola MF, McManus R, Gratton R. A walking intervention improves capillary glucose control in women with gestational diabetes mellitus: A pilot study. *Applied Physiology, Nutrition, and Metabolism*. 2008;**33**:511-517. DOI: 10.1139/H08-018

- [31] Dipla K, Zafeiridis A, Mintziori G, Boutou AK, Goulis DG, Hackney AC. Exercise as a therapeutic intervention in gestational diabetes mellitus. *Endocrine*. 2021;**2**:65-78. DOI: 10.3390/endocrines2020007
- [32] White E, Pivarnik J, Pfeiffer K. Resistance training during pregnancy and perinatal outcomes. *Journal of Physical Activity and Health*. 2014;**11**:1141-1148. DOI: 10.1123/jpah.2012-0350
- [33] Hurley BF, Hanson ED, Sheaff AK. Strength training as a countermeasure to aging muscle and chronic disease. *Sports Medicine*. 2011;**41**:289-306. DOI: 10.2165/11585920-000000000-00000
- [34] Trends in Strength Training—United States, 1998-2004 [Online]. [date unknown]. Available from: <https://www.cdc.gov/mmwr/preview/mmwrhtml/mm5528a1.htm> [Accessed: 13 September 2021]
- [35] Bauer P. ACSM Information on Pregnancy Physical Activity [Online]. American College of Sports Medicine; 2020. Available from: https://www.acsm.org/docs/default-source/files-for-resource-library/pregnancy-physical-activity.pdf?sfvrsn=12a73853_4
- [36] Niemann MJ, Tucker LA, Bailey BW, Davidson LE. Strength training and insulin resistance: The mediating role of body composition. *Journal of Diabetes Research*. 2020;**2020**:e7694825. DOI: 10.1155/2020/7694825
- [37] Dunstan DW, Puddey IB, Beilin LJ, Burke V, Morton AR, Stanton KG. Effects of a short-term circuit weight training program on glycaemic control in NIDDM. *Diabetes Research and Clinical Practice*. 1998;**40**:53-61. DOI: 10.1016/s0168-8227(98)00027-8
- [38] Harrison AL, Shields N, Taylor NF, Frawley HC. Exercise improves glycaemic control in women diagnosed with gestational diabetes mellitus: A systematic review. *Journal of Physiotherapy*. 2016;**62**:188-196. DOI: 10.1016/j.jphys.2016.08.003
- [39] Wang C, Guelfi KJ, Yang H-X. Exercise and its role in gestational diabetes mellitus. *Chronic Diseases and Translational Medicine*. 2016;**2**:208-214. DOI: 10.1016/j.cdtm.2016.11.006
- [40] Jensen TE, Sylow L, Rose AJ, Madsen AB, Angin Y, Maarbjerg SJ, et al. Contraction-stimulated glucose transport in muscle is controlled by AMPK and mechanical stress but not sarcoplasmic reticulum Ca²⁺ release. *Molecular Metabolism*. 2014;**3**:742-753. DOI: 10.1016/j.molmet.2014.07.005
- [41] Nesti L, Pugliese NR, Sciuto P, Natali A. Type 2 diabetes and reduced exercise tolerance: A review of the literature through an integrated physiology approach. *Cardiovascular Diabetology*. 2020;**19**:134. DOI: 10.1186/s12933-020-01109-1
- [42] Gehling DJ, Lecka-Czernik B, Ebraheim NA. Orthopedic complications in diabetes. *Bone*. 2016;**82**:79-92. DOI: 10.1016/j.bone.2015.07.029
- [43] Liubaoerjijin Y, Terada T, Fletcher K, Boulé NG. Effect of aerobic exercise intensity on glycemic control in type 2 diabetes: A meta-analysis of head-to-head randomized trials. *Acta Diabetologica*. 2016;**53**:769-781. DOI: 10.1007/s00592-016-0870-0
- [44] Grøntved A, Rimm EB, Willett WC, Andersen LB, Hu FB. A prospective study of weight training and risk of type 2 diabetes in men. *Archives of Internal Medicine*. 2012;**172**:1306-1312. DOI: 10.1001/archinternmed.2012.3138
- [45] Ishii T, Yamakita T, Sato T, Tanaka S, Fujii S. Resistance training improves insulin sensitivity in NIDDM subjects without altering maximal oxygen

uptake. *Diabetes Care*. 1998;**21**:1353-1355. DOI: 10.2337/diacare.21.8.1353

[46] Cauza E, Hanusch-Enserer U, Strasser B, Ludvik B, Metz-Schimmerl S, Pacini G, et al. The relative benefits of endurance and strength training on the metabolic factors and muscle function of people with type 2 diabetes mellitus. *Archives of Physical Medicine and Rehabilitation*. 2005;**86**:1527-1533. DOI: 10.1016/j.apmr.2005.01.007

[47] Bacchi E, Negri C, Targher G, Faccioli N, Lanza M, Zoppini G, et al. Both resistance training and aerobic training reduce hepatic fat content in type 2 diabetic subjects with nonalcoholic fatty liver disease (the RAED2 Randomized Trial). *Hepatology*. 2013;**58**:1287-1295. DOI: 10.1002/hep.26393

[48] Bacchi E, Negri C, Zanolin ME, Milanese C, Faccioli N, Trombetta M, et al. Metabolic effects of aerobic training and resistance training in type 2 diabetic subjects: A randomized controlled trial (the RAED2 study). *Diabetes Care*. 2012;**35**:676-682. DOI: 10.2337/dc11-1655

[49] Umpierre D, Pa R, Ck K, Cb L, At Z, Mj A, et al. Physical activity advice only or structured exercise training and association with HbA1c levels in type 2 diabetes: A systematic review and meta-analysis. *JAMA*. 2011;**305**. DOI: 10.1001/jama.2011.576

[50] Strasser B, Siebert U, Schobersberger W. Resistance training in the treatment of the metabolic syndrome: A systematic review and meta-analysis of the effect of resistance training on metabolic clustering in patients with abnormal glucose metabolism. *Sports Medicine*. 2010;**40**:397-415. DOI:10.2165/11531380-000000000-00000

[51] Black LE, Swan PD, Alvar BA. Effects of intensity and volume on

insulin sensitivity during acute bouts of resistance training. *Journal of Strength and Conditioning Research*. 2010;**24**:1109-1116. DOI: 10.1519/JSC.0b013e3181cbab6d

[52] Andersen JL, Schjerling P, Andersen LL, Dela F. Resistance training and insulin action in humans: Effects of de-training. *The Journal of Physiology*. 2003;**551**:1049-1058. DOI: 10.1113/jphysiol.2003.043554

[53] Cauza E, Strehblow C, Metz-Schimmerl S, Strasser B, Hanusch-Enserer U, Kostner K, et al. Effects of progressive strength training on muscle mass in type 2 diabetes mellitus patients determined by computed tomography. *Wiener Medizinische Wochenschrift (1946)*. 2009;**159**:141-147. DOI: 10.1007/s10354-009-0641-4

[54] Strasser B, Pesta D. Resistance training for diabetes prevention and therapy: Experimental findings and molecular mechanisms. *BioMed Research International*. 2013; **2013**:805217. DOI: 10.1155/2013/805217

[55] Castaneda C, Layne JE, Munoz-Orians L, Gordon PL, Walsmith J, Foldvari M, et al. A randomized controlled trial of resistance exercise training to improve glycemic control in older adults with type 2 diabetes. *Diabetes Care*. 2002;**25**:2335-2341. DOI: 10.2337/diacare.25.12.2335

[56] Yaspelkis BB. Resistance training improves insulin signaling and action in skeletal muscle. *Exercise and Sport Sciences Reviews*. 2006;**34**:42-46. DOI: 10.1097/00003677-200601000-00009

[57] Holten MK, Zacho M, Gaster M, Juel C, Wojtaszewski JFP, Dela F. Strength training increases insulin-mediated glucose uptake, GLUT4 content, and insulin signaling in skeletal muscle in patients with type 2

diabetes. *Diabetes*. 2004;**53**:294-305.
DOI: 10.2337/diabetes.53.2.294

[58] Kuk JL, Kilpatrick K, Davidson LE, Hudson R, Ross R. Whole-body skeletal muscle mass is not related to glucose tolerance or insulin sensitivity in overweight and obese men and women. *Applied Physiology, Nutrition, and Metabolism*. 2008;**33**:769-774.
DOI: 10.1139/H08-060

[59] Hong S, Chang Y, Jung H-S, Yun KE, Shin H, Ryu S. Relative muscle mass and the risk of incident type 2 diabetes: A cohort study. *PLoS ONE*. 2017;**12**:e0188650. DOI: 10.1371/journal.pone.0188650

[60] Barakat R, Pelaez M, Lopez C, Lucia A, Ruiz JR. Exercise during pregnancy and gestational diabetes-related adverse effects: A randomised controlled trial. *British Journal of Sports Medicine*. 2013;**47**:630-636. DOI: 10.1136/bjsports-2012-091788

[61] Chu SY, Callaghan WM, Kim SY, Schmid CH, Lau J, England LJ, et al. Maternal obesity and risk of gestational diabetes mellitus. *Diabetes Care*. 2007;**30**:2070-2076. DOI: 10.2337/dc06-2559a

[62] American Diabetes Association. Management of diabetes in pregnancy: Standards of medical care in diabetes—2018. *Diabetes Care*. 2018;**41**:S137-S143. DOI: 10.2337/dc18-S013

[63] de Barros MC, Lopes MAB, Francisco RPV, Sapienza AD, Zugaib M. Resistance exercise and glycemic control in women with gestational diabetes mellitus. *American Journal of Obstetrics and Gynecology*. 2010;**203**:556.e1-556.e6. DOI: 10.1016/j.ajog.2010.07.015

[64] Brankston GN, Mitchell BF, Ryan EA, Okun NB. Resistance exercise decreases the need for insulin in overweight women with gestational

diabetes mellitus. *American Journal of Obstetrics and Gynecology*. 2004;
190:188-193. DOI: 10.1016/s0002-9378(03)00951-7

[65] Kazemi N, Ali HS. Comparison the effects of aqua aerobic and resistance training on blood sugar and insulin resistance in women with gestational diabetes mellitus. *Journal of Physical Activity and Hormones*. 2017;**1**:1-18

[66] Kasraeian M, Talebi S, Kazemi N, Bazrafshan K, Asadi N, Idress Ahmad Mohammad R, et al. Insulin resistance and homeostasis model assessment of β -cell function in females with gestational diabetes mellitus: A comparison of aerobic and resistance trainings. *Journal of Advanced Medical Sciences and Applied Technologies*. 2017;**3**:131-138. DOI: 10.32598/jamsat.3.3.131

[67] Sklempe Kokic I, Ivanisevic M, Kokic T, Simunic B, Pisot R. Acute responses to structured aerobic and resistance exercise in women with gestational diabetes mellitus. *Scandinavian Journal of Medicine & Science in Sports*. 2018;**28**:1793-1800. DOI: 10.1111/sms.13076

[68] Refaye GEE, Aziz GFA. Comparative study of circuit resistance training and aerobic training on glycemic control of gestational diabetes mellitus. *Bulletin of Faculty of Physical Therapy*. 2017;**22**:89-95. DOI: 10.4103/bfpt.bfpt_46_16

[69] Stafne SN, Salvesen KÅ, Romundstad PR, Eggebø TM, Carlsen SM, Mørkved S. Regular exercise during pregnancy to prevent gestational diabetes: A randomized controlled trial. *Obstetrics & Gynecology*. 2012;**119**:29-36. DOI: 10.1097/AOG.0b013e3182393f86

[70] Chevenne D, Trivin F, Porquet D. Insulin assays and reference values.

Diabetes & Metabolism. 1999;
25:459-476

[71] Reyes-Muñoz E, Martínez-Herrera EM, Ortega-González C, Arce-Sánchez L, Ávila-Carrasco A, Zamora-Escudero R. HOMA-IR and QUICKI reference values during pregnancy in Mexican women. *Ginecología y Obstetricia de México*. 2017;85:306-313

IntechOpen