Gestational Diabetes:
Risks, Prevention, and Treatments

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1. Introduction & General Information

1.1 Defining Gestational Diabetes

Diabetes is a chronic disease that is estimated to affect 578 million people worldwide by 2030 (International Diabetes Federation [IDF], 2019) and already has a significant economic impact around the world. Gestational diabetes mellitus (GDM), a sub-type of diabetes, is defined as a carbohydrate intolerance that is first detected during pregnancy (Mirghani Dirar & Doupis, 2017; World Health Organization [WHO], 1999) and comprises of abnormal glucose tolerance, or higher than normal blood glucose levels, that may or may not diminish following the birth of an infant. Given that gestational diabetes is multi-causal and increases the likelihood of adverse short-term and long-term health outcomes for mother and child, it is imperative to identify possible risks of developing GDM in order to encourage preventative strategies prior to or during pregnancy.

Although GDM is asymptomatic, it is associated with serious perinatal complications for both the mother and the child, which can be minimized with detection and management of the hyperglycaemia (Meek et al., 2015; Stewart & Murphy, 2015). Mothers with GDM are at risk of a plethora of health issues throughout pregnancy, during birth, and postnatally. These risks include, but are not limited to, pre-eclampsia, gestational hypertension, delivery of an infant that is large for gestational age, unplanned caesarean section, maternal suffering from birth injuries (Crane, Wojtowycz, Dye, Aubry, & Artal, 1997; Kim & Ferrara, 2010; Xiong, Saunders, Wang, & Demianczuk, 2001), hydramnios, developing type 2 diabetes post pregnancy, and mortality (Bryson, Ioannou, Rulyak, & Critchlow, 2003; Kim & Ferrara, 2010). Although GDM is a diagnosis given to the mother, this does not limit the infant from risks before, during, and/or after birth. Infants born from a pregnancy with abnormal glucose thresholds are at a heightened risk for macrosomia, hypoglycemia, adult metabolic syndrome, type 2 diabetes, obesity later in life (Sobngwi et al., 2003; Xiong et al., 2001), and an increased risk of autism spectrum disorders (Xiang et al., 2015). These risks, along with current knowledge of risk factors, preventative strategies, screening strategies, and treatment approaches are expanded on below.

1.2 Prevalence of Gestational Diabetes

Estimates of GDM prevalence vary due to differences in screening threshold values between countries, as well as the differences of susceptibility between populations; however, no matter the testing strategy, GDM prevalence rates over the last decades continue to increase (Ferrara, 2007; Lavery, Friedman, Keyes, Wright, & Ananth, 2017; Public Health Agency of Canada [PHAC], 2014). This rise is believed to be the result of the rise of obesity and type 2 diabetes, as well as the younger onset of these two risk factors (American College of Obstetricians and Gynaecologists [ACOG], 2017). Estimated incidence rates within Western countries are reportedly 5-7% of all pregnancies (Caissutti & Berghella, 2017; Piper, Stewart, & Murphy, 2017) while others estimate that as high as 16% of all live births had some form of hyperglycaemia (i.e., high levels of blood glucose) in pregnancy, 84% of which were cases of

GDM (International Diabetes Federation, 2019). Similar to the rates in other Western countries, 5.45% of Canadian births in 2010/2011 were afflicted with GMD, with the prevalence of GDM in Canada steadily increasing (PHAC, 2014).

1.3 Economic Costs of Gestational Diabetes

Treatment of GDM is costly to a national healthcare system (Chen et al., 2009). Although the current economic costs of gestational diabetes in Canada are not available, US projections suggest that GDM costs more than $1.8 billion dollars a year (Lenoir-Wijnkoop, van der Beek, Garssen, Nujiten, & Uauy, 2015). Lenoir-Wijnkoop and colleagues (2015) calculated that the short-term conservative costs per case of GDM are $7,803 USD more than a normal pregnancy/delivery. Therefore, it is assumed that a reduction in GDM pregnancies would result in fewer complications and decreased healthcare costs worldwide. However, these projections likely underestimate the true costs of GDM because they do not account for the long-term economic costs associated with the future health of infants who were delivered by a mother with GDM, as well as the impact of GDM pregnancies on future generations.

2. Risk Factors

There are a number of risk factors that are out of the control of the mother, which may increase the likelihood a woman developing GDM during her pregnancy. These risk factors include PCOS, ethnicity, genetics, age, fetal sex, and pre-existing diabetes. However, having one or more of the following risk factors does not guarantee a GDM diagnosis, but rather suggests a need for screening during pregnancy for at-risk mothers. Each of these risk factors will be expanded upon below.

2.1 PCOS

Gestational diabetes and polycystic ovary syndrome (PCOS) are the most common endocrine disorders in women of reproductive age (Mustaniemi et al., 2018) with an estimated 5-20% of reproductive-aged women being affected with PCOS globally (Azziz et al., 2016). Women with PCOS may struggle with infertility, obesity, and insulin resistance (Fauser et al., 2012), with insulin resistance accepted as one of the key biochemical features of PCOS (Glueck, Goldenberg, Sieve, & Wang, 2008). Due to their increased risk of insulin resistance (Kjerulff et al., 2011), it is well documented that PCOS is associated with an increased risk of GDM (Ashrafi et al., 2014; Lo et al., 2006; Palomba et al., 2015; Reyes-Munoz et al., 2012). This is because reduced insulin sensitivity during pregnancy predisposes a woman with PCOS to glucose intolerance, therefore increasing the risk of GDM (Lo et al., 2006). However, some studies propose that a high BMI may be a better predictor of GDM than PCOS (Kakoly, Earnest, Moran, Teede, & Joham, 2017). This uncertainty has created the need for further investigation within the field of reproductive health. To further complicate understanding the relationship between PCOS and GDM, Ashrafi et al. (2014) suggests that the field examine the relationship between infertility and infertility treatment and the development of GDM, rather than focusing on PCOS. Ashrafi supported this proposal by highlighting the increased risk of developing GDM for non-PCOS patients who utilized assisted reproductive technologies (ART) as a means of conceiving.

Specifically, the incidence rate of GDM for those who used ART was significantly higher (23-43%, dependent on intervention used) compared to the spontaneous pregnancy group (10%) (Ashraf et al., 2014). Along this line, Ashrafi and colleagues (2014) found the use of progesterone during pregnancy in women who were treated with ART increased the prevalence of GDM in non-PCOS women. These findings are important because many women with PCOS undergo infertility treatments as a means of conceiving. Therefore, the relationship found between PCOS and GDM may be confounded by the use of ART treatments. Overall, metabolic screening before conception or in the early stages of pregnancy may be beneficial for women with PCOS as a means of determining their risk of developing GDM during pregnancy (Ashrafi et al., 2017).

2.2 Ethnicity

Rates of GDM differ between ethnic/racial groups. In fact, one of the most reliable predictors of GDM is the mother's ethnicity (Yuen & Wong, 2015). What's more, women who have migrated from their countries of origin to a western society have a significantly higher rate of GDM than women of a foreign ethnicity who have lived in western countries all their lives (Savitz et al., 2008; Yuen & Wong, 2015). Although the reason for this is not clear, it is hypothesized that it could be related to changes in diet and lifestyle, as well as stress, although there may also be genetic and epigenetic causes (Hedderson, Darbinian, & Ferrara, 2010a). Women who have come to Canada from certain populations within Asia, the Pacific Islands, Africa, and Latin America are at a greater risk of developing GDM compared to women of European background (Yuen & Wong, 2015). Within these identified ethnic groups there are inter-group differences, for example GDM is more common in South Asian women (India, Sri Lanka) than South-East Asian (Vietnam, Cambodia) and East-Asian (Chinese, South Korean) (Yuen & Wong, 2015). It is important to point out that the diagnostic criteria used for GDM impact the prevalence rates among different ethnicities. For example, utilizing the WHO criteria adopted in 2013, prevalence rates decreased in women of Chinese ethnicity, whereas the same criteria increased the prevalence rates in women of Anglo-European ethnicity (Moses et al., 2011). Therefore, current blanket diagnostic criteria may not effectively detect GDM in all ethnic groups and suggests that screening and diagnostic criteria, should be individualized (Shah et al., 2011).

Within Canada, the group most vulnerable to developing diabetes and GDM is First Nations women (Aljohani et al., 2008; Chamerlain et al., 2014; Dyck, Klomp, Tan, Turnell, & Boctor, 2002; Dyck et al., 2010; Dyck et al., 2019). The prevalence of type 2 diabetes overall has been found to be four times higher in Canadian First Nations women than non-First Nations women (Dyck et al., 2010). Recently it was found that the rate of both diabetes and GDM has been increasing for both groups but has risen more steeply for First Nations women (Dyck et al., 2019). Between 1980 and 2009, in Saskatchewan the rate of GDM rose from 1.0% to 6.6% among First Nations and from 0.4% to 3.6% among non-First Nations (Dyck et al., 2019). The incidence of diabetes in pregnancy, including both GDM and diabetes diagnosed prior to pregnancy, was found to be 2-3 times higher among First Nations women (Dyck et al., 2019).

Understanding the reasons for this disparity is complex. First Nations women have more risk factors for GDM, for example, higher blood sugar levels, higher rates of obesity, and higher waist circumferences (Oster & Toth, 2009). Therefore, it is likely that these factors, along with socio-economic factors, and lower prenatal care, contribute to the risk of developing GDM in First Nations women (Shen et al., 2015). It has also been suggested that the ongoing effects of colonization contribute to the higher rates of GDM for First Nations women by amplifying the impact of existing GDM risk factors (Dyck et al., 2019). A qualitative study by Oster, Mayan, and Toth (2014) in Alberta found that First Nations women interviewed experience cultural barriers to receiving care, such as a lack of a holistic approach to care, significant power imbalances within the patient–provider relationship, and a lack of understanding from health care providers.

One factor that may serve to increase the risk, and prevalence, of GDM is diet. Based on a series of randomized control trials, it is recommended that women with GDM consume a diet rich in complex carbohydrates and fibre and low in simple sugar and saturated fats (Hernandez et al., 2013). Following these recommendations may be particularly difficult for Canadian First Nations women. Recent research suggests that pregnant women living in First Nations rural communities have less access to healthy diets and are less likely to take part in physical activity than non-First Nations women in urban communities (Back et al., 2012). If a mother's diet cannot be suitably modified, another option for treatment is insulin therapy (Hernandez et al., 2013), which again might be difficult to obtain in a rural setting in Canada.

The qualitative study by Oster et al., (2014) identified factors that may allow First Nations women to take control of their health and manage their GDM. These factors include having a strong support system (family in particular), a sense of autonomy, and being aware of the challenges of pregnancy and diabetes in particular. It is possible that workshops aimed at improving these factors could hold promise in mitigating the impacts of the surging rates of GDM in First Nations women in Canada.

2.3 Genetics

Pregnancy includes an increase in adipose tissue and weight gain, specifically during the second and third trimester (Buchanan & Xiang, 2005), resulting in some insulin resistance. This increase in insulin resistance is typically not a problem in pregnant women with normal glucose control because their pancreas adjusts their insulin levels in response to resistance. However, women with GDM are believed to have a limited response to insulin secretion and, therefore, cannot compensate for the increase in insulin resistance (Buchanan & Xiang, 2005), a response that is similar to those found in adults with type 2 diabetes. Recognizing the similar characteristics between GDM and type 2 diabetes, researchers examined the relationship between the two types of diabetes and determined that a diabetic family history increases the risk of developing glucose intolerance during pregnancy (Kim, Liu, Valdez, & Beckles, 2009).

With this understanding, most screening criteria includes a family history of diabetes as an indicator of whether an oral glucose tolerance test is advisable.

A recent meta-analysis found that GDM in women with a positive familial history of diabetes was 3.46 times greater than women without a familial history ; however, having two parents affected with diabetes does not appear to increase the risk of developing GDM (Kim et al., 2009). What is interesting is at least one study suggests that women with a mother affected by diabetes are at an increased risk of developing GDM compared to women with a father affected with the disease (Dabelea, 2007). However, this is not replicated in all studies (Kim et al., 2009). Furthermore, a woman with a sibling with a history of diabetes, rather than a parent, was at greater odds of developing GDM than those with just a parent (Kim et al., 2009).

Due to the shared risk factors and similar pathophysiology of GDM and type 2 diabetes, experts have explored the possible association between a family history of type 2 diabetes and women's susceptibility of developing GDM. Family history of diabetes is found to be a strong predictor of GDM (Wung & Lin, 2011). This may be because women with GDM are more likely to carry the susceptibility genes for type 2 diabetes (Wung & Lin, 2011). At least 20 susceptible genes of type 2 diabetes have been studied in women with GDM, with three genes presenting promise in predicting GDM development: the TCF7L2 gene, the KCNQ1 gene, and the CDKAL1 gene (Wung & Lin, 2011). However, each gene appears to be associated with GDM in different populations. For example, the TCF7L2 gene is associated with women from Scandinavian, Korean, Danish, and Greek descent (Cho et al., 2009; Lauenborg et al., 2009; Pappa et al., 2011; Shaat et al., 2007), while the KCNQl gene increased the risk of GDM in women of Korean and Chinese descent (Kwak et al., 2010; Shin et al., 2010; Zhou et al., 2009) and CDKALl was related to GDM in women of Korean (Cho et al., 2009) and Danish descent (Lauenborg et al., 2009). To add confusion, carrying more than one of the other 17 genes may predispose women to develop GDM, while other genes may limit the risk of GDM through a synergistic effect. Therefore, the literature suggests that there is a genetic factor associated with the development of GDM (Wung & Lin, 2011); however, substantial work is needed before definite conclusions can be made.

2.4 Age

Gestational diabetes mellitus is consistently associated with older maternal age (Anna et al., 2008; Dode & Santos, 2009; Kuo et al., 2017), where the risk of GDM increases incrementally beginning at 20 years of age and becomes an even greater risk among women older than 35 years (Anna et al., 2008; Chen et al., 2009), though one study suggests that there is a significant increase in risk of developing GDM as early as 25 years old (Lao, Ho, Chan, & Leung, 2006). Women aged 35 years or older are two to four times more likely to develop GDM than women aged 18-34 (Ferrara et al., 2004; Jolly et al., 2000). PHAC reported that Canadian mothers 30 years or older are more likely to be diagnosed with GDM, with rates increasing by 3% for every 5-year increase in mother's age (6.04% for 30-34 year olds, 9.14% for 35-39 year olds, 12.19% for 40-44 year olds, and 16.78% for 45-49 year olds). With the consistent association between

age and risk of GDM found within the literature, age appears to be one of the most reliable and strongest predictors for GDM (Teh et al., 2011). This is concerning, because the average age of childbirth is over 30 in Canada, with the average age at first birth in 2011 being 28.5 years (Statistics Canada, 2018). Therefore, pregnant women within Canada at or above the age of 25 should be assessed for GDM in order to limit undetected GDM within the Canadian population.

2.5 Fetal Sex

There is recent speculation that the sex of a fetus may be associated with the development of GDM in pregnant women due to the effects fetal sex may have on maternal metabolism. This interest came from the substantial evidence that female fetuses are more insulin-resistant than male fetuses in utero (Ibanez et al., 2008; Shields et al., 2007). A mother's metabolic health may be affected by the circulation of hormones between the mother and the fetus (Reis et al., 2001) which may impact a mother's maternal insulin sensitivity (Xiao, Zhao, Nuyt, Fraser, & Luo, 2014). A recent study found that women pregnant with a female fetus have higher insulin concentrations and lower glucose-to-insulin ratios than women pregnant with a male fetus (Xiao et al., 2014). Likewise, women who carry a male fetus are found to have higher blood glucose levels than their female fetus carrying counterparts (Retnakaran et al., 2015). This is because women pregnant with a male fetus are more likely to have lower β-cell function (i.e., low production and secretion of insulin) during a pregnancy than women bearing a female fetus (Retnakaran et al., 2015). Therefore, women pregnant with a male fetus are at greater risk of developing GDM and may benefit from GDM screening; however, further investigation is warranted.

2.6 Pre-existing Diabetes

The term "pre-existing diabetes" refers to diabetes diagnosed prior to conceiving. Traditionally, diabetes mellitus has been placed into one of two categories: type 1 and type 2. Type 1 diabetes is autoimmune in nature and is characterized by an inability to produce insulin and regulate blood sugar (Diabetes Canada, 2020). This deficit is thought to originate from the body attacking the pancreas, the source of insulin in the body (Katsarou et al., 2017). Roughly 10% of all diabetes mellitus cases are type 1, with the majority of cases developing in childhood or adolescence, although cases have been reported to develop in adulthood (Katsarou et al., 2017). In contrast to type 1, type 2 diabetes is characterized by an inability to utilize the insulin produced by the body or not being able to produce sufficient amounts of insulin (Defronzo, 2015). Over 90% of all diabetes cases are type 2 in nature, with the vast majority of cases developing in adulthood (Defrozo et al., 2015). Pregnant women with pre-existing diabetes are at a higher rate of complications during pregnancy compared to the general population. These complications include, but are not limited to, mortality, congenital malformations, hypertension, preterm delivery, large for gestational age infants, and caesarean delivery (Feig et al., 2014; Feig et al., 2006; MacIntosh et al., 2006), many of which are experienced by women who develop GDM. It is important to understand that GDM only occurs in women who do not already have type 1 or type 2 diabetes; therefore, women who have type 2 diabetes will not develop GDM as they already have diabetes before they were pregnant (Centers for

Disease Control and Prevention, 2019). Thus, women who have pre-existing diabetes do not need to be screened for GDM, but rather, followed by a healthcare provider to ensure that their pre-existing diabetes is being adequately managed throughout their pregnancy.

3. Behavioural Risk Factors

The current section discusses the role of behavioural risk factors associated with the development of GDM that are responsive to interventions. These factors include a mother's weight before or during pregnancy, her nutritional choices, and her sleep patterns. Adjusting one's lifestyle behaviours before or during pregnancy may decrease the chances of developing GDM or limit the effects GDM may have on a mother's and/or her child's short-term and long-term health.

3.1 Sleep

Sleep duration and quality are important risk factors for chronic disease in non-pregnant adults (Knutson, 2010; Morselli, Leproult, Balbo, & Spiegel, 2010; Mullington, Haack, Toth, Serrador, & Meier-Ewert, 2009). In general, women have been sleeping less over the past few decades. Pregnant women are vulnerable to sleep disturbances due to hormonal changes, physical discomfort, or anxiety (Hedman, Pohjasvaara, Tolonen, Suhonen-Malm, & Myllyla, 2002). Sleep patterns are prone to change throughout gestation, with mothers sleeping more during the first trimester, less in the second trimester, and napping frequently in first and third trimesters (Hedman et al., 2002; Lee, Zaffke, & McEnany, 2000), all of which affect total sleep exposure (Mindell & Jacobson, 2000). Although the exact mechanisms are yet to be determined, evidence suggests that insufficient sleep duration and poor sleep quality are associated with metabolic and neuroendocrine alterations that may impair glucose tolerance (Schmid, Jauch-Chara, Hallschmid, & Schultes, 2009; Schmid et al., 2007; Spiegal, Knutson, Leproult, Tasali, & Van Cauter, 2005). Specifically, reduced and prolonged sleep duration are linked to impaired insulin sensitivity and glucose metabolism (Izci-Balserak & Pien, 2014; Reutrakul & Van Cauter, 2014). In terms of GDM, short sleep duration and snoring (i.e., disturbed sleep) are associated with glucose intolerance and gestational diabetes (Facco, Grobman, Kramer, Ho, & Zee, 2010; Qiu, Enquobahrie, Frederick, Abetew, & Williams, 2010) and sleeping duration in the second trimester, rather than the first, is a significant indicator of risk of gestational diabetes (Cai et al., 2017; Rawal, Hinkle, Zhu, Albert, Zhang, 2017). For women who sleep less than 7 hours each night (Rawal et al., 2017), the odds of developing GDM are approximately two-fold to those who sleep 8-9 hours (Facco, Grobman, et al., 2017). This is because shortened sleep durations are associated with worsened glucose control in women with gestational diabetes (Twedt et al., 2015). The mechanisms for the relationship between sleep and glucose tolerance during pregnancy include elevated oxidative stress, increased systemic inflammation, dysregulation of energy homeostasis, and chronic activation of the hypothalamic-pituitary-adrenal axis (Izci-Balserak & Pien, 2014; Reutrakul & Van Cauter, 2014), as well as amniotic dysfunction, endothelial damage, and altered hormonal regulation of energy expenditure (Dempsey, Veasay, Morgan, & O'Donnell, 2010) all of which are associated with adverse pregnancy outcomes (Romero & Badr, 2014). In addition, Facco, Grobman, et al. (2017) found that late

sleep midpoint, or the clock time midpoint of sleep onset and offset of 5 a.m. or later, was associated with GDM. In short, both sleep duration and timing of sleep in the second trimester is associated with the development of GDM.

Not only is the quantity of sleep a risk factor for women developing gestational diabetes, but so too may the quality of sleep a woman experiences throughout pregnancy. Fifteen to 25% of pregnant women report frequent snoring, a symptom of obstructive sleep apnea (Facco, Kramer, Ho, Zee, & Grobman, 2010; Pien, Fife, Pack, Nkwuo, & Schwab, 2005). However, literature regarding the association between sleep quality and the development of GDM is conflicting. The findings appear to be dependent on the method in which the data is collected. For example, self-report questionnaire-based studies report that poor sleep during pregnancy is associated with increased risk of GDM (Wang et al., 2016; Zhong et al., 2018), while studies that objectively measure sleep patterns in women do not observe an association when monitoring daily sleep of pregnant women for 7-days mid-pregnancy (Facco, Parker, et al., 2017). Yet results from a meta-analysis examining 9,795 pregnant women's sleep quality found that sleep-disordered breathing was a significant risk of GDM, with women with sleep disordered breathing having more than a threefold increased risk of GDM (Luque-Fernandez, Bain, Gelaye, Redline, & Williams, 2013). In a large sample study examining the prevalence of sleep disordered breathing in pregnant women, 3.6% of women experience disordered breathing in early pregnancy; an experience that increased to 8.3% of women in mid-pregnancy (Facco, Parker, et al., 2017). Again, women who reported sleep disordered breathing in early and mid-pregnancy were 2.79 times more likely to develop GDM than non-sleep disordered pregnant women (Facco, Parker, et al., 2017). Therefore, due to the simplicity of treating sleep disordered breathing, it is highly suggested that women address sleep disordered breathing, such as sleep apnea, with their healthcare provider. Education on healthy sleeping, screening for, and treating sleeping disorders during pregnancy may aid in controlling blood glucose levels in pregnant mothers with gestational diabetes (Twedt, Bradley, Deiseroth, Althouse, & Facco, 2015).

3.2 Obesity and Weight Gain in Pregnancy

Gestational diabetes and type 2 diabetes share many common risk factors including being overweight or obese. Being overweight and obese are defined as an excessive or abnormal fat accumulation that is likely to impair one's health (Muller & Nirmala, 2018). In terms of complications during pregnancy, having a BMI of greater than 30 increases a mother's risk of developing GDM, making it three times more likely than for a woman with a BMI of 25 or less (Chu et al., 2007), while excessive gestational weight gain that occurs in the first trimester can increase the risk of GDM by a factor of 1.4 (Brunner et al., 2015). Furthermore, Sommer et al. (2014) found that a 0.14 kg increase in abdominal fat per week during the second trimester increased the risk of a mother developing GDM by a factor of 1.31 (Sommer et al., 2014). This finding does not suggest that women should not gain weight throughout their pregnancy (see Table 1 for weight gain recommendations), but rather the finding suggests that women should limit the amount of rapid fat gain around their abdomen during the second trimester.

Therefore, in order to limit the prevalence of GDM in women, efforts have been made in healthcare practice to avoid excessive gestational weight gain that would lead to greater fat deposition and may impair insulin sensitivity (Hedderson, Gunderson, & Ferrara, 2010; Lewis, Carpentier, Adeli, & Giacca, 2002).

Table 1. Recommended weight gain based on women's pre-pregnancy BMI

	Mean rate of weight gain in the 2nd and 3rd trimester		Recommended total weight gain	
Pre-pregnancy BMI	**kg/week**	**lb/week**	**kg**	**lb**
BMI < 18.5	0.5	1.0	12.5 – 18	28 – 40
BMI 18.5-24.9	0.4	1.0	11.5 – 16	25 – 35
BMI 25.0-29.9	0.3	0.6	7 – 11.5	15 – 25
BMI > 30.0	0.2	0.5	5 – 9	11 – 20

(Health Canada, 2014)

Maternal weight has been identified as the strongest predictor of infant macrosomia and can have detrimental effects on a mother's insulin resistance despite normal glucose tolerance (Jolly, Sebire, Harris, Regan, & Robinson, 2003). For every 2 kg of excessive weight gained during GDM management, there was a 32% greater likelihood of insulin increasing detrimentally (Barnes et al., 2020). Therefore, some experts suggest that obese women should not gain any additional weight during pregnancy (Sagedal et al., 2016), while others suggest a targeted weight plan for overweight and obese pregnant women (Bennett et al., 2018). This targeted weight plan generally includes a recommendation that women who were overweight or obese pre-pregnancy be restricted in their weight gain during pregnancy compared to women of a healthy weight pre-pregnancy (Institute of Medicine, 2009) due to their risk of developing GDM. With the inconsistencies found between experts, it may be best to consult a healthcare professional regarding best approaches to weight gain during pregnancy.

Interestingly, interpregnancy weight change can also increase the risk of GDM in women who did not previously develop GDM in their first pregnancy. It is suggested that the effect of this weight change may depend on a woman's pre-pregnant BMI of her first pregnancy (Villamor & Cnattingius, 2006). An observational study of 24,198 mothers and their first two pregnancies determined that the risk of GDM increased with increasing weight gain from first to second pregnancy, especially for women with a BMI less than 25 in their first pregnancy (Sorbye, Skjaerven, Klungsoyr, & Morken, 2017), a finding that was previously supported within the literature (Ehrlich et al., 2011). However, for women who were overweight and obese in their first pregnancy, an interpregnancy weight gain of three or more BMI units had an increased risk of GDM (Villamor & Cnattingius, 2006). Importantly, the highest risk of GDM was found in women who had the greatest weight gain between pregnancies (Bogaerts et al., 2013; Sorbye et al., 2017) compared to women who kept their weight stable between pregnancies (Sorbye et al., 2017). Therefore, efforts must be made to promote healthy weight gain during pregnancy for all women, rather than just for overweight and obese pregnant women. These efforts to

maintain a healthy weight should continue after pregnancy in order to decrease the chances of GDM in subsequent pregnancies.

3.3 Nutrition

Due to the relationship between obesity and the development of GDM, dietary treatment has long been recommended for women who develop GDM. Understanding the inverse relationship between several healthy diets and type 2 diabetes among non-pregnant adults (Fung, McCullough, van Dam, & Hu, 2007; Salas-Salvado et al., 2011), researchers have examined whether dietary factors (irrespective of BMI) before and during pregnancy are related to GDM risk. A large prospective study found that pre-pregnancy adherence to a healthy diet was associated with a significant decrease in GDM risk for pregnant women (Tobias et al., 2012). Studies examining the relationship between GDM and diet suggest that a high intake of red and processed meats, saturated fats, refined grains, sweets, high-fat dairy, and fried foods are associated with a significant elevated risk of developing GDM (Schoenaker, Mishra, Callaway, & Soedamah-Muthu, 2016; Zhang & Ning, 2011; Zhang, Liu, Solomon, & Hu, 2006). Specifically, a high intake of animal protein, in particular red meat, is significantly associated with a greater risk of GDM, while a high intake of vegetable protein, specifically nuts, is significantly associated with a lower risk of GDM (Bao, Bowers, Tobias, Hu, & Zhang, 2013); a similar finding was found in diets that are primarily fruit, green leafy vegetables, poultry, and fish (Zhang, Schulze, Solomon, & Hu, 2006). This relationship between a healthy diet pre-pregnancy and GDM risk may occur due to the micro and macro nutrients available in a healthy, well-balanced diet that prevent metabolic deterioration (Hamer & Chida, 2007). In addition, whole grains that are low in the glycemic index reduce the absorption of glucose and, therefore, insulin requirements (de Munter, Hu, Spiegelman, Franz, & van Damn, 2007), which, in pre-pregnancy, is associated with reduced risk for GDM (Zhang et al., 2006). These findings suggest that a pre-pregnancy diet rich in vegetable protein, high in fruit and vegetable intake, and including whole grains with a low glycaemic index may provide some protection from developing GDM.

3.4 Vitamin D

The majority of pregnant women are vitamin D deficient (Holmes et al., 2009); however, this is believed to be a reflection of a woman's usual vitamin D status prior to pregnancy rather than a consequence of pregnancy (Alzaim & Wood, 2013). Though the mechanisms behind the interaction between vitamin D and GDM and not fully understood, there is growing interest in the documented association of vitamin D deficiency and impaired glucose metabolism (Alvarez & Ashraf, 2010; Pittas, Lau, Hu, & Dawson-Hughes, 2007). Several studies report an association between vitamin D deficiency and GDM (Alzaim & Wood, 2013; Poel et al., 2012). Poor vitamin D status is associated with poor blood glucose control (Clifton-Bligh, McElduff, & McElduff, 2008; Farrant et al., 2009; Lau et al., 2011), and women with GDM are found to have lower vitamin D levels than pregnant women without GDM (Maghbooli et al., 2008).

While some studies suggest that there is no association between first trimester vitamin D levels and the subsequent development of GMD (Makgoba et al., 2011), much of the literature supports a relationship between vitamin D and GDM in at-risk pregnant women. For example, vitamin D deficiency in early pregnancy (approximately 16 weeks gestation) is found to elevate a mother's risk for developing GDM between weeks 24 and 28 of gestation (Soheilykhah et al., 2010; Zhang et al., 2008), with women who are overweight and vitamin D deficient having an even greater risk (6-fold) of developing GDM compared to lean women with adequate vitamin D status (Zhang et al., 2008). Along the same lines, Burris and colleagues (2014) found that second trimester vitamin D levels were inversely associated with glucose levels, which the authors suggested may be associated with increased risk of GDM. To support these findings, a meta-analysis and systematic review of vitamin D and gestational diabetes reported a significant inverse relationship between vitamin D and the incidence of GDM (Poel et al., 2012). Healthcare providers often discuss the need of vitamin D with expectant mothers during their first prenatal appointment. During this time, women are recommended to intake 200-400 IU/day. However, previous findings suggest that 400 IU/day is not adequate to achieve normal vitamin D levels required by pregnant women (Cockburn et al., 1980), with studies supplementing 800-1600 IU vitamin D per day showing little to no effects on third trimester blood levels (Vieth, Chan, & MacFarlane, 2001). Therefore, due to the low likelihood of toxicity, experts suggest that pregnant women, regardless of their risk of developing GDM, supplement their diet with a daily dose of vitamin D ranging between 1000 and 2000 IU/day, while daily doses of 4000 IU/day is recommended for pregnant women who are vitamin D deficient (Mithal & Kalra, 2014).

3.5 Iron

Iron deficiency is common among pregnant women and women are often recommended to supplement iron as part of a healthy prenatal lifestyle (ACOG, 2008). Women are advised to consume 27 mg/day of iron due to its importance as an essential component for the production of hemoglobin for the mother and the fetus, which functions in the delivery of oxygen from the lungs to the tissue and buffers against blood loss during delivery (Wilson, Gummow, McAninch, Miotto, & Roberts, 2018). Evidence suggests that blood iron levels are associated with glucose metabolism. These associations are seen within non-pregnant adults where a deficiency in iron may disturb glucose metabolism and increase the risk for type 2 diabetes (Hansen, Moen, & Mandrup-Poulsen, 2014; Montonen et al., 2012; Zhao et al., 2012). It is important to note, however, that the source of iron is a key factor in whether women are at higher or lower risk of developing GDM. Dietary intake of heme iron rich food (e.g., red meat) is associated with an increased risk of GDM (Bowers et al., 2011; Qiu et al., 2011), where women who consumed the highest quartile of heme iron rich food during pre-pregnancy and early pregnancy were two times more likely to develop GDM than women who were in the lowest quartile (Qiu et al., 2011). In contrast, higher nonheme iron intake before pregnancy has been shown to lower the risk of GDM (Darling, Mitchell, & Werler, 2016), suggesting that diets high in vegetables, fruits, legumes, and nuts (i.e., nonheme iron sources) may provide some protection against GDM (Boers, Tobias, Yeung, Hu, & Zhang, 2012; Zhang et al., 2006). Iron

supplementation has not been found to impact the risk of GDM (Bowers et al., 2011; Chan, Chan, Lam, Tam, & Lao, 2009; Kinnunen, Luoto, Helin, & Hemminki, 2016) and is consequently insufficient in lowering the risk of GDM in at-risk women. Therefore, pregnant women should be advised to consume 27 mg/day of nonheme (i.e., plant based) iron through iron-rich foods in order to attempt to lower their risk of GDM.

3.6 Smoking

Smoking is associated with an abundance of health consequences, including, but not limited to, coronary heart disease, stroke, cancer, respiratory disease, and death (World Health Organization [WHO], 2012). Smoking is also known to affect a mother and her fetus during pregnancy. Specifically, women who smoke may be more likely to develop GDM than women who abstain from smoking during pregnancy. The relationship between smoking and GDM is currently inconclusive. While many studies find a significant association between cigarette smoking and GDM (Leng et al., 2015; Solomon et al., 1997; Zaren, Lindmark, Wilbell, & Folling, 2000; Zhang et al., 2014), several studies also suggest that little to no relationship can be found between smoking during pregnancy and GDM (England et al., 2004; Hosler, Nayak, & Radigan, 2011). Interestingly, maternal smoking history may relate to an increased risk in the development of GDM in offspring daughters (Bao et al., 2016). Specifically, maternal smoking of 25 or more cigarettes per day during pregnancy is associated with a 98% higher risk of GDM during the daughter's own pregnancy (Bao et al., 2016). However, further research is necessary to determine whether the relationship between maternal smoking and GDM during pregnancy is causal. Overall, through an examination of the literature it appears that smoking may pose a risk of developing GDM for already at-risk women. Therefore, due to the additional negative consequences that are associated with smoking, the general consensus among the literature is that all women of reproductive age, prospective and expecting mothers, eliminate smoking as a current lifestyle choice.

4. Protective Factors

With the increasing rates of developing GDM around the world, many women and healthcare practitioners are seeking means of GDM prevention. Similar to the risk factors literature, preventative strategies in relation to GDM are complicated. There are studies that can be found to both support and challenge preventative strategies. The following include the most commonly discussed and supported prevention strategies within the literature. These include exercise, adequate sleep, a well-balanced diet, and probiotics.

4.1 Exercise

Traditionally, pregnant women were advised to reduce their physical activity levels due to the belief that physical activity could reduce placental circulation and result in miscarriages or preterm delivery (Shcramm, Stockbauer, & Hoffman, 1996). However, research conducted in the last two decades suggests that physical activity is part of a well-developed antenatal care plan (ACOG, 2015). Pregnant women who exercise throughout their pregnancy may experience

benefits such as improved physical condition, control of body weight, shorter duration of labour, quicker recovery after birth, prevention of health conditions (e.g., GDM), and reduced risk of premature birth (Blaize, Pearson, & Newcomber, 2015). Beginning or maintaining an exercise routine along with following a healthy diet may reduce the risk of GDM-related outcomes during pregnancy (Shepherd et al., 2017). Taking part in physical activity prior to conception reduces the risk of developing GDM (Colberg, Castorino, & Jovanovic, 2013), and for those diagnosed with GDM, having done pre-conception exercise may contribute to better pregnancy outcomes compared to women who begin an exercise routine during pregnancy (Zhang, Solomon, Manson, & Hu, 2006). Overall, exercise (prior to and during pregnancy) is consistently found to benefit women throughout pregnancy, independent of a GDM diagnosis (Gaston & Cramp, 2011).

The influence of physical activity on GDM outcomes are debated within the literature. A recent study examined the suitability of cycling exercise initiated in early pregnancy. Previous reviews have concluded that there is limited evidence to suggest that physical activity decreases the risks of developing GDM or improves insulin sensitivity during pregnancy (Hans, Middleton, & Crowther, 2012; Kramer & McDonald, 2006). However, recent studies utilizing well-designed randomized control trials show the potential physical activity programs may have for decreasing the risk GDM in pregnant women. For example, cycling for 30 minutes, three times per week, was associated with a significant reduction in GDM frequency in overweight/obese pregnant women (Wang et al., 2017). Other findings suggest that participating in physical activity during the first 20 weeks of pregnancy may result in a 50% reduced risk of GDM (Dempsey et al., 2004). In particular, favourable effects on glucose control and prevention of insulin use are found for women who perform physical activity at <60% of their VO2max, or the maximum amount of oxygen utilized during intense exercise (Davenport, Mottola, & McManus, 2008). In terms of the effects of exercise on insulin sensitivity, women at high risk of developing GDM who took part in a walking and nutritional intervention program maintained the same insulin sensitivity compared to women who were at low risk of developing GDM, and did not develop GDM (Mottola et al., 2005). Furthermore, taking part in a physical activity program pre-pregnancy or in early pregnancy may have the greatest effects of reducing the risk of GDM for at-risk women (Sanabria-Martinez et al., 2015; Tobias, Zhang, van Dam, Bowers, & Hu, 2011). Interestingly, a recent meta-analysis found that utilizing an exercise regimen that combined multiple types of exercise (e.g., weightlifting, cardio, and stretching) had the greatest effect at reducing the risk of GDM in at-risk women (Sanabria-Martinez et al., 2015). Overall, regular, moderate-intensity exercise before and during pregnancy is part of a healthy lifestyle that may decrease the risk of developing GDM. Therefore, it is advised that mothers who are experiencing a pregnancy without contraindications (i.e., a high-risk pregnancy) continue or begin a physical activity regimen that includes 150 minutes of moderate-intensity physical activity each week to achieve reductions in pregnancy complications such as GDM (Mottola et al., 2018).

4.2 Sleep

Interestingly, napping may reduce some of the effects of low quantity/quality sleep; women who report not napping were more likely to meet the threshold of gestational diabetes (Rawal et al., 2017). Although naps are often prescribed as a means of achieving the minimum 8-9 hours/day suggested for pregnant women, longer nap duration may pose an additional threat to developing GDM. For example, one study found an association between long duration naps in early pregnancy and GDM (Balserak, Jackson, Ratcliffe, Pack, & Pien, 2013). However, this finding is challenged by a more recent study that examined the relationship between sleep duration, compensatory daytime napping, and GDM, where napping was found to modify the association between sleep duration (too long or too short) and gestational diabetes (Rawal et al., 2017). Specifically, the risk of GDM was not significantly related to sleep duration for women who napped frequently; however, a significant association was found between sleep duration and GDM for women who rarely or never napped in their second trimester (Rawal et al., 2017). In conclusion, pregnant women should be advised to aim for 8-9 hours of quality sleep, as well as be monitored for disordered sleeping patterns throughout the pregnancy. Short, uninterrupted daytime naps may be suggested for women who achieve less than 7 hours of sleep.

4.3 Nutrition

Women who are overweight or obese are at an increased risk of developing GDM during pregnancy in comparison to women of a normal weight (Olafdottir, Skulladottir, Thorsdottir, Hauksson, & Steingrimdottir, 2006). Likewise, increased weight gain throughout the pregnancy is associated with an increased risk of developing GDM (Hedderson, Gunderson, & Ferrara, 2010; Liu, Tang, & Wang, 2014). Due to the risk of developing GDM associated with weight, an extensive amount of literature focuses on the prevention of GDM through nutritional adherence. Nutritional manipulation is known to decrease the risks of developing type 2 diabetes in non-pregnant adults. However, conflicting support is found for the same relationship between specific diets and decreased risk of gestational diabetes (Agha-Jaffar, Oliver, Johnston, & Robinson, 2016; Rogozinska, Chamillard, Hitman, Kahn, & Thangaratinam, 2015).

The effects of diet during pregnancy and its association with developing GDM is inconsistent within the literature. For example, a meta-analysis in 2012 determined that a dietary intervention in any form results in a 61% risk reduction in GDM (Thangaratinam et al., 2012), while a subsequent study found changes to diet had no impact on gestational weight gain or incidence of GDM (Dodd et al., 2014). Although the literature has yet to come to a consensus on the efficacy of diet in lowering the risks of GDM, experts suggest the need for a moderation of carbohydrate intake and increase micronutrient intake by at-risk women (Tobias et al., 2012); however, most women with GDM do not meet the suggested micronutrient intake suggested by national health standards in the U.S. (Louie, Markovic, Ross, Foote, & Brand-Miller, 2013). Evidence suggests that a prudent diet, or a diet of primarily vegetables, fruit, fish, and poultry, is associated with decreased risk of developing GDM compared to a westernized

diet (i.e., high intake of red and processed meat, pizza, french fries, candy, and refined grains) (Chen, Hu, Yeung, Willett, & Zhang, 2009; Tryggvadottir, Medek, Birgisdottir, Geirsson, & Gunnarsdottir, 2016). Therefore, diets that encourage the consumption of vegetables, fruits, plant proteins, and lean meats may reduce the likelihood of developing GDM in at-risk women (Tobias et al., 2012). Such diet interventions include the Mediterranean diet, the Dietary Approaches to Stop Hypertension (DASH) diet, and the Healthy Eating Index diet, all of which are inversely associated with type 2 diabetes (Fung, McCullough, van Dam, & Hu, 2007; Liese, Nichols, Sun, D'Agostino, & Haffner, 2009; Salas-Salvado et al., 2011). These findings support current guidelines in Canada that suggest a well-balanced diet approach may be beneficial to managing weight and glucose levels (Health Link BC, 2014).

4.4 Probiotics

The potential use of probiotics as a means of controlling glucose levels within pregnant women has been of great interest (Lindsay, Walsh, Brennan, & McAuliffe, 2013; Musso, Gambino, & Cassader, 2011). Probiotics are live microorganisms that are administered as a means of improving or restoring the gut flora, and when administered in adequate amounts may promote health benefits for the host (Isolauri, Rautava, Collado, & Salminen, 2015). Probiotics pose no long-term negative consequences for mothers or infants and are, therefore, deemed safe for use during pregnancy (Luoto, Laitinen, Nermes, & Isolauri, 2010). Supporting literature suggests that the consumption of probiotics may improve glucose control in pregnant women (Laitinen, Poussa, & Isolauri, 2009) and decrease the rates of GDM for at-risk women (Luoto et al, 2010). Similarly, probiotic yogurt, one of the most common sources of probiotics, has been found to help maintain insulin concentrations in healthy pregnant women (Asemi et al., 2013), a finding that was supported by a recent meta-analysis (Zheng, Feng, Zheng, & Xiao, 2018). However, similar findings have not translated to overweight or obese pregnant women (Lindsay et al., 2014), a finding that may be due to the lack of dietary manipulation. Specifically, participants within the Laitinen et al. (2009) study were provided counselling on a low glycaemic diet in addition to consuming probiotic rich yogurt, while Lindsay et al. (2014) focused solely on probiotic consumption. Therefore, the effects of probiotics on glucose control may require additional nutritional changes for the pregnant mother. Supporting Lindsay et al.'s findings, a recent study examining the effects of two forms of probiotics administered during the second trimester in overweight and obese women found that probiotics did not prevent the development of GDM in at-risk pregnant mothers (Callaway et al., 2019). Overall, the findings suggest that the administration of probiotics during pregnancy may have the greatest effect on glucose control for women who are of healthy weight and consume a well-balanced, low glycaemic diet.

5. Impacts on Mothers' Health

While the diagnosis of GDM may be frightening for many mothers, this condition comes with an added layer of possible health risks that may affect a mother's health during or after pregnancy. These health concerns include preeclampsia, delivery risks, and the development of type 2 diabetes

post-delivery. One commonly discussed condition among the GDM literature is hydramnios (i.e., excessive amniotic fluid); however, two studies suggest that the proposed relationship between GDM and hydramnios may be unfounded. Shoham et al. (2001) and Wolf et al. (2017) found no relationship between third trimester hydramnios and GDM diagnosis among women with reported hydramnios. Therefore, the current review will focus on current maternal risks that may be cause for concern.

5.1 Preeclampsia

Conditions associated with increased insulin resistance, like GDM, may predispose women to gestational hypertension (Solomon & Seely, 2001), better understood as high blood pressure during pregnancy. Gestational hypertension and preeclampsia, a form of gestational hypertension, are more frequent among GDM mothers (Xiong et al., 2001). Interestingly, due to insulin resistance, women with GDM experience hypertensive disorders two to three times more than women experiencing a nondiabetic pregnancy (Innes & Winsatt, 1999). Preeclampsia is diagnosed in pregnant women with new onset hypertension and proteinuria in the second half (i.e., 20 weeks) of their pregnancy (ACOG, 2013); however, preeclampsia can also be diagnosed in hypertensive women who show signs of pulmonary edema, progressive renal insufficiency, impaired liver function, thrombocytopenia, or new onset cerebral or visual disturbances (ACOG, 2013). Preeclampsia increases the risk of serious consequences such as maternal (WHO, 2005) and fetal (Altman et al., 2002) morbidity and mortality, and future risk of maternal cardiovascular disease (Mosca et al., 2011) and stroke (Bushnell et al., 2014). Currently, the only known cure is the delivery of the infant.

Women who develop preeclampsia are more insulin resistant pre-pregnancy (Valdés, Sepúlveda-Martínez, Manukián, & Parra-Cordero, 2014) and in their first and second trimesters (Hauth et al., 2011). In fact, insulin resistance at 22-26 weeks (i.e., the weeks of typical GDM diagnosis) is a significant predictor of preeclampsia (Hauth et al., 2011). GDM and preeclampsia share many risk factors including maternal age, ethnicity, and obesity (Mudd, Owe, Mottola, & Pivarnik, 2013; Schneider, Freerksen, Röhrig, Hoeft, & Maul, 2012). A study examining 647,392 pregnancies found that when controlling for the similar risk factors between the two conditions, women with GDM were 1.29 times more likely to develop preeclampsia (Schneider et al., 2012), a finding that was supported further by research conducted in Canada (Nerenberg et al., 2013). Interestingly, poor glycemic control, pre-pregnancy obesity, and gestational weight gain are associated with an increased risk of preeclampsia (Barquiel et al., 2014). These findings suggest that by addressing the risk factors associated with GDM, mothers may be able to decrease their risk for GDM and preeclampsia. This is important because both pregnancy diseases are associated with maternal mortality.

5.2 Delivery Risks

GDM often results in a fetus that is deemed as macrosomic, or large for gestational age. A larger fetus presents a number of complications for the mother during delivery. Studies suggest that GDM is one of the strongest predictors of macrosomia (Mathew, Machado, Al-Ghabshi, &

Al-Haddabi, 2005; Mohammadbeigi et al., 2013). This is because GDM elevates the mother's blood glucose and insulin levels, which in turn circulates insulin from the mother to the fetus resulting in excessive fat deposits and macrosomia. Macrosomic fetuses often have larger shoulder and extremity circumferences, a decreased head-to-shoulder ratio, significantly higher body fat, and thicker upper-extremity skinfolds (KC, Shakya, & Zhang, 2015). These proportions may present challenges for the mother during delivery, such as prolonged labour in which the fetus may become stuck in the birth canal, the need for instrumental delivery (i.e., use of forceps or vacuum), or the inability for a vaginal birth and, therefore, need for an unplanned caesarean section (Turkmen, Johansson, & Dahmoun, 2018). Women who are pregnant with an infant deemed to have macrosomia are more likely to experience preterm birth, have increased risk of postpartum hemorrhage, and have a higher risk for caesarean delivery (Henriksen, 2008; Jastrow et al., 2010; Weissmann-Brenner et al., 2012). There is also a greater risk of laceration and tear of the vaginal tissue when the fetus is macrosomic, as well as greater likelihood of perineal tearing (King, Korst, Miller, & Ouzounian, 2012; Najafian & Cheraghi, 2012; Turkmenet al., 2018). Lastly, mothers carrying a macrosomic fetus are three to five times more likely to experience genital tract injury and uterine atony, or the inability of the uterus muscle to properly contract (Lazer et al., 1986). Mothers who deliver a macrosomic fetus are more likely to require longer stays in hospital post birth (Turkmen et al., 2018) because of perineal tears, genital track injuries, and/or the need for caesarean section deliveries (Irion & Boulvain, 1998). Therefore, due to the risks associated with GDM to the mother's health and safety during labour, pre-pregnancy and prenatal glucose control is imperative for both the well-being of the mother and fetus, and in addition there is a need for close monitoring of at-risk mothers during pregnancy and post birth while in hospital.

5.3 Type 2 Diabetes Post Pregnancy

For most women, delivery marks the reversal of GDM. However, some women who developed GDM during pregnancy will encounter glucose intolerance for up to a few years after giving birth. This is because the development of GDM is significantly associated with the development of type 2 diabetes later in life (Rayanagoudar et al., 2016). Anywhere from 10-31% of women who are diagnosed with type 2 diabetes report a history of GDM in at least one previous pregnancy (Cheng & Byth, 2003). Jarvela and colleagues (2006) followed 435 women diagnosed with GDM for a mean interval of six years post-birth. Of the 435 women followed, 4.6% went on to develop type 1 diabetes and 5.3% developed type 2 diabetes following a diagnosis of GDM. Women who developed type 1 diabetes had three common characteristics: < 30 years of age, required insulin to control glucose levels (i.e., nutrition and exercise interventions were insufficient), and tested positive for insulin antibodies (Jarvela et al., 2006). Rather than GDM increasing the risk of developing type 1 diabetes, the authors concluded that screening for GDM during pregnancy served to identify women at risk of developing diabetes later in life (Jarvela, 2006). Interestingly, a South Korean based study utilizing a similar paradigm as the study by Jarvela found that 12.5% of women diagnosed with GDM went on to develop type 2 diabetes within two months of delivery, and each subsequent year following delivery the

number of women diagnosed with type 2 diabetes increased at a rate of 6.8% a year (Kwak et al., 2013).

The literature suggests that the association between GDM and type 2 diabetes may be due in part to the shared risk factors of the two forms of diabetes. For example, a family history of diabetes, being overweight/obese, being of older age, and one's ethnic background are all risk factors for developing GDM and type 2 diabetes (Lauenborg, Grarup, & Damm, 2009). Furthermore, a second pregnancy is associated with a threefold increase in developing type 2 diabetes for women who developed GDM in their first pregnancy (Peters, Kyos, & Xiang, 1996), suggesting that multiple occurrences of insulin resistance may increase a woman's likelihood of developing type 2 diabetes. A meta-analysis examining the likelihood of developing type 2 diabetes post GDM pregnancy found that future risk of diabetes was influenced primarily by gestational glycaemic status (Rayanagoudar et al., 2016); however, additional factors such as hypertensive disorders, preterm delivery, and gestational age during a GDM pregnancy, as well as maternal BMI, ethnicity, and family history, were also associated with developing type 2 diabetes later in life (Rayanagoudar et al., 2016). It is also worth mentioning that other groups only go so far as to say that GDM only serves to identify women already at risk of developing type 2 diabetes (Rice, Illanes, & Mitchell, 2012). Following birth, guidelines suggest that women with GDM be followed up in the early stages after delivery to identify their risks of developing type 2 diabetes (National Institute for Health and Care Excellence, 2008). This is because mothers who remain glucose intolerant at 6 to 12 weeks postpartum are at a high risk of developing type 2 diabetes within five years (Khandelwal, 2008). Yet despite these recommendations, less than one-fifth of mothers with GDM are screened (McGovern et al., 2014). Therefore, the literature suggests that clear communication between primary and secondary healthcare providers regarding the mother's risks of developing future diabetes is imperative and women must be informed of their individual risks for developing type 2 diabetes in the future (Rayanagoudar et al., 2016).

6. Impacts on Child Health

It is well-documented that children of mothers with GDM are at risk for developing a range of unfavourable health conditions. These health conditions include perinatal death and stillbirth, macrosomia (and related injuries), hypoglycemia, childhood and adulthood weight gain, type 1 or type 2 diabetes, and cardiovascular disease. Although other offspring health conditions are loosely linked to a mother's development of GDM, the following present sufficient evidence to warrant concern.

6.1 Perinatal Death and Stillbirth

Women who develop GDM during their pregnancy are at an increased risk of pregnancy complications including stillbirth and perinatal mortality (Shand, Bell, McElduff, Morris, & Roberts, 2008). A recent cohort study found that the odds of perinatal death were increased by 30% for infants born at term by a mother with GDM (Billonnet et al., 2017) compared to

nondiabetic mothers. However, when controlling for pregnancies that were diagnosed as GDM postnatally, this risk was only seen in pregnancies that went untreated for GDM (Billonnet et al., 2017). Furthermore, studies conducted in Canada and Sweden found that the risk of perinatal death during a GDM pregnancy was significantly lower or similar to nondiabetic pregnancies when the mother was treated with a GDM intervention (Fadl et al., 2010; Feig et al., 2014). Yet controversy continues for whether an association occurs between GDM and perinatal mortality. Relatedly, there is some discussion in the literature regarding the relationship between GDM and still birth. This relationship was recognized as early as the 1960s, where O'Sullivan and colleagues (1966) observed an increased incidence of still birth for women who went undiagnosed or were inadequately treated for GDM. However, as screening, diagnosis, and treatment have become more consistent, the association between GDM and stillbirth has become more complicated. Nonetheless, a recent study found that women with GDM are at greater risk of experiencing a stillbirth after 35 weeks than their nondiabetic counterparts (Rosenstein et al., 2012), a risk that persisted until 42 weeks gestation. Supporting evidence suggests that women with GDM are 1.25 times more likely to experience a stillbirth after 28 weeks compared to mothers with normal glucose tolerance (Hutcheon, Kuret, Joseph, Sabr, & Lim, 2013). Together, the findings imply that there is some risk of fetal mortality due to GDM; however, these associations are likely reliant on mismanaged care. Therefore, mothers with GDM should be monitored closely for fetal health and development throughout pregnancy.

6.2 Fetal Macrosomia

Gestational diabetes and elevated fasting plasma glucose levels during pregnancy are reported to be significant risk factors for macrosomia (Shi et al., 2014; Turkmen et al., 2018), with even relatively mild hyperglycemia being associated with a significant increase in macrosomia (Zawiejska, Wender-Ozegowska, Radzicka, & Brazert, 2014). Due to the inability for insulin to cross the placenta, a fetus must secrete insulin independently in order to combat the high glucose levels that characterize GDM (Sweet, Grayson, & Pollack, 2013). The fetus develops hyperinsulinemia as a means of combatting this overabundance of glucose crossing the placenta, resulting in macrosomia (Pederson, 1967). Macrosomia, or a birth weight greater than 4000 grams regardless of gestational age (Costa, Paulinelli, & Barbosa, 2012), is the main cause of acute perinatal complications for mother and infant. Macrosomic infants are more likely to experience shoulder dystocia (Robinson et al., 2003; Mission, Ohno, Cheng, & Caughey, 2012), are more likely to experience deprivation of adequate oxygen levels (hypoxia) (Johnson & Schoeni, 2011), are at an increased risk of neonatal, post-natal, and infant death (Boulet et al., 2003), and at increased risk of childhood obesity later in life (Morton, 2006), compared to infants of normal weight.

Fetuses from diabetic pregnancies become macrosomic due to a unique pattern of overgrowth that occurs from subcutaneous fat accumulation in the abdominal and interscapular areas (McFarland, Trylovich, & Langer, 1998). Their birth weight correlates with the second and third trimester post meal blood levels rather than fasting glucose levels (Jovanovic-Peterson et al.,

1991). In terms of direct effects on the fetus, being large for gestational age is associated with shoulder dystocia (Boulvain et al., 2015; Irion & Boulvain, 1998; Lurie, Insler, & Hagay, 1996). This risk decreases if the mother is induced at 38-39 weeks gestation (Lurie et al., 1996). Similarly, many studies find an increased risk of obstetric brachial plexus injury and neonatal fractures in pregnancies with macrosomia compared to normal gestational weight pregnancies (Beta et al., 2019; Kin, Korst, Miller, & Ouzounian, 2012; Morikawa et al., 2013). Apgar scores, or the overall health of an infant after delivery, are often found to be lower in infants born with a larger than gestational age weight (Raio et al., 2003; Turkmen et al., 2018). This is troubling, as Apgar scores indicate the vitality and well-being of a newborn (Turkmen et al., 2018) and low scores (i.e., a score below seven) at five minutes is associated with increased risk of neurologic disability and even neonatal death (Ehrenstein, 2009). Therefore, not only are infants at risk of injury, but they are also at risk of decreased health and well-being immediately after birth that may result in poor health in the long-term. These findings demonstrate a strong association between GDM and increased risk of injury and poor health in infants.

6.3 Hypoglycemia

Neonatal hypoglycemia is a metabolic abnormality that is characterized by the inability of a newborn to maintain glucose homeostasis (De et al., 2011). A plasma glucose level of less than 30 mg/dl or 1.65 mmol/l in the first 24 hours of life (Stomnaroska-Damcevski et al., 2015) is generally accepted as neonatal hypoglycemia. Hypoglycemia occurs in approximately 8-30% of infants born to mothers with GDM versus 3% of non-diabetic women (Alemu, Alayinka, Baydoun, Hoch, & Akpinar-Elsi, 2017; Ferrara et al., 2007; Metzger et al., 2014; Rozance & Hay, 2006; Sarkar et al., 2003). However, the literature presents inconsistent findings as to whether neonatal hypoglycemia is related to poor maternal glycemic control, neonatal weight at birth, or gestational age at delivery (Alemu et al., 2017). This metabolic abnormality is associated with shakiness, tachycardia, lethargy, and temperature irregularities (Ramos et al., 2012; Vannucci & Vannucci, 2001). Glucose homeostasis is crucial for the overall physical development of a newborn (Rozance & Hay, 2006). If not detected early, prolonged neonatal hypoglycemia is associated with poor health outcomes including seizure, coma, cyanotic episodes, apnea, bradycardia or respiratory distress, hypothermia (Burden, Botiu, & Teodorescu, 2009; Najati & Saboktakin, 2010), and neurological injury (Alemu et al., 2017; Ramos et al., 2012). With the issues associated with neonatal hypoglycemia, attention should be given to infants of diabetic mothers. Therefore, newborns of GDM mothers should be tested for plasma glucose levels and monitored closely to ensure glucose homeostasis occurs prior to discharge.

6.4 Weight Gain

Studies have revealed that overweight, obesity, and maternal weight gain during pregnancy not only increases a mother's risk for developing GDM, but it also increases the risk of overweight/obesity in the offspring (Crume et al., 2011; Lawlor, Lichtenstein, & Langstrom, 2011). Clausen et al. (2009) examined the relationship of GDM and an offspring's risk of becoming overweight and found that adult offspring of women with diet treated GDM were

two times more likely to be overweight than adult offspring from a nondiabetic pregnancy. Further supporting the relationship between GDM and offspring risks of becoming overweight, Boerschmann and colleagues (2010) found that children born from GDM pregnancies were more likely to be overweight at ages 2, 8, and 11 than children from non-diabetic pregnancies. These findings suggest that exposure to hyperglycemia during pregnancy may partly explain the increased prevalence of overweight in children of a GDM pregnancy. However, some experts argue that it is not GDM that results in overweight offspring, but rather the mother's weight during pregnancy (Catalano & Ehrenberg, 2006) or common lifestyle factors within the family (Nilsson, Carlsson, & Landin-Olsson, 2013) that may be the greatest risk factors contributing to an offspring's likelihood to be overweight or obese. Furthermore, a systematic review of the literature warned that conclusions could not be drawn to suggest the later life obesity is truly caused by in utero exposure to hyperglycemia, due to the lack of consistent findings within the literature (Kim, England, Sharma, & Njoroge, 2011). Therefore, while there is a lack of consensus within the literature regarding the influence of exposure to hyperglycemia and its effects on the fetus in terms of long-term weight gain, there is a consensus that mothers should eat a healthy, well-balanced diet and provide similar healthy choices to their children. It is hoped that through a healthy lifestyle, women will keep a healthy weight, decrease their risk of developing GDM during pregnancy, and project these healthy behaviours toward the children.

6.5 Diabetes

There is evidence to suggest that GDM may have lasting effects on the fetus, specifically in terms of increasing the offspring's risk of developing diabetes later in life (Dabelea & Crume, 2011; Reusens, Ozanne, & Remacle, 2007). Parental diabetes is consistently shown to be a substantial risk factor in the development of adult onset diabetes (Alcolado & Alcolado, 1991; Lindsay et al., 2000; Martin et al., 1985; Thomas et al., 1994). A longitudinal study demonstrated that offspring born to one parent with diabetes, regardless of gender, increased the likelihood of developing type 2 diabetes 3.5-fold, with offspring of mothers diagnosed with diabetes greatly elevated this risk to 9.7-fold (Meigs et al., 2000). In utero exposure to diabetes appears to greatly affect the chances of offspring developing type 2 diabetes later in life. For example, the risk of offspring diabetes was significantly higher in siblings born after a mother's diagnosis of type 2 diabetes compared to siblings born before the mother developed type 2 diabetes (Dabelea, 2000). Due to the similarities between type 2 diabetes and GDM, these findings suggest that similar effects would be seen in offspring of a mother diagnosed with GDM. This is because GDM exposes the fetus to a proinflammatory environment, which may influence the fetal epigenome (Fernandez-Morera, 2010; Kwak & Park, 2016). This relationship appears to be consistent when looking at the children born to mothers diagnosed with GDM. Garcia-Vargas and colleagues (2012) showed that at 20 years the cumulative risk of developing type 2 diabetes for the offspring of women diagnosed with GDM was 20% and Blotsky and colleagues (2019) reported GDM in a sample of over 73,000 women in Canada was associated with a higher rate of pediatric type 2 diabetes in their offspring.

A Pima Indian study examined the effects of impaired glucose tolerance in the third trimester of pregnancy and found that young adults who were born to mothers with impaired glucose tolerance in pregnancy were at a greater risk of developing type 2 diabetes (Franks et al., 2006). By 20 years old the offspring were at a 15% risk of developing type 2 diabetes and this risk increased to 30% by the age of 24 years (Franks et al., 2006). Lastly, a Danish study found that interuterine hyperglycemia is associated with type 2 diabetes in adult offspring (Clausen et al., 2008). Specifically, the offspring of women with GDM were eight times more likely to develop diabetes/pre-diabetes (Clausen et al., 2008).

6.6 Autism

Exposure to maternal hyperglycemia may have lasting effects on a fetus's organ development and function (Freinkel, 1980). Autism spectrum disorders (ASD) are neurodevelopmental disorders that are characterized by impairments in social interactions, communications, and restricted, repetitive behaviours (American Psychiatric Association, 2013). Some evidence suggests that maternal diabetes during pregnancy may be associated with a child's development of ASD (Cargener, Spiegelman, & Buka, 2009; Xu, Jing, Bowers, Liu, & Bao, 2014), with recent studies suggesting a similar finding between GDM and ASD (Xiang, Wang, & Martinez, 2015). Importantly, Xiang et al. (2015) found that timing of treatment of GDM was an important factor in the risk of development of ASD in the child. The authors suggest that this relationship may be the result of exposure to untreated hyperglycemia during early critical brain development. Furthermore, Sacks et al. (2016) found a significant linear association between the severity of GDM and the risk of neuropsychiatric disorders, including ASD, in children. This association was proposed to be attributed to the in utero exposure to imbalanced glucose levels during the development of the central nervous system or the indirect effect of epigenetics (Sacks et al., 2016). However, because these findings are recent and limited in support, more research is necessary before concrete conclusions can be made regarding the relationship between ASD and GDM.

6.7 Cardiovascular Disease

Studies have determined a relationship between GDM and blood pressure of offspring (Boney, Verma, Tucker, & Vohr, 2005; Tam et al., 2008). However, other associations between cardiovascular disease in offspring and GDM are limited. Most of the research conducted on the effects of maternal hyperglycemia disorders and cardiovascular risks in offspring are conducted among mothers with type 1 or type 2 diabetes rather than GDM (Di Bernardo et al., 2017). Recently, experts have begun to examine the risks associated specifically with GDM and offspring cardiovascular outcomes to determine whether differences exist. A recent study found that offspring of a mother that was treated for GDM by diet and exercise were at a greater risk of developing cardiovascular disease in later life than offspring of mothers treated for GDM by insulin (Leybovitz-Haleluya, Wainstock, Landau, & Sheiner, 2018). Further, offspring of mothers with GDM are also reported to have higher systolic blood pressure than offspring of mothers who were non-diabetic during pregnancy (Bunt, Tataranni, & Salbe, 2005). However, a more recent study found that this association could only be found in male offspring and may be

influenced by maternal pre-pregnancy BMI (Aceti et al., 2012). Therefore, it appears that a relationship between GDM and an offspring's likelihood of developing cardiovascular disease in later life may be of concern, yet further research is needed within the context of GDM pregnancies.

7. Screening & Treatment

While several management strategies exist to lessen the effects of GDM, an efficacious prevention strategy has yet to be found. This lack of preventative measures is due to the complexity of GDM. The following strategies described below are methods of identification, and interventions commonly prescribed by healthcare providers as a means of controlling glucose levels in women with GDM.

7.1 Screening & Diagnosis

Screening for GDM has had a checkered past. Questions are raised as to whether GDM should be screened at all, if screening should be based on risk factors only, what screening methods are the best approach, and how well screening methods detect GDM (Agarwal, 2016). Many countries use selective screening criteria to determine whether GDM screening should take place. Risk factors that may suggest the need for GDM screening include a mother's BMI of greater than 30 kg/m2, previous GDM diagnosis, previous macrosomia, family history of diabetes mellitus, and ethnicity with a high prevalence of diabetes mellitus (WHO, 2016). This approach to determine whether screening for GDM is necessary results in a subset of mothers who do not receive appropriate care throughout their pregnancy (Teh et al., 2011), especially since GDM is asymptomatic (Chan, Wong, & Ho, 2008). Although a multitude of screening methods have been used (e.g., direct glucose measurements, indirect glucose measurements, and markers of diabetes), the gold standard of screening for GDM has yet to be found, which has resulted in a lack of international consensus regarding which test to use, the timing of screening method, and the optimal cut-off points for diagnosis (Rani & Begum, 2016). Currently, the glucose challenge test (GCT) and fasting plasma glucose (FPG) test are believed to be the best predictor of GDM (Agarwal, 2016). However, WHO recommends utilizing the oral glucose tolerance test (OGTT); a test that is utilized by the Canadian healthcare system.

GDM is diagnosed at any point throughout a pregnancy; however, the typical time frame of diagnosis is between 24- and 28-weeks gestation (WHO, 2016). Placental hormones may mediate insulin resistance, which increases GDM while the pregnancy progresses (Rani & Begum, 2016). Therefore, testing early on in the pregnancy may not be helpful. In Canada, the current screening strategy to detect whether a mother has GDM includes a two-hour 75 g oral glucose tolerance test. Abnormal glucose tolerance is diagnosed at any time throughout the pregnancy if at least one of the following criteria are met: a fasting plasma glucose level of 5.1-6.9mmol/L; 1-hour plasma glucose of 10.0mmol/L following a 75 g oral glucose load; 2-hour plasma glucose of 8.5-11mmol/L following a 75 g oral glucose load (WHO, 2016). Similarly, diabetes mellitus, better known as type 1 or type 2 diabetes, is diagnosed if at least one of the

following criteria are met using the same screening strategy: fasting plasma glucose of 7.0 mmol/L; 2-hour plasma glucose of 11.1 mmol/L following a 75 g oral glucose load; random plasma glucose 11.1 mmol/L in the presence of diabetes symptoms (WHO, 2016).

7.2 Lifestyle Interventions

Managing GDM appropriately may result in fewer maternal and fetal complications during and after pregnancy (Buchanan, Xiang, & Page, 2012). Most women diagnosed with GDM can be prompted to effectively manage their glucose levels through a lifestyle intervention comprising dietary counselling, encouragement of physical activity, and monitoring blood glucose levels (Lapolla, Dalfra, & Fedele, 2009). Prenatal nutrition and enhanced physical activity are the key interventions for glycemic control as a means of GDM treatment (Feig et al., 2018; Gutierrez & Reader, 2005) where the goal of a medically prescribed diet in GDM patients is to establish a diet that meets the normal maternal weight gain and fetal growth, optimize glycemic control, avoid ketoacidosis, and reduce glucose levels after meals (Lapolla et al., 2009). A meta-analysis examining 18 randomized controlled trials suggested that a modified dietary intervention decreased fasting and postprandial glucose and lowered the need for medication treatment for mothers with GDM (Yamamoto et al., 2018). Although the literature fails to present an agreed upon dietary recommendation applicable to all mothers with GDM, a daily caloric intake and distribution model based on the American Diabetes Association's recommendations for managing type 2 diabetes was developed (Lapolla et al., 2009; see Table 2). The recommendations were modified to incorporate dietary needs for fetal development, specifically the increased need for additional carbohydrates that promote fetal brain development. Pregnant mothers with GDM are also advised to be conscious of the glycemic index of their consumed carbohydrates. Low glycemic index foods are suggested due to the spike in glucose levels post meal (Clapp, 1998).

Table 2. Recommended calorie intake and nutrient distribution in GDM women

BMI	**Calories (kcal/kg of actual weight)**	**Caloric distribution (% of calories/meal)**
<19.8	36-40 kcal/kg of weight	Breakfast 10%-15%
19.8-26	30 kcal/kg of weight	Snack 5%-10%
26.1-33	24 kcal/kg of weight	Lunch 20%-30%
>33	12-18 kcal/kg of weight	Snack 5%-10%
		Dinner 30%-40%
+340-452 kcal in 2nd and 3rd trimester		Snack 5%-10% (25g CHO + 10g P)
Nutrient distribution (% of calories/nutrient)		
Complex carbohydrates and fiber: 45%-50% Protein: 15%-20% Mono and polyunsaturated fats: 30%-35%		

(Lapolla, Dalfra, & Fedele, 2009)

With the habitual consumption of coffee among many women of reproductive age, concerns regarding the safety of caffeine during pregnancy has been a topic of relevance within the healthcare field. Although much of these concerns focus on the safety of caffeine exposure to the fetus, interest has also been directed towards the effects of coffee intake in relation to the development of GDM. Sparking this interest are the documented effects of caffeine in the type 2 diabetes literature. Specifically, caffeine is found to increase insulin resistance (Huxley et al., 2009) and decrease risk for type 2 diabetes in non-pregnant adults (van Dam & Hu, 2005). In a large prospective cohort study, moderate caffeine consumption during the first trimester was not associated with a change in risk of developing GDM (Hinkle, Laughon, Catov, Olsen, & Bech, 2014). Unlike type 2 diabetes, the consumption of caffeine did not significantly decrease the likelihood of developing GDM (Hinkle et al., 2014). Therefore, the literature suggests that moderate consumption of caffeine during pregnancy will have little to no effect on the likelihood of developing GDM for expectant mothers. However, pre-pregnancy consumption of 0.5-7 cups of coffee/week is found to be associated with a reduced risk of GDM (Adeney, Williams, Schiff, Qiu, & Sorensen, 2007).

Exercise counselling, along with nutritional modification, is commonly used as a first line therapy in stabilizing glucose levels during pregnancy (Padayachee & Coombes, 2015). Physical activity is recognized for its beneficial effects on insulin sensitivity outside of pregnancy (Goodyear & Kahn, 1998). This is because exercise stimulates glucose uptake in the muscle (Goodyear & Kahn, 1998). However, during pregnancy, moderately-intense physical activity appears to only affect post-meal glucose levels (Ehrlich et al., 2017). The Society of Obstetricians and Gynecologists of Canada (SOGC) suggest that women with GDM follow the same physical activity guidelines as other pregnant women (Davies, Wolfe, Mottola, & MacKinnon, 2003). Specifically, 20-30 minutes of moderate-intensity exercise per day is believed to lower glucose levels in women with GDM (ACOG, 2015). What must be stressed, however, is the need to be aware of the immediate effects exercise may have on insulin sensitivity. To address this concern, Savvaki et al. (2018) suggest that women with GDM, especially those on insulin therapy, follow similar recommendations made for pregnant women with type 1 diabetes in regard to carbohydrate consumption prior to or at the onset of exercise (see Table 3). Nevertheless, exercise is part of a healthy, safe pregnancy and may provide multiple benefits to women diagnosed with GDM (see Table 4 for recommendations).

Table 3. Suggested carbohydrate intake or other actions based on blood glucose levels at the start of exercise

Pre-exercise blood glucose	Carbohydrate intake or other actions
< 90 mg/dl (< 5.0 mmol/l)	Ingest 15-30 g of fast-acting carbohydrate before the start of exercise, depending on the size of the individual and intended activity; some activities that are brief (< 30 min) or at a very high intensity (weight training, interval training) may not require any additional carbohydrate intake. For prolonged activities at moderate intensity, consume additional carbohydrate, as needed (0.5-1.0 g/kg body mass/h of exercise), based on blood glucose testing results.
90-150 mg/dl (5.0-8.3 mmol/l)	Start consuming carbohydrate at the onset of most exercise (0.5-1.0 g/kg body mass/h of exercise), depending on the type of exercise and the amount of active insulin.
150-250 mg/dl (8.3-13.9 mmol/l)	Initiate exercise and delay consumption of carbohydrate until blood glucose concentrations are < 150 mg/dl (< 8.3 mmol/l).
250-350 mg/dl (13.9-19.4 mmol/l)	Test for ketones. Do not perform any exercise if moderate-to-large amounts of ketones are present. Initiate mild-to-moderate intensity exercise. Intense exercise should be delayed until glucose concentrations are < 250 mg/dl. Intense exercise may exaggerate the hyperglycemia.
≥ 350 mg/dl (≥ 19.4 mmol/l)	Test for ketones. Do not perform any exercise if moderate-to-large amounts of ketones are present. If ketones are negative (or trace), consider conservative insulin correction (e.g., 50% correction) before exercise, depending on active insulin status. Initiate mild-to-moderate exercise and avoid intense exercise until glucose concentrations decrease.

(Savvaki et al., 2018)

Table 4. Exercise recommendations for GDM

	Aerobic	Resistance	Flexibility-Balance
Frequency	3-4 times/week	At least 2 times/week	2-3 times/week
Intensity	50-60% VO_{2max}	12-13 Borg's Scale	Stretch till slight discomfort Balance workout light to moderate
Time	45 min with 5-min breaks every 15 min	**ACSM recommendations*: 5-10 exercises 10-15 repetitions (1 set) ***ESSA recommendations*: 8-10 exercises 8-10 repetitions (2 sets)	Stretch 10-30 s, 2-4 repetitions/exercise
Type	Walking, jogging, running, elliptical machine, cycling, swimming, aqua-aerobics	Sitting position exercises, pilates, yoga, exercises with free weights, elastic band exercises, weight-bearing exercises	Yoga, pilates, tai chi, dynamic stretch, static stretch

**ACSM, American College of Exercise Medicine; **ESSA, Exercise and Sport Science Australia (Savvaki et al., 2018)*

7.3 Glucose Lowering Therapies

For women who do not achieve glycemic targets within two weeks of initiating lifestyle modifications, glucose lowering therapy may be used (Dhulkotia, Ola, Fraser, & Farrell, 2010; Feig et al., 2018; Moore, Clokey, Rappaport, & Curet, 2010; Rowan, Hague, Gao, Battin, & Moore, 2008). Although insulin injections may be effective in lowering glucose levels in many pregnant women, experts have begun to examine the effectiveness of prescribing alternative glucose lowering treatment agents, such as metformin, for the treatment of GDM. While metformin performs slightly better than insulin at regulating glucose levels (Balsells et al., 2015; Poolsup, Suksomboon, & Amin, 2014), metformin has yet to be approved for the treatment of GDM (Bowker et al., 2017).

Insulin is a hormone that increases cellular uptake, use, and storage of glucose in the tissues and liver cells and regulates fat storage within adipose cells (Gray, McGuire, Cohen, & Little, 2017). Women with GDM may be prescribed insulin as a means of controlling glucose levels during pregnancy; however, this form of therapy is typically recommended if nutrition therapy has failed to maintain target glucose levels (Duarte-Gardea, 2013). For women with GDM who require insulin, isophane, an intermediate-acting insulin, is preferred (Metzger et al., 2007). Research suggests that insulin therapy for women with GDM has the potential to be an equally effective intervention strategy for treating glucose intolerance to that of nutrition adjustment therapy (Brown, Grzeskowiak, Williamson, Downie, & Crowther, 2017). Rapid-acting insulin analogs appear to be safe for use during pregnancy, with some studies showing greater glycemic control compared to regular insulin therapy (Banerjee et al., 2009). Lispro, a type of

rapid-acting insulin analog, is of particular interest for GDM treatment because it is found to not cross the placenta except for when administered in very high dosages (Broskovic et al., 2003). Findings from a meta-analysis suggest that women with GDM that are treated with lispro were less likely to experience severe maternal hypoglycemia (Lv, Wang, & Xu, 2015); however, conflicting evidence is found on whether taking lispro may result in infant macrosomia (Lv, Wang, & Xu, 2015; Lapolla, Dalfra, & Fedele, 2005; Pettitt, Ospina, Kolaczynski, & Jovanovic, 2003). The amount of insulin to be administered for GDM is 0.6-0.8 U/kg body weight in the first trimester, 1.0U/kg body weight in the second trimester, and 1.2 U/kg body weight in the third trimester, with the total amount of insulin distributed in the morning (two thirds of the daily dose) and in the evening (one third of the daily dose) (Duarte-Gardea, 2013).

Due to side effects, such as increased appetite, weight gain, and hypoglycaemia, associated with the administration of insulin (Zhao et al., 2015), metformin is often prescribed alongside insulin to improve glycaemic control and reduce the needed insulin dose (Feig et al., 2016; Ibrahim et al., 2014; Vella et al., 2010). Metformin is an insulin sensitizer traditionally prescribed as a means of achieving ovulation for women with PCOS; yet since 2004, metformin has been tested as a possible intervention to mitigate GDM in early pregnancy (Vanky et al., 2004). However, there are currently observable risks associated with the use of metformin over insulin (e.g., slightly lower gestational age at delivery; Rowan et al., 2011) and it is unclear whether metformin meets the standards of a GDM intervention due to the inconclusive results between randomized control trials and observational studies. A recent meta-analysis of randomized control trials examining the effectiveness of metformin as a prevention intervention of GDM suggests that metformin provides little contribution to averting GDM in women at high risk (Doi et al., 2020). Additional risks have been identified for the use of metformin during pregnancy. For example, several studies have found that compared to those who had taken insulin, women who take metformin are at an increased risk of preterm delivery (Balsells et al., 2015). Metformin is also known to cross the placenta, posing possible long-term effects on children exposed to metformin in utero (Blair, Rosenberg, & Palermo, 2019), although little evidence has been found. Nonetheless, the findings from combining insulin and metformin together as a treatment for GDM are promising. Recent findings suggest that improved glycaemic control towards the end of a pregnancy may be possible with the concurrent use of insulin and metformin, showing more favourable results than did insulin alone (Balsells et al., 2015). Therefore, metformin alone may not adequately address the need for a method to prevent GDM in at-risk women, but together metformin and insulin may lower the risks of GDM.

8. Conclusion

With the rise of obesity and type 2 diabetes, rates of gestational diabetes mellitus are expected to continue to rise in the next decade. This is alarming because GDM is associated with serious perinatal complications for both the mother and the child. Mothers who develop GDM during their pregnancy are more likely to suffer from preeclampsia during pregnancy, experience a more

traumatic delivery, and are at greater risk of developing type 2 diabetes after pregnancy. Specifically, during delivery, women with GDM are more likely to deliver an infant that is large for gestational age, which can result in prolonged labour, need for instrumental delivery, experiencing perineal tearing, genital tract injury and uterine atony, requiring an unplanned caesarean section, having a postpartum hemorrhage, or dying from childbirth. In addition to these grave concerns for the well-being of mothers, there are many short- and long-term risks for the infant. These risks include perinatal death and stillbirth, macrosomia, hypoglycemia, childhood and adulthood weight gain, type 1 or type 2 diabetes, and cardiovascular disease.

Although there is no international standard for screening women for GDM, Canada has implemented the glucose challenge test advised by WHO. Pregnant Canadian women, especially at-risk mothers, are advised to take this screening test between 24 and 28 weeks gestation as part of an early detection approach to prenatal care. Prevention, early detection, and treatment of GDM are instrumental in combatting the associated health risks of GDM. Women who are older, of non-white descent, have PCOS, are pregnant with a male fetus, and/or have one or more parent with diabetes are at the greatest risk of developing GDM. However, behavioural risk factors such as smoking, overweight/obesity, poor nutrition, insufficient sleep, low physical activity, and low iron and vitamin D levels are nearly as instrumental as uncontrollable factors in putting women at risk of developing GDM. This means that women should be screened for GDM regardless of their biological risk.

Lifestyle changes may reduce the risk of developing GDM in at-risk mothers; however, intervention studies have shown mixed results in the efficacy of lifestyle changes for the prevention or treatment of GDM. Nevertheless, changes such as taking part in multiple forms of exercise for approximately 30 minutes a day; eating a well-balanced diet rich in fruits and vegetables, plant protein, and whole grains, and limited in processed food; taking a probiotic and vitamin D supplement; and sleeping approximately 8 hours each night, may provide some benefit to at-risk women. Although many women make a point to change their lifestyle during pregnancy, experts suggest incorporating preventative measures pre-pregnancy. For those who continue to struggle with glucose control, insulin and metformin may provide some additional aid in glucose regulation. In conclusion, there are clear risks for both the mother and child associated with developing GDM, and there are recommendations for both the prevention and management of GDM, but the evidence for their effectiveness is not strong and more research is needed in determining the best means of addressing the increasing rates of GDM.

References

Aceti, A., Santhakumaran, S., Logan, K. M., Philips, L. H., Prior, E., … Modi, N. (2012). The diabetic pregnancy and offspring blood pressure in childhood: A systematic review and meta-analysis. *Diabetologia, 55*, 3114-3127. https://doi.org/10.1007/s00467-019-4201-x

Adeney, K. L., Williams, M. A., Schiff, M. A., Qiu, C., & Sorensen, T. K. (2007). Coffee consumption and the risk of gestational diabetes mellitus. *Acta Obstetricia et Gynecologica Scandinavica, 86*, 161-166. https://doi.org/10.1080/00016340600994992

Agarwal, M. M. (2016). Gestational diabetes mellitus: Screening with fasting plasma glucose. *World Journal of Diabetes, 7*(14), 279-289. https://doi.org/10.4239/wjd.v7.i14.279

Agha-Jaffar, R., Oliver, N., Johnston, D., & Robinson, S. (2016). Gestational diabetes mellitus: Does an effective prevention strategy exist? *Nature Reviews, Endocrinology, 12*(3), 533-546. https://doi.org/10.1038/nrendo.2016.88.

Alcolado, J. C., & Alcolado, R. (1991). Importance of maternal history of non-insulin dependent diabetic patients. *British Medical Journal, 302*, 1178-1180. https://doi.org/10.1159/000052916

Alemu, B. T., Olayinka, O., Baydoun, H. A., Hoch, M., & Akpinar-Elci, M. (2017). Neonatal hypoglycemia in diabetic mothers: A systematic review. *Current Pediatric Research, 21*(1), 42-53. Retrieved from https://digitalcommons.odu.edu/commhealth_fac_pubs/25

Aljohani, N., Rempel, B. M., Ludwig, S., Morris, M., McQuillen, K., … Shen, G. X. (2008). Gestational diabetes in Manitoba during a twenty-year period. *Clinical and Investigative Medicine, 31*(3), E131-E137. https://doi.org/10.25011/cim.v31i3.3470

Almgren, P., Lehtovirta, M., Isomaa, B., Sarelin, L., Taskinen, M. R., … Groop, L. (2011). Heritability and familiality of type 2 diabetes and related quantitative traits in the Botnia Study. *Diabetologia, 54*(11), 2811-2819. https://doi.org/10.1007/s00125-011-2267-5

Altman, D., Carroli, G., Duley, L., Farrell, B., Moodley, J., Neilson, J., & Smith, D. (2002). Do women with pre-eclampsia, and their babies, benefit from magnesium sulphate? The Magpie Trial: A randomised placebo-controlled trial. *Lancet, 359*(9321), 1877-1890. https://doi.org/10.1016/s0140-6736(02)08778-0

Alvarez, J. A., Ashraf, A. (2010). Role of vitamin D in insulin secretion and insulin sensitivity for glucose homeostasis. *International Journal of Endocrinology, 2010*, 351385. https://doi.org/10.1155/2010/351385

Alzaim, M., & Wood, R. J. (2013). Vitamin D and gestational diabetes mellitus. *Nutrition Reviews, 71*(3), 158-167. https://doi.org/10.1111/nure.12018

American College of Obstetricians and Gynecologists [ACOG]. (2008). ACOG practice bulletin no. 95: Anemia in pregnancy. *Obstetrics and Gynecology, 112*, 201-207. https://doi.org/10.1097/AOG.0b013e3181809c0d

American College of Obstetricians and Gynecologists [ACOG]. (2013). Hypertension in pregnancy. Report of the American College of Obstetricians and Gynecologists' Task Force on Hypertension in Pregnancy. *Obstetrics and Gynecology, 122(5),* 1122-1131. https://doi.org/10.1097/01.AOG.0000437382.03963.88

American College of Obstetricians and Gynecologists [ACOG]. (2015). Physical activity and exercise during pregnancy and postpartum period. ACOG committee opinion 650. *Obstetrics and Gynecology, 126*(6), e135-e142. https://www.acog.org/Clinical-Guidance-and-Publications/Committee-Opinions/Committee-on-Obstetric-Practice/Physical-Activity-and-Exercise-During-Pregnancy-and-the-Postpartum-Period?IsMobileSet=false

American College of Obstetricians and Gynecologists [ACOG]. (2017). Practice bulletin no. 180: Gestational diabetes mellitus. *Obstetrics and Gynecology, 30*(1), e17-e37. https://doi.org/10.1097/AOG.0000000000002159

American Psychiatric Association [APA]. (2013). *Diagnostic and Statistical Manual of Mental Disorders*. 5th ed. Washington, DC: American Psychiatric Association.

Anand, S. S., Gupta, M., Teo, K. K., Schulze, K. M., Desai, D., … Gerstein, H. (2017). Causes and consequences of gestational diabetes in South Asians living in Canada: results from a prospective cohort study. *CMAJ Open, 5*(3), E604-E611. https://doi.org/10.9778/cmajo.20170027

Anna, V., van Der Pleog, H. P., Cheung, N. W., Huxley, R. R., & Bauman, A. E. Sociodemographic correlates of the increasing trend in prevalence of gestational diabetes mellitus in a large population of women between 1995 and 2005. *Diabetes Care, 3112*, 2288-2293. https://doi.org/10.2337/dc08-1038

Asemi, Z., Samimi, M., Tabassi, Z., Naghibi Rad, M., Rahimi Foroushani, A., Khorammian, H., & Esmaillzadeh, A. (2013). The effect of daily consumption of probiotic yoghurt on insulin resistance in pregnant women: a randomized controlled trial. *European Journal of Clinical Nutrition, 67*, 71-74. https://doi.org/10.1038/ejcn.2012.189

Ashrafi, M., Gosili, R., Hosseini, R., Arabipoor, A., Ahmadi, J., & Chehrazi, M. (2014). Risk of gestational diabetes mellitus in patients undergoing assisted reproductive techniques. *European Journal of Obstetrics and Gynecology and Reproductive Biology, 176*, 149-152. https://doi.org/10.1016/j.ejogrb.2014.02.009

Ashrafi, M., Sheikhan, F., Arabipoor, A., Hosseini, R., Nourbakhsh, F., & Zolfaghari, Z. (2014). Gestational diabetes mellitus risk factors in women with polycystic ovary syndrome (PCOS). *Obstetrics and Gynecology and Reproductive Biology,* 181, 195-199. https://doi.org/10.1016/j.ejogrb.2014.07.043

Ashrafi, M., Sheikhan, F., Arabipoor, A., Rouhana, N., Hosseini, R., & Zolfaghari, Z. (2017). Gestational diabetes mellitus and metabolic disorder among different phenotypes of polycystic ovary syndrome. *Oman Medical Journal, 32*(3), 214-220. https://doi.org/10.5001/omj.2017.40

Azziz, R., Carmina, E., Chen, Z., Dunaif, A., Laven, J. S., … Yildiz, B. O. (2016). Polycystic ovary syndrome. *Nature Reviews Disease Primers, 2*, 16057. https://doi.org/10.1038/nrdp.2016.57

Back, L., Hui, A., Reid, A., Sevenhuysen, G., Gardiner, P., … Shen, G. X. (2012). Comparison of physical activity and nutritional intake in First Nations pregnant women in remote communities and urban-living pregnant women. *Canadian Journal of Diabetes, 36*(2), 64-67. https://doi.org/10.1016/j.jcjd.2012.04.001

Balsells, M., Garcia-Patterson, A., Sola, I., Roque, M., Gich, I., & Corcoy, R. (2015). Glibenclamide, metformin, and insulin for the treatment of gestational diabetes: A systematic review and meta-analysis. *British Medical Journal, 350*, h102. https://doi.org/10.1136/bmj.h102

Balserak, B. I., Jackson, N., Ratcliffe, S. A., Pack, A. I., & Pien, G. W. (2013). Sleep-disordered breathing and daytime napping are associated with maternal hyperglycemia. *Sleep and Breathing, 17*, 1093-1102. https://doi.org/ 10.1007/s11325-013-0809-4

Banerjee, M., Bhattacharya, A., Hughes, S. M., & Vice, P. A. (2009). Efficacy of insulin lispro in pregnancies complicated with pregestational diabetes mellitus. *Practical Diabetes International, 26*(9), 366-70. https://doi.org/10.1002/pdi.1423

Bao, W., Bowers, K., Tobias, D. K., Hu, F. B., & Zhang, C. (2013). Pre-pregnancy dietary protein intake, major dietary protein sources, and the risk of gestational diabetes mellitus. *Diabetes Care, 36*(7), 2001-2008. https://doi.org/ 10.2337/dc12-2018

Bao, W., Michels, K. B., Tobias, D. K., Li, S., Chavarro, J. E., … Zhang, C. (2016). Parental smoking during pregnancy and the risk of gestational diabetes in the daughter. *International Journal of Epidemiology, 45*, 160-169. https://doi.org/10.1093/ije/dyv334

Barnes, R. A., Wong, T., Ross, G. P., Griffiths, M. M., Smart, C. E., … Flack, J. R. (2020). Excessive weight gain before and during gestational diabetes mellitus management: What is the impact? *Diabetes Care, 43*, 74-81. https://doi.org/ 10.2337/dc19-0800

Barquiel, B., Herranz, L., Grande, C., Castro-Dufourny, I., Llaro, M., … Pallardo, L. F. (2014). Body weight, weight gain and hyperglycaemia are associated with hypertensive disorders of pregnancy in women with gestational diabetes. *Diabetes and Metabolism, 40(3),* 204-210. https://doi.org/ 10.1016/j.diabet.2013.12.011

Bennett, C. J., Walker, R. E., Blumfield, M. L., Gwini, S. M., Ma, J., … Truby, H. (2018). Interventions designed to reduce excessive gestational weight gain can reduce the incidence of gestational diabetes mellitus: A systematic review and meta-analysis of randomized control trials. *Diabetes Research and Clinical Practice, 141*, 69-79. https://doi.org/ 10.1016/j.diabres.2018.04.010

Beta, J., Khan, N., Fiolna, M., Khalil, A., Ramadan, G., & Akolekar, R. (2019). Maternal and neonatal complications of fetal macrosomia: Cohort study. *Ultrasound in Obstetrics and Gynecology, 54*(3), 319-325. https://doi.org/10.1002/uog.20278

Blaire, R. A., Rosenberg, E. A., & Palermo, N. E. (2019). The use of non-insulin agents in gestational diabetes: Clinical considerations in tailoring therapy. *Current Diabetes Reports, 19*, 158. https://doi.org/10.1007/s11892-019-1243-1

Blaize, N., Pearson, K. J., & Newcomer, S. C. (2015). Impact of maternal exercise during pregnancy on offspring chronic disease susceptibility. *Exercise and Sport Sciences Reviews, 43*(4), 198-203. https://doi.org/ 10.1249/JES.0000000000000058

Blotsky, A. L., Rahme, E., Dahhou, M., Nakhla, M., & Dasgupta, K. (2019). Gestational diabetes associated with incident diabetes in childhood and youth: a retrospective cohort study. *Canadian Medical Association Journal, 191*(15), e410-e417. https://doi.org/10.1503/cmaj.181001

Boerschmann, H., Pfluger, M., Henneberger, L., Zielger, A. G., & Hummel, S. (2010). Prevalence and predictors of overweight and insulin resistance in offspring of mothers with gestational diabetes mellitus. *Diabetes Care, 33*(8), 1845-1849. https://doi.org/ 10.2337/dc10-0139

Bogaerts, A., Van den Bergh, B. R., Ameye, L., Witters, I., Martens, E., Timmerman, D., & Devlieger, R. (2013). Interpregnancy weight change and risk for adverse perinatal outcome. *Obstetrics and Gynecology, 122*(5), 999-1009. https://doi.org/10.1097/AOG.0b013e3182a7f63e.

Boney, C. M., Verma, A., Tucker, R., & Vohr, B. R. (2005). Metabolic syndrome in childhood: association with birth weight, maternal obesity, and gestational diabetes mellitus. *Pediatrics, 115*(3), e290-e296. https://doi.org/10.1542/peds.2004-1808

Boskovic, R., Feig, D. S., Derewlany, L., Knie, B., Portnoi, G., & Koren, G. (2003). Transfer of insulin lispro across the human placenta: In vitro perfusion studies. *Diabetes Care, 26*(5), 1390-1394. https://doi.org/10.2337/diacare.26.5.1390

Boulet, S. L., Alexander, G. R., Salihu, H. M., & Pass, M. (2003). Macrosomic births in the United States: Determinants, outcomes, and proposed grades of risk. *American Journal of Obstetrics and Gynecology, 188*(5), 1372-1378. https://doi.org/10.1067/mob.2003.302

Boulvain, M., Senat, M. V., Perrotin, F. Winer, N., Beucher, G., … Rozenberg, P. (2015). Induction of labour versus expectant management for large-for-date fetuses: A randomised controlled trial. *The Lancet, 385*(9987), 2600-2605. https://doi.org/ 10.1016/S0140-6736(14)61904-8

Bowers, K., Tobias, D. K., Yeung, E., Hu, F. B., & Zhang, C. (2012). A prospective study of pre-pregnancy dietary fat intake and risk of gestational diabetes. *American Journal of Clinical Nutrition, 95*(2), 446-453. https://doi.org/10.3945/ajcn.111.026294

Bowers, K., Yeung, E., Williams, M. A., Qi, L., Tobias, D. K., Hu, F. B., & Zhang, C. (2011). A prospective study of pre-pregnancy dietary iron intake and risk for gestational diabetes mellitus. *Diabetes Care, 34*(7), 1557-1563. https://doi.org/10.2337/dc11-0134

Bowker, S. L., Savu, A., Yeung, R. O., Johnson, J. A., Ryan, E. A., & Kaul, P. (2017). Patterns of glucose-lowering therapies and neonatal outcomes in the treatment of gestational diabetes in Canada, 2009-2014. *Diabetic Medicine, 34*(9), 1296-1302. https://doi.org/10.1111/dme.13394

Brown, J., Grzeskowiak, L., Williamson, K., Downie, M. R., & Crowther, C. A. (2017). Insulin for the treatment of women with gestational diabetes. *The Cochrane Database of Systematic Reviews, 2017*, CD012037. https://doi.org/10.1002/14651858.CD012037.pub2

Brunner, S., Stecher, L., Ziebarth, S., Nehring, I., Rifas-Shiman, S. L., … von Kries, R. (2015). Excessive gestational weight gain prior to glucose screening and the risk of gestational diabetes: A meta-analysis. *Diabetologica, 58*(10), 2229-2237. https://doi.org/10.1007/s00125-015-3686-5

Bryson, C. L., Ioannou, G. N., Rulyak, S. J., & Critchlow, C. (2003). Association between gestational diabetes and pregnancy-induced hypertension. *American Journal of Epidemiology, 158*(12), 1148-1153. https://doi.org/10.1093/aje/kwg273

Buchanan, T. A., & Xiang, A. H. (2005). Gestational diabetes mellitus. *Journal of Clinical Investigation, 115*(3), 485-491. https://doi.org/ 10.1172/JCI24531

Buchanan, T. A., Xiang, A. H., & Page, K. A. (2012). Gestational diabetes mellitus: Risks and management during and after pregnancy. *Nature Reviews, Endocrinology, 8*(11), 639-649. https://doi.org/10.1038/nrendo.2012.96

Bunt, J. C., Tataranni, P. A., & Salbe, A. D. (2005). Intrauterine exposure to diabetes is a determinant of hemoglobin A(1)c and systolic blood pressure in pima Indian children. *Journal of Clinical Endocrinology and Metabolism, 90*(6), 3225-3229. https://doi.org/10.1210/jc.2005-0007

Burdan, D. R., Botiu, V., & Teodorescu, D. (2009). Neonatal hypoglycemia - The incidence of the risk factors in Salvator Vuia obstetrics-gynecology hospital, Arad. *Timisoara Medical Journal, 59*, 5. Retrieved from http://tmj.ro/pdf/2009_number_1_7722108355124753.pdf

Burris, H. H., & Camargo, C. A. (2014). Vitamin D and gestational diabetes mellitus. *Current Diabetes Reports, 14*(1), 451-457. https://doi.org/ 10.1007/s11892-013-0451-3

Bushnell, C., McCullough, L. D., Awad, I. A., Chireau, M. V., Fedder, W. N., … Council for High Blood Pressure Research. (2014). Guidelines for the prevention of stroke in women: a statement for healthcare professionals from the American Heart Association/American Stroke Association. *Stroke, 45*(5), 1545-1588. https://doi.org/10.1161/01.str.0000442009.06663.48

Cai, S., Tan, S., Gluckman, P. D., Godfrey, K. M., Saw, S., … Gooley, J. J. (2017). Sleep quality and nocturnal sleep duration in pregnancy and risk of gestational diabetes mellitus. *Sleep*, *40*(2), zsw058. https://doi.org/10.1093/sleep/zsw058

Callaway, L. K., McIntyre, H. D., Barrett, H. L., Foxcroft, K., Tremellen, A., … Nitert, M. D. (2019). Probiotics for the prevention of gestational diabetes mellitus in overweight and obese women: Findings from the SPRING double-blinded randomized controlled trial. *Diabetes Care, 42*(3), 364-371. https://doi.org/10.2337/dc18-2248

Carolan, M., Davey, M. A., Biro, M. A., & Kealy, M. (2012). Maternal age, ethnicity, and gestational diabetes mellitus. *Midwifery, 28*(6), 778-783. https://doi.org/

Catalano, P. M., Farrell, K., Thomas, A., Huston-Presley, L., Mencin, P., de Mouzon, S. H., & Amini, S. B. (2009). Perinatal risk factors for childhood obesity and metabolic dysregulation. *American Journal of Clinical Nutrition, 90*(5), 1303-1331. https://doi.org/10.1016/j.midw.2011.08.014

Catalano, P. M., Tyzbir, E. D., Roman, N. M., Amini, S. B., & Sims, E. A. (1991). Longitudinal changes in insulin release and insulin resistance in nonobese pregnant women. *American Journal of Obstetrics and Gynecology, 165*(6), 1667-1672. https://doi.org/10.1016/0002-9378(91)90012-G

Chamberlain, C., Banks, E., Joshy, G., Diouf, I., Oats, J. J., Gubhaju, L., & Eades, S. (2014). Prevalence of gestational diabetes mellitus among indigenous women and comparison with non-indigenous Australian women: 1990-2009. *The Australian & New Zealand Journal of Obstetrics and Gynaecology, 54*(5), 433-440. https://doi.org/10.1111/ajo.12213

Chamberlain, C., Joshy, G., Li, H., Oats, J., Eades, S., & Banks, E. (2015). The prevalence of gestational diabetes mellitus (GDM) among Aboriginal and Torres Strait Islander women in Australia: A systematic review and meta-analysis. *Diabetes Metabolism Research and Reviews, 31*(3), 234-247. https://doi.org/10.1002/dmrr.2570

Chan, K. K., Chan, B. C., Lam, K. F., Tam, S., & Lao, T. T. (2009). Iron supplement in pregnancy and development of gestational diabetes – a randomised placebo-controlled trial. *British Journal of Obstetrics and Gynecology, 116*(6), 789-798. https://doi.org/10.1111/j.1471-0528.2008.02014.x

Chan, L. Y. S., Wong, S. F., & Ho, L. C. (2008). Diabetic family history is an isolated risk factor for gestational diabetes after 30 years of age. *Acta Obstetricia et Gynecologica Scandinavia, 81*(2), 115-117. https://doi.org/10.1034/j.1600-0412.2002.810205.x

Chen, L., Hu, F. B., Yeung, E., Willett, W., & Zhang, C. (2009). Prospective study of pre-gravid sugar-sweetened beverage consumption and the risk of gestational diabetes mellitus. *Diabetes Care*, *32*(12), 2236-2241. https://doi.org/10.2337/dc09-0866

Chen, Y., Quick, W. W., Yang, W., Zhang, Y., Baldwin, A., … Dall, T. M. (2009). Cost of gestational diabetes mellitus in the United States in 2007. *Population Health Management, 12*(3), 165-171. https://doi.org/10.1089/pop.2009.12303

Cheung, N. W., & Byth, K. (2003). Population health significance of gestational diabetes. *Diabetes Care, 26*(7), 2005-2009. https://doi.org/10.2337/diacare.26.7.2005

Cho, Y. M., Kim, T. H., Lim, S., Choi, S. H., Shin, H. D., ... Jang, H. C. (2009). Type 2 diabetes-associated genetic variants discovered in the recent genome-wide association studies are related to gestational diabetes mellitus in the Korean population. *Diabetologia, 52*(2), 253-261. https://doi.org/10.1007/s00125-008-1196-4

Chu, S. Y., Callaghan, W. M., Kim, S. Y., Schmid, C. H., Lau, J., England, L. J., & Dietz, P. M. (2007). Maternal obesity and risk of gestational diabetes mellitus. *Diabetes Care, 30*(8), 2070-2076. https://doi.org/10.2337/dc06-2559a

Clapp, J. F. (1998). Effect of dietary carbohydrate on the glucose and insulin response to mixed caloric intake and exercise in both nonpregnant and pregnant women. *Diabetes Care, 21*(2), B107-B112. Retrieved from https://search.proquest.com/docview/223025339?accountid=12378

Clausen, T. D., Mathiesen, E. R., Hansen, T., Pederson, O., Jensen, D. M., … Damm, P. (2009). Overweight and the metabolic syndrome in adult offspring of women with diet-treated gestational diabetes mellitus or type 1 diabetes. *Journal of Clinical Endocrinology and Metabolism, 94*(7), 2464-2470. https://doi.org/10.1210/jc.2009-0305

Clausen, T. D., Mathiesen, E. R., Hansen, T., Pedersen, O., Jensen, D. M., Lauenborg, J., & Damm, P. (2008). High prevalence of type 2 diabetes and pre-diabetes in adult offspring of women with gestational diabetes mellitus or type 1 diabetes: The role of intrauterine hyperglycemia. *Diabetes Care, 31*(2), 340-346. https://doi.org/10.2337/dc07-1596

Clifton-Bligh, R. J., McElduff, P., & McElduff, A. (2008). Maternal vitamin D deficiency, ethnicity and gestational diabetes. *Diabetic Medicine, 25*(6), 678-684. https://doi.org/10.1111/j.1464-5491.2008.02422.x

Cockburn, F., Belton, N. R., Purvis, R. J., Giles, M. M., Brown, J. K., … Pocock, S. J. (1980). Maternal vitamin D intake and mineral metabolism in mothers and their newborn infants. *British Medical Journal, 281*(11), 11-14. https://doi.org/10.1136/bmj.281.6232.11

Colberg, S. R., Castorino, K., & Jovanovič, L. (2013). Prescribing physical activity to prevent and manage gestational diabetes. *World Journal of Diabetes, 4*(6), 256-262. https://doi.org/10.4239/wjd.v4.i6.256

Costa, B. M., Paulinelli, R. R., & Barbosa, M. A. (2012). Association between maternal and fetal weight gain: Cohort study. *Sao Paulo Medical Journal, 130*(4), 242-247. https://doi.org/10.1590/S1516-31802012000400007

Crane, S. S., Wojtowycz, M. A., Dye, T. D., Aubry, R. H., & Artal, R. (1997). Association between pre-pregnancy obesity and the risk of caesarean delivery. *Obstetrics and Gynecology, 89*(2), 213-216. https://doi.org/10.1016/S0029-7844(96)00449-8

Crume, T. L., Ogden, L., Daniels, S., Hamman, R. F., Norris, J. M., & Dabelea, D. (2011). The impact of in utero exposure to diabetes on childhood body mass index growth trajectories: the EPOCH study. *The Journal of Pediatrics, 158*(6), 941-946. https://doi.org/10.1016/j.jpeds.2010.12.007

Dabelea, D. (2007). The predisposition to obesity and diabetes in offspring of diabetic mothers. Diabetes Care, 30, S169-S174. https://doi.org/10.2337/dc07-s211

Dabelea, D., & Crume, T. (2011). Maternal environment and the transgenerational cycle of obesity and diabetes. *Diabetes, 60*(7), 1849-1855. https://doi.org/10.2337/db11-0400

Dabelea, D., Hansen, R. L., Lindsay, R. S., Pettitt, D. J., Imperatore, G., … Knowler, W. C. (2000). Intrauterine exposure to diabetes conveys risks for type 2 diabetes and obesity: a study of discordant sibships. *Diabetes, 49*(12), 2208-2211. https://doi.org/10.2337/db11-0400

Darling, A. M., Mitchell, A. A., & Werler, M. M. (2016). Preconceptional iron intake and gestational diabetes mellitus. *International Journal of Environmental Research Public Health, 13*(6), E525. https://doi.org/10.3390/ijerph13060525

Davenport, M. H., Mottola, M. F., & McManus, R. (2008). A walking intervention improves capillary glucose control in women with gestational diabetes mellitus: A pilot study. *Applied Physiology, Nutrition, and Metabolism, 33*(3), 511-517. https://doi.org/10.1139/H08-018

Davies, G., Wolfe, L., Mottola, M., & MacKinnon, C. (2003). Joint SOGC/CSEP clinical practice guideline: Exercise in pregnancy and the postpartum period. *Canadian Journal of Applied Physiology, 28*(3), 330-341. https://doi.org/ 10.1139/h03-024

De, A. K., Biswas, R., Samanta, M., & Kundu, C. K. (2011). Study of blood glucose level in normal and low birth weight newborns and impact of early breast feeding in a tertiary care centre. *Annals of Nigerian Medicine, 5*(2), 53-58. Retrieved from http://www.anmjournal.com/text.asp?2011/5/2/53/92951

Dempsey, J. C., Sorensen, T. K., Williams, M. A., Lee, I. M., Miller, R. S., Dashow, E. E., & Luthy, D. A. (2004). Prospective study of gestational diabetes mellitus risk in relation to maternal recreational physical activity before and during pregnancy. *American Journal of Epidemiology, 159*(7), 663-670. https://doi.org/ 10.1093/aje/kwh091

Dempsey, J. A., Veasey, S. C., Morgan, B. J., & O'Donnell, C. P. (2010). Pathophysiology of sleep apnea. *Physiological Reviews, 90*(1), 47-112. https://doi.org/ 10.1152/physrev.00043.2008

de Munter, J. S., Hu, F. B., Spiegelman, D., Franz, M. & van Dam, R. M. (2007) Whole grain, bran, and germ intake and risk of type 2 diabetes: A prospective cohort study and systematic review. *PLoS Medicine, 4*(8), 1385-1395. https://doi.org/10.1371/journal.pmed.0040261

Dhulkotia, J. S., Ola, B., Fraser, R., & Farrell, T. (2010). Oral hypoglycemic agents vs insulin in management of gestational diabetes: A systematic review and meta-analysis. *American Journal of Obstetrics and Gynecology, 203*(5), e1-e9. https://doi.org/ 10.1016/j.ajog.2010.06.044

Dirar, A. H. M., & Doupis, J. (2017). Gestational diabetes from A to Z. *World Journal of Diabetes, 8*(12), 489-511. https://doi.org/ 10.4239/wjd.v8.i12.489

Dodd, J. M., Turnbull, D., McPhee, A. J., Deussen, A. R., Grivell, R. M., … Robinson, J. S., (2014). Antenatal lifestyle advice for women who are overweight or obese: LIMIT randomized trial. *British Medical Journal, 328*, g1285. https://doi.org/ 10.1136/bmj.g1285

Dode, M. A. S. O., & Santos, I. S. (2009). Non classical risk factors for gestational diabetes mellitus: a systematic review of the literature. *Cadernos de Saude Publica, 25*(3), S341-S35. https://doi.org/10.1590/S0102-311X2009001500002

Doi, S. A. R., Furuya-Kanamori, L., Toft, E., Musa, O. A. H., Islam, N., Clark, J., & Thalib, L. (2020). Metformin in pregnancy to avert gestational diabetes in women at high risk: Meta-analysis of randomized controlled trials. *Obesity Reviews, 21*(1), e12964. https://doi.org/ 10.1111/obr.12964

Duarte-Gardea, M. (2013). Nutritional management for gestational diabetes. In A. M. Coulston, C. J. Boushey, & M. G. Ferruzzi, *Nutrition in the prevention and treatment of disease* (3rd ed.) (pp. 629-642). San Diego, CA: Academic Press.

Dyck, R., Klomp, H., Tan, L. K., Turnell, R. W., & Boctor, M. A. (2002). A comparison of rates, risk factors, and outcomes of gestational diabetes between aboriginal and non-aboriginal women in the Saskatoon health district. *Diabetes Care, 25*(3), 487-493. https://doi.org/ 10.2337/diacare.25.3.487

Dyck, R., Osgood, N., Lin, T. H., Gao, A., & Stang, M. R. (2010). Epidemiology of diabetes mellitus among First Nations and non-First Nations adults. *Canadian Medical Association Journal, 182*(3), 249-256. https://doi.org/ 10.1503/cmaj.090846

Dyck, R. F., Karunanayake, C., Pahwa, P. Stang, M., & Osgood, N. (2019). Epidemiology of Diabetes in Pregnancy Among First Nations and Non-First Nations Women in Saskatchewan, 1980–2013. Part 1: Populations, Methodology and Frequencies (1980–2009); Results From the DIP: ORRIIGENSS Project. *Canadian Journal of Diabetes*. https://doi.org/10.1016/j.jcjd.2019.10.005

Ehrenstein, V. (2009). Association of Apgar scores with death and neurologic disability. *Clinical Epidemiology, 1*, 45-53. https://doi.org/ 10.2147/clep.s4782

Ehrlich, S. F., Hedderson, M. M., Feng, J., Davenport, E. R., Gunderson, E. P., & Ferrara, A. (2011). Change in body mass index between pregnancies and the risk of gestational diabetes in a second pregnancy. *Obstetrics and Gynecology, 117*(6), 1323-1330. https://doi.org/ 10.1097/AOG.0b013e31821aa358

England, L. J., Levine, R. J., Qian, C., Soule, L. M., Schisterman, E. F., Yu, K. F., & Catalano, P. M. (2004). Glucose intolerance and risk of gestational diabetes mellitus in nulliparous women who smoke during pregnancy. *American Journal of Epidemiology, 160*(12), 1205-1213. https://doi.org/ 10.1093/aje/kwh340

Facco, F., Grobman, W., Kramer, J., Ho, K., & Zee, P. (2010). Self-reported short sleep duration and frequent snoring in pregnancy: Impact on glucose metabolism. *American Journal of Obstetrics and Gynecology, 203*(2), 142.e1-142.e5. https://doi.org/10.1016/j.ajog.2010.03.041

Facco, F., Kramer, J., Ho, K., Zee, P., & Grobman, W. (2010). Sleep disturbances in pregnancy. *Obstetrics and Gynecology, 115*(1), 77-83.

Facco, F. L., Grobman, W. A., Reid, K. J., Parker, C. B., Hunter, S. M., … Zee, P. C. (2017). Objectively measured short sleep duration and later sleep midpoint in pregnancy are associated with higher risk of gestational diabetes. *American Journal of Obstetrics and Gynecology, 217*(4), 447.e1-447.e13. https://doi.org/ 10.1016/j.ajog.2017.05.066

Facco, F. L., Parker, C. B., Reddy, U. M., Silver, R. M., Koch, M. A., … Zee, P. C. (2017). Association between sleep-disordered breathing and hypertensive disorders of pregnancy and gestational diabetes mellitus. *Obstetrics and Gynecology, 129*(1), 31-41. https://doi.org/ 10.1097/AOG.0000000000001805

Farrant, H. J., Krishnaveni, G. V., Hill, J. C., Boucher, B. J., Fisher, D. J., … Fall, C. H. (2009). Vitamin D insufficiency is common in Indian mothers but is not associated with gestational diabetes or variation in newborn size. *European Journal of Clinical Nutrition, 63*(5), 646-652. https://doi.org/ 10.1038/ejcn.2008.14

Feig, D. S., Berger, H., Donovan, L., Godbout, A., Kader, T., Keely, E., & Sanghera, R. (2018). *Diabetes and pregnancy*. Retrieved from http://guidelines.diabetes.ca/browse/chapter36

Feig, D. S., Hwee, J., Shah, B. R., Booth, G. L., Bierman, A. S., & Lipscombe, L. L. (2014). Trends in incidence of diabetes in pregnancy and serious perinatal outcomes: A large, population-based study in Ontario, Canada, 1996-2010. *Diabetes Care, 37*(6), 1590-1596. https://doi.org/10.2337/dc13-2717

Feig, D. S., Murphy, K., Asztalos, E., Tomlinson, G., Sanchex, J., … Barrett, J. F. R. (2016). Metformin in women with type 2 diabetes in pregnancy (MiTy): A multi-center randomized controlled trial. *BMC Pregnancy and Childbirth, 16*(1), 1-8. https://doi.org/10.1186/s12884-016-0954-4

Feig, D. S., Razzaq, A., Sykora, K., Hux, J. E., & Anderson, G. M. (2006). Trends in deliveries, prenatal care, and obstetrical complications in women with pregestational diabetes: A population-based study in Ontario, Canada, 1996-2001. *Diabetes Care, 29*(2), 232-235. https://doi.org/10.2337/diacare.29.02.06.dc05-1482

Fernández-Morera, J. L., Rodriguez-Rodero, S., Menendez-Torre, E., & Fraga, M. F. (2010) The possible role of epigenetics in gestational diabetes: cause, consequence, or both. *Obstetrics and Gynecology International, 2010*, 1-7. https://doi.org/10.1155/2010/605163

Ferrara, A. (2007). Increasing prevalence of gestational diabetes mellitus: A public health perspective. *Diabetes Care, 30*, 141-146. https://doi.org/ 10.2337/dc07-s206

Ferrara, A., Kahn, H. S., Quesenberry, C. P., Riley, C., & Hedderson, M. M. (2004). An increase in the incidence of gestational diabetes mellitus: Northern California, 1991-2000. *Journal of Obstetrics and Gynecology, 103*(3), 526-533. https://doi.org/10.1097/01.AOG.0000113623.18286.20

Ferrara, A., Weiss, N. S., Hedderson, M. M., Quesenberry Jr., C. P., Selby, J. V., … Sacks, D. A. (2007). Pregnancy plasma glucose levels exceeding the American Diabetes Association thresholds, but below the National Diabetes Data Group thresholds for gestational diabetes mellitus, are related to the risk of neonatal macrosomia, hypoglycaemia and hyperbilirubinaemia. *Diabetologia, 50*(2), 298-306. https://doi.org/10.1007/s00125-006-0517-8

Franks, P. W., Looker, H. C., Kobes, S., Touger, L., Tataranni, P. A., Hanson, R. L., & Knowler, W. C. (2006). Gestational glucose tolerance and risk of type 2 diabetes in young Pima Indian offspring. *Diabetes, 55*(2), 460-465. https://doi.org/ 10.2337/diabetes.55.02.06.db05-0823

Freinkel, N. (1980). Of pregnancy and progeny. *Diabetes*, *29*(12), 1023-1035. https://doi.org/ 10.2337/diab.29.12.1023

Fung, T. T., McCullough, M., van Dam, R. M., & Hu, F. B. (2007). A prospective study of overall diet quality and risk of type 2 diabetes in women. *Diabetes Care, 30*(7), 1753-1757. https://doi.org/ 10.2337/dc06-2581

Garcia-Vargas, L., Addison, S. S., Nistala, R., Kurukulasuriya, D., & Sowers, J. R. (2012). Gestational diabetes and the offspring: Implication in the development of the cardiorenal metabolic syndrome in offspring. *CardioRenal Medicine, 2*(2), 134-142. https://doi.org/ 10.1159/000337734

Gardener, H., Spiegelman, D., & Buka, S. L. (2009). Prenatal risk factors for autism: comprehensive meta-analysis. *British Journal of Psychiatry, 195*, 7-14. https://doi.org/10.1192/bjp.bp.108.051672

Gaston, A., & Cramp, A. (2011). Exercise during pregnancy: A review of patterns and determinants. *Journal of Science and Medicine in Sport, 14*(4), 299-305. https://doi.org/ 10.1016/j.jsams.2011.02.006

Glueck, C. J., Goldenberg, N., Sieve, L., & Wang, P. (2008). An observational study of reduction of insulin resistance and prevention of development of type 2 diabetes mellitus in women with polycystic ovary syndrome treated with metformin and diet. *Metabolism, 57*(7), 954-960. https://doi.org/ 10.1016/j.metabol.2008.02.011

Goodyear, L. J., & Kahn, B. B. (1998). Exercise, glucose transport, and insulin sensitivity. *Annual Review of Medicine, 49*, 235-261. https://doi.org/10.1146/annurev.med.49.1.235

Gray, S. G., McGuire, T. M., Cohen, N., & Little, P. J. (2017). The emerging role of metformin in gestational diabetes mellitus. *Diabetes, Obesity, and Metabolism, 19*(6), 765-772. https://doi.org/ 10.1111/dom.12893

Guariguata, L., Linnenkamp, U., Beagley, J., Whiting, D. R., & Cho, N. H. (2014). Global estimates of the prevalence of hyperglycaemia in pregnancy. *Diabetes Research and Clinical Practice, 103*(2), 176-185. https://doi.org/10.1016/j.diabres.2013.11.003

Guideline Development Group, The. (2008). Management of diabetes from preconception to the postnatal period: summary of NICE guidance. *British Medical Journal, 336*(7646), 714-717. https://doi.org/ 10.1136/bmj.39505.641273.AD

Gutierrez, Y. M., & Reader, D. M. (2005). Medical nutrition therapy. In A. M. Thomas, & Y. M. Gutierrez (Eds.), *American dietetic association: Guide to gestational diabetes mellitus* (pp. 45-64). Chicago, IL: ADA.

Hallberg, L. (1988). Iron balance in pregnancy. In H. Berger (Eds.), *Vitamins and minerals in pregnancy and lactation* (pp. 115-127). New York, NY: Raven Press.

Hamer, M., & Chida, Y. (2007). Intake of fruit, vegetables, and antioxidants and risk of type 2 diabetes: Systematic review and meta-analysis. *Journal of Hypertension, 25*(12), 2361-2369. https://doi.org/10.1097/HJH.0b013e3282efc214

Hamza, A., Herr, D., Solomayer, E. F., & Meyberg-Solomayer, G. (2013). Polyhrdramnios: Causes, diagnosis, and therapy. *Geburtshilfe and Frauenheilkunde, 73*(12), 1241-1246. https://doi.org/10.1055/s-0033-1360163

Hans, S., Middleton, P., & Crowther, C. A. (2012). Exercise for pregnant women for preventing gestational diabetes mellitus. *Cochrane Database of Systematic Reviews, 7*, CD009021. https://doi.org/ 10.1002/14651858.CD009021.pub2

Hansen, J. B., Moen, I. W., & Mandrup-Poulsen, T. (2014). Iron: The hard player in diabetes pathophysiology. *Acta Physiologica, 210*(4), 717-32. https://doi.org/ 10.1111/apha.12256

Hauth, J. C., Clifton, R. G., Roberts, J. M., Myatt, L., Spong, C. Y., … Eunice Kennedy Shriver National Institute of Child Health and Human Development Maternal-Fetal Medicine Units Network. (2011). Maternal insulin resistance and preeclampsia. *American Journal of Obstetrics and Gynecology, 204*(4), 327.e1-327.e6. https://doi.org/10.1016/j.ajog.2011.02.024

Health Canada. (2014). Prenatal nutrition guidelines for health professionals: Gestational weight gain. Retrieved from https://www.canada.ca/en/health-canada/services/canada-food-guide/resources/prenatal-nutrition/eating-well-being-active-towards-healthy-weight-gain-pregnancy-2010.html#t2

Health Link BC. (2014). Healthy eating guidelines: For women with gestational diabetes. Retrieved from https://www.healthlinkbc.ca/hlbc/files/healthyeating/pdf/women-gestational-diabetes.pdf

Hedderson, M., Darbinian, J., & Ferrara, A. (2010a). Disparities in the risk of gestational diabetes by race-ethnicity and country of birth. Paediatric and Perinatal Epidemiology, 24(5), 441-448.

Hedderson, M. M., Gunderson, E. P., & Ferrara, A. (2010b). Gestational weight gain and risk of gestational diabetes mellitus. *Obstetrics and Gynaecology, 115*(5), 597-604. https://doi.org/10.1097/AOG.0b013e3181cfce4f

Hedman, C., Pohjasvaara, T., Tolonen, U., Suhonen-Malm, A. S., & Myllyla, V. V. (2002). Effects of pregnancy on mothers' sleep. *Sleep Medicine, 3*, 37-42. https://doi.org/10.1016/s1389-9457(01)00130-7

Henriksen, T. (2008). The macrosomic fetus: A challenge in current obstetrics. *Acta Obstetricia et Gynecologica Scandinavica, 87*(2), 134-145. https://doi.org/10.1080/00016340801899289

Hernandez, T. L., Anderson, M. A., Chartier-Logan, C., Friedman, J. E., & Barbour, L. A. (2013). Strategies in the nutritional management of gestational diabetes. *Clinical Obstetrics & Gynecology, 56*(4), 803-815. https://doi.org/ 10.1097/GRF.0b013e3182a8e0e5

Hinkle, S. N., Laughon, S. K., Catov, J. M., Olsen, J., & Bech, B. H. (2014). First trimester coffee and tea intake and risk of gestational diabetes mellitus: A study within a national birth cohort. *British Journal of Obstetrics and Gynaecology, 122*(3), 420-428. https://doi.org/10.1111/1471-0528.12930

Hitchen, L. (2007). More mothers are dying from causes related to obesity. *British Medical Journal, 335*(7631), 1175. https://doi.org/10.1136/bmj.39420.350799.DB

Holmes, V. A., Barnes, M. S., Alexander, H. D., McFaul, P., & Wallace, J. M. (2009). Vitamin D deficiency and insufficiency in pregnant women: A longitudinal study. *British Journal of Nutrition, 102*(6), 876-881. https://doi.org/10.1017/S0007114509297236

Hosler, A. S., Nayak, S. G., & Radigan, A. M. (2011). Stressful events, smoking exposure and other maternal risk factors associated with gestational diabetes mellitus. *Paediatric and Perinatal Epidemiology, 25*(6), 566-574. https://doi.org/10.1111/j.1365-3016.2011.01221.x

Hutcheon, J. A., Kuret, V., Joseph, K. S., Sabr, Y., & Lim, K. (2013). Immortal time bias in the study of stillbirth risk factors: The example of gestational diabetes. *Epidemiology, 24*(6), 787-790. https://doi.org/10.1097/EDE.0b013e3182a6d9aa

Huxley, R., Lee, C. M., Barzi, F., Timmermeister, L., Czernichow, S., … Woodward, M. (2009). Coffee, decaffeinated coffee, and tea consumption in relation to incident type 2 diabetes mellitus: A systematic review with meta-analysis. *Archives of Internal Medicine, 169*(22), 2053-2063. https://doi.org/ 10.1001/archinternmed.2009.439

Ibanez, L., Sebastiani, G., Lopez-Bermejo, A., Diaz, M., Gomez-Roig, M. D., & de Zegher, F. (2008). Gender specificity of body adiposity and circulating adiponectin, visfatin, insulin, and insulin growth factor-I at term birth: relation to prenatal growth. *Journal of Clinical Endocrinology and Metabolism, 93*(7), 2774-2778. https://doi.org/10.1210/jc.2008-0526

Ibrahim, M. I., Hamdy, A., Shafik, A., Taha, S., Anwar, M., & Faris, M. (2014). The role of adding metformin in insulin-resistant diabetic pregnant women: A randomized controlled trial. *Archives of Gynecology and Obstetrics, 289*(5), 959-965. https://doi.org/10.1007/s00404-013-3090-7

Innes, K. E., & Wimsatt, J. H. (1999). Pregnancy-induced hypertension and insulin resistance: Evidence for a connection. *Acta Obstetricia et Gynecologica Scandinavica, 78*, 263-284. Retrieved from https://obgyn.onlinelibrary.wiley.com/doi/pdf/10.1034/j.1600-0412.1999.780401.x

Institute of Medicine. (2009). *Weight gain during pregnancy: Reexamining the guidelines*. Washington, DC: National Academies Press.

International Diabetes Federation. (2019). *IDF diabetes atlas* (9th ed.) Brussels, BE: International Diabetes Federation.

Irion, O., & Boulvain, M. (1998). Induction of labour for suspected fetal macrosomia. *Cochrane Database of Systematic Reviews*, *2*. https://doi.org/10.1002/14651858.CD000938

Isolauri, E., Rautava, S., Collado, M. C., & Salminen, S. (2015). Role of probiotics in reducing the risk of gestational diabetes. *Diabetes, Obesity, and Metabolism, 17*(8), 713-719. https://doi.org/10.1111/dom.12475

Izci-Balserak, B., & Pien, G. W. (2014). The relationship and potential mechanistic pathways between sleep disturbances and maternal hyperglycemia. *Current Diabetes Report, 14*(2), 459. https://doi.org/10.1007/s11892-013-0459-8

Jarvela, I. Y., Juutinen, J., Koskela, P., Hartikainen, A. L., Kulmala, P., Knip, M., & Tapanainen, J. S. (2006). Gestational diabetes identifies women at risk for permanent type 1 and type 2 diabetes in fertile age: Predictive role of autoantibodies. *Diabetes Care, 29*(3), 607–612. https://link.gale.com/apps/doc/A143240979/EAIM?u=usaskmain&sid=EAIM&xid=a463d00c

Jastrow, N., Roberge, S., Gauthier, R. J., Laroche, L., Duperron, L., Brassard, N., & Bujold, E. (2010). Effect of birth weight on adverse obstetric outcomes in vaginal birth after cesarean delivery. *Obstetrics and Gynecology, 115*(2 Pt 1), 338-343. https://doi.org/10.1097/AOG.0b013e3181c915da

Johns, E. C., Denison, F. C., Norman, J. E., & Reynolds, R. M. (2018). Gestational diabetes mellitus: Mechanisms, treatment, and complications. *Trends in Endocrinology and Metabolism, 29*(11), 743-754. https://doi.org/10.1016/j.tem.2018.09.004

Johnson, R. C., & Schoeni, R. F. (2011). The influence of early-life events on human capital, health status, and labor market outcomes over the life course. *The B.E. Journal of Economic Analysis and Policy, 11*(3), 1-18. https://doi.org/10.2202/1935-1682.2521

Jolly, M., Sebire, N., Harris, J., Robinson, S., & Regan, L. (2000). The risks associated with pregnancy in women aged 35 years or older. *Human Reproduction, 15*(11), 2433-2437. https://doi.org/10.1093/humrep/15.11.2433

Jolly, M. C., Sebire, N. J., Harris, J. P., Regan, L., & Robinson, S. (2003). Risk factors for macrosomia and its clinical consequences: A study of 350,311 pregnancies. *European Journal of Obstetrics, Gynecology, and Reproductive Biology, 111*, 9-14. https://doi.org/10.1016/s0301-2115(03)00154-4

Jovanovic-Peterson, L., Peterson, C. M., Reed, G. F., Metzger, B. E., Mills, J. L., Knopp, R. H., & Aarons, J. H. (1991). Maternal postprandial glucose levels and infant birth weight: The Diabetes in Early Pregnancy Study. The National Institute of Child Health and Human Development: diabetes in early pregnancy study. *American Journal of Obstetrics and Gynecology, 164*, 103-111. https://doi.org/10.1016/0002-9378(91)90637-7

Kakoly, N. S., Earnest, A., Moran, L. J., Teede, H. J., & Joham, A. E. (2017). Group-based developmental BMI trajectories, polycystic ovary syndrome, and gestational diabetes: A community-based longitudinal study. *BMC Medicine, 15*, 195. https://doi.org/10.1186/s12916-017-0957-7

KC, K., Shakya, S., & Zhang, H. (2015). Gestational diabetes mellitus and macrosomia: A literature review. *Annals of Nutrition and Metabolism, 66*(suppl 2), 14-20. https://doi.org/10.1159/000371628

Khandelwal, M. (2008). GDM: Postpartum management to reduce long-term risks. *Current Diabetes Report, 8*(4), 287-293. https://doi.org/10.1007/s11892-008-0051-9

Kim, C., & Ferrara, A. (2010). *Gestational diabetes during and after pregnancy*. London, UK: Springer.

Kim, C., Liu, T., Valdez, R., & Beckles, G. L. (2009). Does frank diabetes in first-degree relatives of a pregnant woman affect the likelihood of her developing gestational diabetes mellitus or nongestational diabetes? *American Journal of Obstetrics and Gynecology, 201*(6), 576.e1-576.e6. https://doi.org/10.1016/j.ajog.2009.06.069

Kim, S. Y., England, J. L., Sharma, J. A., & Njoroge, T. (2011). Gestational diabetes mellitus and risk of childhood overweight and obesity in offspring: A systematic review. *Experimental Diabetes Research, 2011*, 541308. https://doi.org/10.1155/2011/541308

King, J. R., Korst, L. M., Miller, D. A., & Ouzounian, J. G. (2012). Increased composite maternal and neonatal morbidity associated with ultrasonographically suspected fetal macrosomia. *The Journal of Maternal-Fetal & Neonatal Medicine, 25*(10), 1953-1959. https://doi.org/10.3109/14767058.2012.674990

Kinnunen, T. I., Luoto, R., Helin, A., & Hemminki, E. (2016). Supplemental iron intake and the risk of glucose intolerance in pregnancy: Re-analysis of a randomised controlled trial in Finland. *Maternal and Child Nutrition, 12*, 74-84. https://doi.org/10.1111/mcn.12139

Knutson, L. (2010). Sleep duration and cardiometabolic risk: A review of the epidemiologic evidence. *Best Practice and Research: Clinical Endocrinology and Metabolism, 24*(5), 731-743. https://doi.org/10.1016/j.beem.2010.07.001

Kramer, M. S., & McDonald, S. W. (2006). Aerobic exercise for women during pregnancy. *Cochrane Database Systematic Review, 2*, CD000180. https://doi.org/10.1002/14651858.CD000180.pub2

Kuo, C. H., Chen, S. C., Fang, C. T., Nien, F. J., Wu, E. T., … Li, H. Y. (2017). Screening gestational diabetes mellitus: The role of maternal age. *PLoS One, 12*(3), e0173049. https://doi.org/ 10.1371/journal.pone.0173049

Kwak, S. H., Kim, X. H., Cho, Y. M., Choi, S. H., Jang, H. C., & Park, K. S. (2010). Polymorphisms in KCNQl are associated with gestational diabetes in a Korean population. *Hormone Research in Paediatrics, 74*(5), 333-338. https://doi.org/10.1159/000313918

Kwak, S. H., & Park, K. S. (2016). Recent progress in genetic and epigenetic research on type 2 diabetes. *Experimental and Molecular Medicine, 48*, e220. https://doi.org/10.1038/emm.2016.7

Laitinen, K., Poussa, T., Isolauri, E., & The Nutrition, Allergy, Mucosal Immunology and Intestinal Microbiota Group. (2009). Probiotics and dietary counselling contribute to glucose regulation during and after pregnancy: A randomised controlled trial. *British Journal of Nutrition, 101*(11), 1679-1687. https://doi.org/10.1017/S0007114508111461

Lao, T. T., Ho, L. F., Chen, B. C. P., & Leung, W. C. (2006). Maternal age and prevalence of gestational diabetes mellitus*. Diabetes Care, 29*(4), 948-949. https://doi.org/10.2337/diacare.29.04.06.dc05-2568

Lapolla, A., Dalfrà, M. G., & Fedele, D. (2005). Insulin therapy in pregnancy complicated by diabetes: Are insulin analogs a new tool? *Diabetes Metabolism Research and Reviews, 21*(3), 241-252. https://doi.org/ 10.1002/dmrr.551

Lapolla, A., Dalfra, M. G., & Fedele, D. (2009). Management of gestational diabetes mellitus. *Diabetes Metabolic Syndrome and Obesity: Targets and Therapy, 17*(2), 73-82. https://doi.org/10.2147/dmsott.s3407

Lau, S. L., Gunton, J. E., Athayde, N. P., Byth, K., & Cheung, N. W. (2011). Serum 25-hydroxyvitamin D and glycated haemoglobin levels in women with gestational diabetes mellitus. *The Medical Journal of Australia, 194*(7), 334-337. https://doi.org/10.5694/j.1326-5377.2011.tb03000.x

Lauenborg, J., Grarup, N., & Damm, P. (2009). Common type 2 diabetes risk gene variants associated with gestational diabetes. *Journal of Clinical Endocrinology and Metabolism, 94*, 145-150. https://doi.org/10.1210/jc.2008-1336

Lauenborg, J., Grarup, N., Damm, P., Borch-Johnsen, K., Jorgensen, X., Pedersen, O., & Hansen, X. (2009). Common type 2 diabetes risk gene variants associate with gestational diabetes. *Journal of Clinical Endocrinology and Metaboliism, 94*(1), 145-150. https://doi.org/10.1210/jc.2008-1336

Lavery, J. A., Friedman, A. M., Keyes, K. M., Wright, J. D., & Ananth, C. V. (2017). Gestational diabetes in the United States: Temporal changes in prevalence rates between 1979 and 2010. *British Journal of Obstetrics and Gynaecology, 124*(5), 804-813. https://doi.org/10.1111/1471-0528.14236

Lawlor, D. A., Lichtenstein, P., & Långström, N. (2011). Association of maternal diabetes mellitus in pregnancy with offspring adiposity into early adulthood: Sibling study in a prospective cohort of 280,866 men from 248,293 families. *Circulation*, *123*(3), 258-265. https://doi.org/10.1161/CIRCULATIONAHA.110.980169

Lawrence, J. M., Contreras, R., Chen, W., & Sacks, D. A. (2008). Trends in the prevalence of preexisting diabetes and gestational diabetes mellitus among a racially/ethnically diverse population of pregnant women, 1999-2005. *Diabetes Care, 31*(5), 899–904. https://doi.org/10.2337/dc07-2345

Lazer, S., Biale, Y., Mazor, M., Lewenthal, H., & Insler, V. (1986). Complications associated with the macrosomic fetus. Journal of Reproductive Medicine for the Obstetrician and Gynecologist, *31*(6), 501– 505.

Lee, K. A., Zaffke, M. E., & McEnany, G. (2000). Parity and sleep patterns during and after pregnancy. *Obstetrics and Gynecology, 95*, 14-18. https://doi.org/10.1016/s0029-7844(99)00486-x

Leng, J., Shao, P., Zhang, C., Tian, H., Zhang, F., … Yang, X. (2015). Prevalence of gestational diabetes mellitus and its risk factors in Chinese pregnant women: A prospective population-based study in Tianjin, China. *PLoS One, 10*(3), e0121029. https://doi.org/ 10.1371/journal.pone.0121029

Lewis, G. F., Carpentier, A., Adeli, K., & Giacca, A. (2002). Disordered fat storage and mobilization in the pathogenesis of insulin resistance and type 2 diabetes. *Endocrine Reviews, 23*(2), 201-229. https://doi.org/10.1210/edrv.23.2.0461

Leybovitz-Haleluya, N., Wainstock, T., Landau, D., & Sheiner, E. (2018). Maternal gestational diabetes mellitus and the risk of subsequent pediatric cardiovascular diseases of the offspring: a population-based cohort study with up to 18 years of follow up. *Acta Diabetologica*, *55*(10), 1037–1042. https://doi.org/10.1007/s00592-018-1176-1

Liese, A. D., Nichols, M., Sun, X., D'Agostino Jr., R. B., & Haffner, S. M. (2009). Adherence to the DASH Diet is inversely associated with incidence of type 2 diabetes: The insulin resistance atherosclerosis study. *Diabetes Care, 32*(8), 1434-1436. https://doi.org/ 10.2337/dc09-0228

Lindsay, K., Kennelly, M., Culliton, M., Smith, T., Maguire, O. C., … McAuliffe, F. M. (2014). Probiotics in obese pregnancy do not reduce maternal fasting glucose: A double-blind, placebo-controlled, randomized trial (probiotics in pregnancy study). *The American Journal of Clinical Nutrition, 99*(6), 1432-1439. https://doi.org/10.3945/ajcn.113.079723

Lindsay, K. L., Walsh, C. A., Brennan, L., & McAuliffe, F. M. (2013). Probiotics in pregnancy and maternal outcomes: A systematic review. *Journal of Maternal-Fetal and Neonatal Medicine, 26*(8), 772-778. https://doi.org/10.3109/14767058.2012.755166

Lindsay, R. S., Dabelea, D., Roumain, J., Hanson, R. L., Bennett, P. H., & Knowler, W. C. (2000). Type 2 diabetes and low birth weight: The role of paternal inheritance in the association of low birth weight and diabetes. *Diabetes, 49*(3), 445-449. https://doi.org/10.2337/diabetes.49.3.445

Liu, Z., Ao, D., Yang, H., & Wang, Y. (2014). Gestational weight gain and risk of gestational diabetes mellitus among Chinese women. *Chinese Medical Journal, 127*(7), 1255-1260. https://doi.org/10.3760/cma.j.issn.0366-6999.20132772

Lo, J. C., Feigenbaum, S. L., Escobar, G. J., Yang, J., Crites, Y. M., & Ferrara, A. (2006). Increased prevalence of gestational diabetes mellitus among women with diagnosed polycystic ovary syndrome: A population-based study. *Diabetes Care, 29*(8), 1915-1917. https://doi.org/10.2337/dc06-0877

Louie, J. C. Y., Markovic, T. P., Ross, G. P., Foote, D., & Brand-Miller, J. C. (2013). Higher glycemic load diet is associated with poorer nutrient intake in women with gestational diabetes mellitus. *Nutrition Research, 33*(4), 259-265. https://doi.org/10.1016/j.nutres.2013.02.008

Luoto, R., Laitinen, K., Nermes, M., & Isolauri, E. (2010). Impact of maternal probiotic-supplemented dietary counselling on pregnancy outcome and prenatal and postnatal growth: A double-blind, placebo-controlled study. *British Journal of Nutrition, 103*(12), 1792-1799. https://doi.org/10.1017/S0007114509993898

Lurie, S., Insler, V., & Hagay, Z. J. (1996). Induction of labor at 38 to 39 weeks of gestation reduces the incidence of shoulder dystocia in gestational diabetic patients class A2. *American Journal of Perinatology*, *13*(5), 293-296. https://doi.org/10.1055/s-2007-994344

Luque-Fernandez, M. A., Bain, P. A., Gelaye, B., Redline, S., & Williams, M. A. (2013). Sleep-disordered breathing and gestational diabetes mellitus. *Diabetes Care, 36*(10), 3353-3360. https://doi.org/10.2337/dc13-0778

Lv, S., Wang, J., & Xu, Y. (2015). Safety of insulin analogs during pregnancy: A meta-analysis. *Archives of Gynecology and Obstetrics, 292*(4), 749-756. https://doi.org/10.1007/s00404-015-3692-3

Macintosh, M. C., Fleming, K. M., Bailey, J. A., Doyle, P., Modder, J., … Miller, A. (2006). Perinatal mortality and congenital anomalies in babies of women with type 1 or type 2 diabetes in England, Wales, and Northern Ireland: Population based study. *British Medical Journal, 333*(7560), 177. https://doi.org/10.1136/bmj.38856.692986.AE

Maghbooli, Z., Hossein-Nezhad, A., Karimi, F., Shafaei, A. R., & Larijani, B. (2008). Correlation between vitamin D3 deficiency and insulin resistance in pregnancy. *Diabetes Metabolism Research and Reviews, 24*, 27-32. https://doi.org/10.1002/dmrr.737

Makgoba, M., Nelson, S. M., Savvidou, M., Messow, C. M., Nicolaides, K., & Sattar, N. (2011). First-trimester circulating 25-hydroxyvitamin d levels and development of gestational diabetes mellitus. *Diabetes Care, 34*(5), 1091-1093. https://doi.org/10.2337/dc10-2264

Martin, A. O., Simpson, J. L., Ober, C., & Freinkel, N. (1985). Frequency of diabetes mellitus in mothers of probands with gestational diabetes: possible maternal influence on the predisposition to gestational diabetes. *American Journal of Obstetrics and Gynecology, 151*(4), 471-475. https://doi.org/10.1016/0002-9378(85)90272-8

Mathew, M., Machado, L., Al-Ghabshi, R., & Al-Haddabi, R. (2005). Fetal macrosomia. Risk factor and outcome. *Saudi Medical Journal, 26*, 96-100.

McFarland, M. B., Trylovich, C. G., & Langer, O. (1998). Anthropometric differences in macrosomic infants of diabetic and nondiabetic mothers. *Journal of Maternal-Fetal Medicine, 7*(6), 292-295. https://doi.org/10.1002/(sici)1520-6661(199811/12)7:6<292::aid-mfm7>3.0.co;2-a

McGovern, A., Butler, L., Jones, S., van Vlymen, J., Sadek, K., … de Lusignan, S. (2014). Diabetes screening after gestational diabetes in England: a quantitative retrospective cohort study. *British Journal of General Practice, 64*(618), e17-e23. https://doi.org/10.3399/bjgp14X676410

McLeod, L., & Ray, J. G. (2002). Prevention and detection of diabetic embryopathy. *Community Genetics, 5*, 33-39. https://doi.org/10.1159/000064629

Meek, C. L., Lewis, H. B., Patient, C., Murph, H. R., & Simmons, D. (2015). Diagnosis of gestational diabetes mellitus: Falling through the net. *Diabetologia, 58*(9), 2003-2012. https://doi.org/10.1007/s00125-015-3647-z

Meigs, J. B., Cupples, L. A., & Wilson, P. W. (2000). Parental transmission of type 2 diabetes: The Framingham Offspring Study. *Diabetes, 49*(12), 2201-2207. https://doi.org/10.2337/diabetes.49.12.2201

Metzger, M. A., Schushan-Eisen, I., Lubin, D., Moran, O., Kuint, J., & Mazkereth, R. (2014). Delivery room breastfeeding for prevention of hypoglycaemia in infants of diabetic mothers. *Fetal and Pediatric Pathology, 33*, 23-28. https://doi.org/10.3109/15513815.2013.842271

Mindell, J. A., & Jacobson, B. J. (2000). Sleep disturbances during pregnancy. *Journal of Obstetric, Gynecologic, and Neonatal Nursing, 29*(6), 590-597. https://doi.org/10.1111/j.1552-6909.2000.tb02072.x

Mission, J. F., Ohno, M. S., Cheng, Y. W., & Caughey, A. B. (2012). Gestational diabetes screening with the new IADPSG guidelines: A cost-effectiveness analysis. *American Journal of Obstetrics and Gynecology, 207*, 326.e1-326.e9. https://doi.org/10.1016/j.ajog.2012.06.048

Mithal, A., & Karla, S. (2014). Vitamin D supplementation in pregnancy. *Indian Journal of Endocrinology and Metabolism, 18*(5), 593-596. Retrieved from http://www.ijem.in/text.asp?2014/18/5/593/139204

Mohammadbeigi, A., Farhadifar, F., Soufi Zadeh, N., Mohammadsalehi, N., Rezaiee, M., & Aghaei, M. (2013). Fetal macrosomia: Risk factors, maternal, and perinatal outcome. *Annals of Medical and Health Sciences Research, 3*(4), 546-550. https://doi.org/10.4103/2141-9248.122098

Montonen, J., Boeing, H., Steffen, A., Lehmann, R., Fritsche, A., … Pischon, T. (2012). Body iron stores and risk of type 2 diabetes: Results from the European Prospective Investigation into Cancer and Nutrition (EPIC)-potsdam study. *Diabetologia, 55*(10), 2613-2621. https://doi.org/10.1007/s00125-012-2633-y

Moore, L. E., Clokey, D., Rappaport, V. J., & Curet, L. B. (2010). Metformin compared with glyburide in gestational diabetes: A randomized controlled trial. *Obstetrics and Gynecology, 115*, 55-59. https://doi.org/10.1097/AOG.0b013e3181c52132

Moosazadeh, M., Asemi, Z., Lankarani, K. B., Tabrizi, R., Maharlouei, N., … Akbari, M. (2017). Family history of diabetes and the risk of gestational diabetes mellitus in Iran: A systematic review and meta-analysis. *Diabetes and Metabolic Syndrome: Clinical Research and Reviews, 11*(1), S99-S104. https://doi.org/10.1016/j.dsx.2016.12.016

Morikawa, M., Cho, K., Yamada, T., Yamada, T., Sato, S., & Minakami, H. (2013). Fetal macrosomia in Japanese women. *Journal of Obstetrics and Gynaecology Research, 39*(5), 960-965. https://doi.org/10.1111/j.1447-0756.2012.02059.x

Morselli, L., Leproult, R., Balbo, M., & Spiegel, K. (2010). Role of sleep duration in the regulation of glucose metabolism and appetite. *Best Practice and Research: Clinical Endocrinology and Metabolism, 24*(5), 687-702. https://doi.org/10.1016/j.beem.2010.07.005

Morton, S. B. (2006). Maternal nutrition and fetal growth and development. In P. D. Gluckman & M. A. Hanson (Eds.), *Developmental origins of health and disease* (pp. 98-129). Cambridge, UK: Cambridge University Press.

Mosca, L., Benjamin, E. J., Berra, K., Bezanson, J. L., Dolor, R. J., … Wenger, N. K. (2011). Effectiveness-based guidelines for the prevention of cardiovascular disease in women--2011 update: A guideline from the American Heart Association. *Circulation, 123*(11), 1243-1262. https://doi.org/10.1161/CIR.0b013e31820faaf8

Moses, R. G., Morris, G. J., Petocz, P., San Gil, F., & Garg, D. (2011). The impact of potential new diagnostic criteria on the prevalence of gestational diabetes mellitus in Australia. *The Medical Journal of Australia, 194*(7), 338-340. https://doi.org/10.5694/j.1326-5377.2011.tb03001.x

Mottola, M. F., Davenport, M. H., Ruchat, S. M., Davies, G. A., Poitras, V. J., … Zehr, L. (2018). 2019 Canadian guidelines for physical activity throughout pregnancy. *British Journal of Sports Medicine, 52*, 1339-1346. https://doi.org/10.1136/bjsports-2018-100056

Mottola, M. F., Lander, S., Giroux, I., Hammond, J. M. S., Lebrun, C., McManus, R., & Sopper M. M. (2005). Glucose and insulin responses in women at risk for gestational diabetes mellitus before and after a Nutrition and Exercise Lifestyle Intervention Program (NELIP). *Medicine and Science in Sports Exercise, 37*, S309-S310.

Mudd, L. M., Owe, K. M., Mottola, M. F., & Pivarnik, J. M. (2013). Health benefits of physical activity during pregnancy: an international perspective. *Medicine and Science in Sports and Exercise, 45*(2), 268-277. https://doi.org/10.1249/MSS.0b013e31826cebcb

Muller, S., & Nirmala, M. (2018). Effects of pre-pregnancy maternal body mass index on gestational diabetes mellitus. *International Journal of Engineering and Technology, 7*(1.9), 279-282. Retrieved from https://pdfs.semanticscholar.org/da2d/4c52ecf6e275808d44b66afccb0294b9578f.pdf

Mullington, J. M., Haack, M., Toth, M., Serrador, J., & Meier-Ewert, H. (2009). Cardiovascular, inflammatory and metabolic consequences of sleep deprivation. *Progress in Cardiovascular Diseases, 51*(4), 294-302. https://doi.org/10.1016/j.pcad.2008.10.003

Musso, G., Gambino, R., & Cassader, M. (2011). Interactions between gut microbiota and host metabolism predisposing to obesity and diabetes. *Annual Review of Medicine, 62*, 361-380. https://doi.org/10.1146/annurev-med-012510-175505

Mustaniemi, S., Vaarasmaki, M., Eriksson, J. G., Gissler, M., Laivuori, H., … Morin-Papunen, L. (2018). Polycystic ovary syndrome and risk factors for gestational diabetes. *Endocrine Connections, 7*(7), 859-869. https://doi.org/10.1530/EC-18-0076

Najafian M., & Cheraghi, M. (2012). Occurrence of fetal macrosomia rate and its maternal and neonatal complications: A 5-year cohort study. *ISRN Obstetrics and Gynecology, 2012*, 1-5. https://doi.org/10.5402/2012/353791

Najati, N., & Saboktakin, L. (2010). Prevalence and underlying etiologies of neonatal hypoglycemia. *Pakistan Journal of Biological Sciences, 13*(15), 753-756. https://doi.org/10.3923/pjbs.2010.753.756

National Institute for Health and Care Excellence [NIHCE]. (2008). *Diabetes in pregnancy: Management of diabetes and its complications from preconception to the postnatal period. Clinical Guideline 63*. London, UK: National Institute for Health and Care Excellence. Retrieved from https://www.nice.org.uk/guidance/ng3/resources/diabetes-in-pregnancy-management-from-preconception-to-the-postnatal-period-51038446021

Nerenberg, K. A., Johnson, J. A., Leung, B., Savu, A., Ryan, E. A., Chik, C. L., & Kaul, P. (2013*).* Risks of gestational diabetes and preeclampsia over the last decade in a cohort of Alberta women. *Journal of Obstetrics and Gynaecology Canada, 35*(11), 986-994*.* https://doi.org/10.1016/S1701-2163(15)30786-6

Nilsson, C., Carlsson, A., & Landin-Olsson, M. (2013). Increased risk for overweight among Swedish children born to mothers with gestational diabetes mellitus. *Pediatric Diabetes, 15*, 57-66. https://doi.org/10.1111/pedi.12059

Olafsdottir, A. S., Skuladottir, G. V., Thorsdottir, I., Hauksson, A., & Steingrimsdottir, L. (2006). Maternal diet in early and late pregnancy in relation to weight gain*. International Journal of Obesity, 30*(3), 492-499. https://doi.org/10.1038/sj.ijo.0803184

Oster, R. T., Mayan, M., & Toth, E. L. (2014). Diabetes in pregnancy among first nations women. *Qualitative Health Research, 24*(11), 1469-1480. https://doi.org/10.1177/1049732314545089

Oster, R. T., & Toth, E. L. (2009). Differences in the prevalence of diabetes risk-factors among first nation, metis and non-aboriginal adults attending screening clinics in rural Alberta, Canada. *Rural and Remote Health, 9*(2), 1170. Retrieved from www.rrh.org.au/journal/article/1170

O'Sullivan, J. B., Gellis, S. S., Dandrow, R. V., & Tenney, B. O. (1966). The potential diabetic and her treatment in pregnancy. *Obstetrics and Gynecology, 27*(5), 683-689.

Padayachee, C., & Coombes, J. S. (2015). Exercise guidelines for gestational diabetes. *World Journal of Diabetes, 6*(8), 1033-1044. https://doi.org/10.4239/wjd.v6.i8.1033

Palomba, S., de Wilde, M. A., Falbo, A., Koster, M. P., La Sala, G. B,. & Fauser, B. C. (2015). Pregnancy complications in women with polycystic ovary syndrome. *Human Reproduction Update*, *21*(5), 575-592. https://doi.org/ 10.1093/humupd/dmv029

Pappa, K. I., Gazouli, M., Economou, K., Daskalakis, G., Anastasiou, E., Anagnou, N. P., & Antsaklis, A. (2011). Gestational diabetes mellitus shares polymorphisms of genes associated with insulin resistance and type 2 diabetes in the Greek population. *Gynecological Endocrinology, 27*(4), 267-272. https://doi.org/10.3109/09513590.2010.490609

Pederson, J. (1967). *The pregnant diabetic and her newborn: Problems and management* (pp. 128-137). Baltimore, MD: Williams & Wilkins.

Peters, R. K., Kjos, S. L., Xiang, A., & Buchanan, T. A. (1996). Long term diabetogenic effect of single pregnancy in women with previous gestational diabetes mellitus. *Lancet, 347*(8996), 227-230. https://doi.org/10.1016/s0140-6736(96)90405-5

Pettitt, D. J., Ospina, P., Kolaczynski, J. W., & Jovanovic, L. (2003). Comparison of an insulin analog, insulin aspart, and regular human insulin with no insulin in gestational diabetes mellitus. *Diabetes Care, 26*(1), 183-186. https://doi.org/10.2337/diacare.26.1.183

Pien, G. W., Fife, D., Pack, A. I., Nkwuo, J. E., & Schwab, R. J., (2005). Changes in symptoms of sleep-disordered breathing during pregnancy. *Sleep, 28*(10), 1299-1305. https://doi.org/10.1093/sleep/28.10.1299

Pittas, A. G., Lau, J., Hu, F. B., & Dawson-Hughes, B. (2007). The role of vitamin D and calcium in type 2 diabetes. A systematic review and meta-analysis. *Journal of Clinical Endocrinology and Metabolism, 92*(6), 2017-2029. https://doi.org/10.1210/jc.2007-0298

Poel, Y. H., Hummel, P., Lips, P., Stam, F., van der Ploeg, T., & Simsek, S. (2012); Vitamin D and gestational diabetes: A systematic review and meta-analysis*. European Journal of Internal Medicine, 23*(5), 465-469. https://doi.org/10.1016/j.ejim.2012.01.007

Poolsup, N., Suksomboon, N., & Amin, M. (2014). Efficacy and safety of oral antidiabetic drugs in comparison to insulin in treating gestational diabetes mellitus: A meta-analysis. *PLoS One, 9*(10), e109985. https://doi.org/10.1371/journal.pone.0109985

Public Health Agency of Canada, [PHAC]. (2014). *Maternal diabetes in Canada.* Retrieved at https://www.canada.ca/en/public-health/services/publications/healthy-living/maternal-diabetes-canada.html

Raio, L., Ghezzi, F., Di Naro, E., Buttarelli, M., Franchi, M., Durig, P., & Bruhwiler, H. (2003). Perinatal outcome of fetuses with a birth weight greater than 4500 g: An analysis of 3356 cases. *European Journal of Obstetrics & Gynecology and Reproductive Biology*, *109*(2), 160-165. https://doi.org/10.1016/s0301-2115(03)00045-9

Ramos, G. A., Hanley, A. A., Aguayo, J., Warshak, C. R., Kim, J. H., & Moore, T. R. (2012). Neonatal chemical hypoglycemia in newborns from pregnancies complicated by type 2 and gestational diabetes mellitus – the importance of neonatal ponderal index. *The Journal of Maternal-Fetal & Neonatal Medicine, 25*(3), 267-271. https://doi.org/10.3109/14767058.2011.573828

Rani, P. R., & Begum, J. (2016). Screening and diagnosis of gestational diabetes mellitus, where do we stand. *Journal of Clinical and Diagnostic Research, 10*(4), qe01-qe04. https://doi.org/10.7860/JCDR/2016/17588.7689

Rawal, S., Hinkle, S. N., Zhu, Y., Albert, P. S., & Zhang, C. (2017). A longitudinal study of sleep duration in pregnancy and subsequent risk of gestational diabetes: Findings from a prospective, multiracial cohort. *American Journal of Obstetrics and Gynecology, 216*(4), 399.e1-399.e8. https://doi.org/10.1016/j.ajog.2016.11.1051

Rayanagoudar, G., Hashi, A. A., Zamora, J., Khan, K. S., Hitman, G. A., & Thangaratinam, S. (2016). Quantification of the type 2 diabetes risk in women with gestational diabetes: a systematic review and meta-analysis of 95,750 women. *Diabetologia, 59*(7), 1403-1411. https://doi.org/10.1007/s00125-016-3927-2

Reis, F. M., Florio, P., Cobellis, L., Luisi, S., Severi, F. M., … Petraglia, F. (2001). Human placenta as a source of neuroendocrine factors. *Biology of the Neonate, 79*(3-4), 150-156. https://doi.org/10.1159/000047083

Retnakaran, R., Kramer, C. K., Ye, C., Kew, S., Hanley, A. J., … Zinman, B. (2015). Fetal sex and maternal risk of gestational diabetes mellitus: the impact of having a boy. *Diabetes Care, 38*(5), 844-851. https://doi.org/10.2337/dc14-2551

Reusens, B., Ozanne, S. E., & Remacle, C. (2007). Fetal determinants of type 2 diabetes. *Current Drug Targets, 8*(8), 935-941. https://doi.org/10.2174/138945007781386866

Reutrakul, S., & Van Cauter, E. (2014). Interactions between sleep, circadian function, and glucose metabolism: implications for risk and severity of diabetes. *Annals of the New York Academy of Sciences, 1311*, 151-173. https://doi.org/10.1111/nyas.12355

Reyes-Munoz, E., Castellanos-Barroso, G., Ramirez-Eugenio, B. Y., Ortega-Gonzalez, C., Parra, A., Castillo-Mora, A., & De la Jara-Diaz, J. F. (2012). The risk of gestational diabetes mellitus among Mexican women with a history of infertility and polycystic ovary syndrome. *Fertility and Sterility, 97*(6), 1467-1471. https://doi.org/10.1016/j.fertnstert.2012.03.023

Robinson, H., Tkatch, S., Mayes, D. C., Bott, N., & Okun, N. (2003). Is maternal obesity a predictor of shoulder dystocia? *Obstetrics and Gynecology, 101*, 24-27. https://doi.org/10.1016/s0029-7844(02)02448-1

Rogozinska, E., Chamillard, M., Hitman, G. A., Khan, K. S., & Thangaratinam, S. (2015). Nutritional manipulation for the primary prevention of gestational diabetes mellitus: A meta-analysis of randomized studies. *PLoS One, 10*(2), e0115526. https://doi.org/10.1371/journal.pone.0115526

Romero, R., & Badr, M. S. (2014). A role for sleep disorders in pregnancy complications: challenges and opportunities. *American Journal of Obstetrics and Gynecology, 210*(1), 3-11. https://doi.org/10.1016/j.ajog.2013.11.020

Rosenstein, M. G., Cheng, Y. W., Snowden, J. M., Nicholson, J. A., Doss, A. E., & Caughey, A. B. (2012). Risk of stillbirth and infant death stratified by gestational age in women with gestational diabetes. *American Journal of Obstetrics and Gynecology, 206*(4), 309.e1-309.e7. https://doi.org/10.1016/j.ajog.2012.01.014

Rowan, J. A., Hague, W. M., Gao, W., Battin, M. R., & Moore, M. P. (2008). MIG Trial Investigators. Metformin versus insulin for the treatment of gestational diabetes. *The North England Journal of Medicine, 358*(19), 2003-2015. https://doi.org/10.1056/NEJMoa0707193

Rozance, P. J., & Hay, W. W. (2006). Hypoglycemia in newborn infants: Features associated with adverse outcomes. *Neonatology, 90*, 74-86. https://doi.org/10.1159/000091948

Sacks, K. N., Friger, M., Shoham-Vardi, I., Abokaf, H., Spiegel, E., … Sheiner, E. (2016). Prenatal exposure to gestational diabetes mellitus as an independent risk factor for long-term neuropsychiatric morbidity of the offspring. *American Journal of Obstetrics and Gynecology, 215*(3), 380.e1-380.e7. https://doi.org/10.1016/j.ajog.2016.03.030

Sagedal, L. R., Øverby, N. C., Bere, E., Torstveit, M. K., Lohne-Seiler, H., … Vistad, I. (2016). Lifestyle intervention to limit gestational weight gain: the Norwegian Fit for Delivery randomised controlled trial. *British Journal of Obstetrics and Gynecology, 124*(1), 97-109. https://doi.org/ 10.1111/1471-0528.13862

Salas-Salvadó, J., Bullo, M., Babio, N., Martinez-Gonzalez, M. A., Ibarrola-Jurado, N., Basora, J., ... Ros, E. (2011). Reduction in the Incidence of type 2 diabetes with the Mediterranean Diet: Results of the PREDIMED-Reus nutrition intervention randomized trial. *Diabetes Care, 34*, 14-19. https://doi.org/ 10.2337/dc10-1288

Sanabria-Martinez, G., Garcia-Hermoso, A., Poyatos-Leon, R., Alvarez-Beuno, C., Sanchez-Lopez, M., & Martinez-Vizcaino, V. (2015). Effectiveness of physical activity interventions on preventing gestational diabetes mellitus and excessive maternal weight gain: A meta-analysis. *British Journal of Obstetrics and Gynaecology, 122*(9), 1167-1174. https://doi.org/10.1111/1471-0528.13429

Sarkar, S., Watman, J., Seigel W. M., & Schaeffer, H. A. (2003). A prospective controlled study of neonatal morbidities in infants born at 36 weeks or more gestation to women with diet-controlled gestational diabetes (GDM-class Al). *Journal of Perinatology, 23*(3), 223-228. https://doi.org/10.1038/sj.jp.7210882

Savitz, D. A., Janevic, T. M., Engel, S. M., Kaufman, J. S., & Herring, A. H. (2008). Ethnicity and gestational diabetes in New York City, 1995-2003. *British Journal of Obstetrics and Gynecology, 115*(8), 969-978. https://doi.org/10.1111/j.1471-0528.2008.01763.x

Savvaki, D., Taousani, E., Goulis, D. G., Tsirou, E., Voziki, E., … Tokmakidis, S. P. (2018). Guidelines for exercise during normal pregnancy and gestational diabetes: A review of international recommendations. *Hormones, 17*, 521-529. https://doi.org/10.1007/s42000-018-0085-6

Schmid, S. M., Jauch-Chara, K., Hallschmid, M., & Schultes, B. (2009). Mild sleep restriction acutely reduces plasma glucagon levels in healthy men. *The Journal of Clinical Endocrinology and Metabolism, 94*(12), 5169-5173. https://doi.org/10.1210/jc.2009-0969

Schmid, S. M., Hallschmid, M., Jauch-Chara, K., Bandorf, N., Born, J., & Schultes, B. (2007). Sleep loss alters basal metabolic hormone secretion and modulates the dynamic counterregulatory response to hypoglycemia. *The Journal of Clinical Endocrinology and Metabolism, 92*(8), 3044-3051. https://doi.org/10.1210/jc.2006-2788

Schneider, S., Freerksen, N., Rohrig, S., Hoeft, B., & Maul, H. (2012). Gestational diabetes and preeclampsia--similar risk factor profiles? *Early Human Development, 88*(3), 179-84. https://doi.org/10.1016/j.earlhumdev.2011.08.004

Schoenaker, D. A., Mishra, G. D., Callaway, L. K., & Soedamah-Muthu, S. S. (2016). The role of energy, nutrients, foods, and dietary patterns in the development of gestational diabetes mellitus: A Systematic review of observational studies. *Diabetes Care, 39*(1), 16-23. https://doi.org/10.2337/dc15-0540

Schramm, W., Stockbauer, J., & Hoffman, H. (1996). Exercise, employment, other daily activities, and adverse pregnancy outcome. *American Journal of Epidemiology, 143*(3), 211-218. https://doi.org/10.1093/oxfordjournals.aje.a008731

Shaat, N., Lernmark, A., Karlsson, E., Ivarsson, S., Parikh, H., Berntorp, K., & Groop, L. (2007). A variant in the transcription factor 7-like 2 (TCF7L2) gene is associated with an increased risk of gestational diabetes mellitus. *Diabetologia, 50*(5), 972-979. https://doi.org/10.1007/s00125-007-0623-2

Shah, A., Stotland, N. E., Cheng, Y. W., Ramos, G. A., & Caughey, A. B. (2011). The association between body mass index and gestational diabetes mellitus varies by race/ethnicity. *American Journal of Perinatology, 28*(7), 515-520. https://doi.org/10.1055/s-0031-1272968

Shand, A. W., Bell, J. C., McElduff, A., Morris, J., & Roberts, C. L. (2008). Outcomes of pregnancies in women with pre-gestational diabetes mellitus and gestational diabetes mellitus: A population-based study in New South Wales, Australia, 1998-2002. *Diabetes Medicine, 25*(6), 708-715. https://doi.org/10.1111/j.1464-5491.2008.02431.x

Shen, G. X., Shafer, L. A., Martens, P. J., Sellers, E., Torshizi, A. A., … Dean, H. J. (2015). Does First Nations ancestry modify the association between gestational diabetes and subsequent diabetes: A historical prospective cohort study among women in Manitoba, Canada. *Diabetic Medicine, 33*(9), 1245-1252. https://doi.org/10.1111/dme.12962

Shepherd, E., Gomersall, J. C., Tieu, J., Han, S., Crowther, C. A., & Middleton, P. (2017). Combined diet and exercise interventions for preventing gestational diabetes mellitus. *Cochrane Database of Systematic Reviews, 11*, CD010443. https://doi.org/10.1002/14651858.CD010443.pub3

Shi, P., Yang, W., Yu, Q., Zhao, Q., Li, C., … Yan, W. (2014). Overweight, gestational weight gain and elevated fasting plasma glucose and their association with macrosomia in Chinese pregnant women. *Maternal and Child Health Journal, 18*, 10-15. https://doi.org/10.1007/s10995-013-1253-6

Shields, B. M., Knight, B., Hopper, H., Hill, A. Powell, R. J., Hattersley, A. T., & Clark, P. M. (2007). Measurement of cord insulin and insulin-related peptides suggests that girls are more insulin resistant than boys at birth. *Diabetes Care, 30*(10), 2661-2666. https://doi.org/10.2337/dc06-1501

Shin, H. D., Park, B. L., Shin, H. J., Kirn, J. Y., Park, S., Kirn, B., & Kirn, S. H. (2010). Association of KCNQl polymorphisms with the gestational diabetes mellitus in Korean women. *Journal of Clinical Endocrinology and Metabolism, 95*, 445-449. https://doi.org/10.1210/jc.2009-1393

Shoham, I., Wiznitzer, A., Silberstein, T., Fraser, D., Holcberg, G., Katz, M., & Mazor, M. (2001). Gestational diabetes complicated by hydramnios was not associated with increased risk of perinatal morbidity and mortality. *European Journal of Obstetrics and Gynecolgy and Reproductive Biology, 100*, 46-49. https://doi.org/10.1016/s0301-2115(01)00426-2

Sibai, B., Dekker, G., & Kupfermic, M. (2005). Pre-eclampsia. *Lancet, 365*(9461), 785-799. https://doi.org/10.1016/S0140-6736(05)17987-2

Sobngwi, E., Boudou, P., Mauvais-Jarvis, F., Leblanc, H., Velho, G., … Gautier, J. F. (2003). Effect of a diabetic environment in utero on predisposition to type 2 diabetes. *Lancet, 361*(9372), 1861-1865. https://doi.org/10.1016/S0140-6736(03)13505-2

Soheilykhah, S., Mojibian, M., Rashidi, M., Rahimi-Saghand, S., & Jafari, F. (2010). Maternal vitamin D status in gestational diabetes mellitus. *Nutrition in Clinical Practice, 25*(5), 524-527. https://doi.org/ 10.1177/0884533610379851

Solomon, C. G., & Seely, E. W. (2001). Brief review: Hypertension in pregnancy: A manifestation of the insulin resistance syndrome? *Hypertension, 37*(2), 232-239. https://doi.org/10.1161/01.hyp.37.2.232

Solomon, C. G., Willett, W. C., Carey, V. J., Rich-Edwards, J., Hunter, D. J., … Manson, J. E. (1997). A prospective study of pregravid determinants of gestational diabetes mellitus. *Journal of American Medical Association, 278*(13), 1078-1083. https://doi.org/10.1001/jama.1997.03550130052036

Sommer, C., Morkrid, K., Jenum, A. K., Sletner, L., Mosdol, A., & Birkeland, K. I. (2014). Weight gain, total fat gain, and regional fat gain during pregnancy and the association with gestational diabetes: A population-based cohort study. *International Journal of Obesity, 38*, 76-81. https://doi.org/10.1038/ijo.2013.185

Sorbye, L. M., Skjaervan, R., Klungsoyr, K., & Morken, N. H. (2017). Gestational diabetes mellitus and interpregnancy weight change: A population-based cohort study. *PLoS Medicine, 14*(8), e1002367. https://doi.org/10.1371/journal.pmed.1002367

Spiegel, K., Knutson, K., Leproult, R., Tasali, E., & Van Cauter, E. (2005). Sleep loss: A novel risk factor for insulin resistance and type 2 diabetes. *Journal of Applied Physiology, 99*(5), 2008-2019. https://doi.org/10.1152/japplphysiol.00660.2005

Statistics Canada. (2018). Fertility: Fewer children, older moms. Retrieved from https://www150.statcan.gc.ca/n1/pub/11-630-x/11-630-x2014002-eng.htm

Stewart, Z. A., & Murphy, H. R. (2015). Gestational diabetes. *Medicine, 43*(1), 44-47. https://doi.org/10.1016/j.mpmed.2014.10.010

Stomnaroska-Damcevski, O., Petkovska, E., Jancevska, S., & Danilovski, D. Neonatal hypoglycemia: A continuing debate in definition and management. *Prilozi, 36*(3) 91-97. https://doi.org/ 10.1515/prilozi-2015-0083

Sweet, C. B., Grayson, S., & Pollack, M. (2013). Management strategies for neonatal hypoglycemiz. *Journal of Pediatric Pharmacology, 18*(3), 199-208. https://doi.org/10.5863/1551-6776-18.3.199

Tait, N. H. (2014). Patient and caregiver perspectives of health provision practices for First Nations and Métis women with gestational diabetes mellitus accessing care in Winnipeg, Manitoba. *BMC Health Services Research, 14*, 440. https://doi.org/10.1186/1472-6963-14-440

Tam, W. H., Ma, R. C. W., Yang, X., Ko, G. T., Tong, P. C., … Chan, J. C. (2008). Glucose intolerance and cardiometabolic risk in children exposed to maternal gestational diabetes mellitus in utero. *Pediatrics, 122*(6), 1229-1234. https://doi.org/10.1542/peds.2008-0158

Teh, W. T., Teede, H. J., Paul, E., Harrison, C. L., Wallace, E. M., & Allan, C. (2011). Risk factors for gestational diabetes mellitus: Implications for the application of screening guidelines. *Australian and New Zealand Journal of Obstetrics and Gynaecology, 51*(1), 26-30. https://doi.org/10.1111/j.1479-828X.2011.01292.x

Thangaratinam, S. Rogozinska, E., Jolly, K., Glinkowski, S., Roseboom, T., … Khan, K. S. (2012). Effects of interventions in pregnancy on maternal weight and obstetric outcomes: Meta-analysis of randomized evidence. *British Medical Journal, 344*, e2088. https://doi.org/10.1136/bmj.e2088

Thomas, F., Balkau, B., Vauzelle-Kervroedan, F., & Papoz, L. (1994). Maternal effect and familial aggregation in NIDDM. The CODIAB study. *Diabetes, 43*, 63-67. https://doi.org/ 10.2337/diab.43.1.63

Tobias, D. K., Zhang, C., Chavarro, J., Bowers, K., Rich-Edwards, J., … Hu, F. B. (2012). Pre-pregnancy adherence to dietary patterns and lower risk of gestational diabetes mellitus. *American Journal of Clinical Nutrition, 96*(2), 289-295. https://doi.org/10.3945/ajcn.111.028266

Tobias, D. K., Zhang, C., Van Dam, R. M., Bowers, K., & Hu, F. B. (2011). Physical activity before and during pregnancy and risk of gestational diabetes mellitus: A meta-analysis. *Diabetes Care, 34*, 223-229. https://doi.org/ 10.2337/dc10-1368

Tryggvadottir, E. A., Medek, H., Birgisdottir, B. E., Geirsson, R. T., & Gunnarsdottir, I. (2016). Association between healthy maternal dietary pattern and risk for gestational diabetes mellitus. *European Journal of Clinical Nutrition, 70*, 237-242. https://doi.org/ 10.1038/ejcn.2015.145

Turkman, S., Johansson, S., & Dahmoun, M. (2018). Foetal macrosomia and foetal-maternal outcomes at birth. *Journal of Pregnancy, 2018*, 1-9. https://doi.org/10.1155/2018/4790136

Twedt, R., Bradley, M., Deiseroth, D., Althouse, A., & Facco, F. (2015). Sleep duration and blood glucose control in women with gestational diabetes mellitus. *Obstetrics and Gynecology*, *126*(2), 326-331. https://doi.org/ 10.1097/AOG.0000000000000959

Qiu, C., Enquobahrie, D., Frederick, I. O., Abetew, D., & Williams, M. A. (2010). Glucose intolerance and gestational diabetes risk in relation to sleep duration and snoring during pregnancy: A pilot study. *BMC Women's Health, 10*, 17. https://doi.org/10.1186/1472-6874-10-17

Qiu, C., Zhang, C., Gelaye, B., Enquobahrie, D. A., Frederick, I. O., & Williams, M. A. (2011). Gestational diabetes mellitus in relation to maternal dietary heme iron and nonheme iron intake. *Diabetes Care, 34*, 1564-1569. https://doi.org/10.2337/dc11-0135

Valdés, E., Sepúlveda-Martínez, A., Manukián, B., & Parra-Cordero, M. (2014). Assessment of pregestational insulin resistance as a risk factor of preeclampsia. *Gynecologic and Obstetric Investigation, 77*(2), 111-116. https://doi.org/10.1159/000357944

van Dam, R. M., & Hu, F. B. (2005). Coffee consumption and risk of type 2 diabetes: A systematic review. *Journal of American Medical Association, 294*, 97-104. https://doi.org/10.1001/jama.294.1.97

Vanky, E., Salvesen, K. A., Heimstad, R., Fougner, K. J., Romundstad, P., & Carlsen, S. M. (2004). Metformin reduces pregnancy complications without affecting androgen levels in pregnant polycystic ovary syndrome women: Results of a randomized study. *Human Reproduction, 19*(8), 1734-1740. https://doi.org/10.1093/humrep/deh347

Vannucci, R. C., & Vannucci, S. J. (2001). Hypoglycemic brain injury. *Seminars in neonatology, 6*(2), 147-155. https://doi.org/10.1053/siny.2001.0044

Vella, S., Buetow, L., Royle, P., Livingstone, S., Colhoun, H. M., & Petrie, J. R. (2010). The use of metformin in type 1 diabetes: A systematic review of efficacy. *Diabetologia, 53*(5), 809-820. https://doi.org/10.1007/s00125-009-1636-9

Vieth, R., Chan, P. C., & MacFarlane, G. D. (2001). Efficacy and safety of vitamin D3 intake exceeding the lowest observed adverse effect level. *The American Journal of Clinical Nutrition, 73*(2), 288-294. https://doi.org/10.1093/ajcn/73.2.288

Villamor, E., & Cnattingius, S. (2006). Interpregnancy weight change and risk of adverse pregnancy outcomes: A population-based study. *Lancet, 368*(9542), 1164-1170. https://doi.org/10.1016/S0140-6736(06)69473-7

Wang, C., Wei, Y., Zhang, X., Zhang, Y., Xu, Q., … Yang, H. (2017). A randomized clinical trial of exercise during pregnancy to prevent gestational diabetes mellitus and improve pregnancy outcome in overweight and obese pregnant women. *American Journal of Obstetrics and Gynecology, 216*(4), 340-351. https://doi.org/10.1016/j.ajog.2017.01.037

Wang, H., Leng, J., Li, W., Wang, L., Zhang, C., … Yang, X. (2016). Sleep duration and quality, and risk of gestational diabetes mellitus in pregnant Chinese women. *Diabetes Medicine, 34*, 44-50. https://doi.org/ 10.1111/dme.13155

Weissmann-Brenner, A., Simchen, M. J., Zilberberg, E., Kalter, A., Weisz, B., Achiron, R., & Dulitzky, M. (2012). Maternal and neonatal outcomes of macrosomic pregnancies. *Medical Science Monitor, 18*(9), PH77-PH81. https://doi.org/10.12659/msm.883340

Wilson, D., de la Ronde, S., Brascoupe, S., Apale, A. N., Barney, L., … Society of Rural Physicians of Canada. (2013). Health professionals working with First Nations, Inuit, and Metis consensus guideline. *Journal of Obstetrics and Gynaecology Canada, 35*(6), 550-558. https://doi.org/10.1016/S1701-2163(15)30915-4

Wilson, R. L., Gummow, J. A., McAninch, D., Bianco-Miotto, T., & Roberts, C. T. (2018). Vitamin and mineral supplementation in pregnancy: Evidence to practice. *Women's and Children's Therapeutics Review, 48*(2), 186-192. https://doi.org/10.1002/jppr.1438

Wolf, M. F., Peleg, D., Stahl-Rosenzwieg, T., Kurzweil, Y., & Yogev, Y. (2017). Isolated polyhydramnios in the third trimester: Is a gestational diabetes evaluation of value? *Gynecological Endocrinology, 33*(11), 849-852. https://doi.org/10.1080/09513590.2017.1323857

World Health Organization, [WHO]. (1999). *Definition, Diagnosis and Classification of Diabetes Mellitus and its Complications*. Department of Noncommunicable Disease Surveillance. chrome-extension://oemmndcbldboiebfnladdacbdfmadadm/https://apps.who.int/iris/bitstream/10665/66040/1/WHO_NCD_NCS_99.2.pdf

World Health Organization, [WHO]. (2005). World health report: Make every mother, and child count. https://www.who.int/whr/2005/en/

World Health Organization, [WHO]. (2016). *WHO recommendation on the diagnosis of gestational diabetes in pregnancy. WHO Reproductive Health Library*. https://extranet.who.int/rhl/topics/preconception-pregnancy-childbirth-and-postpartum-care/antenatal-care/who-recommendation-diagnosis-gestational-diabetes-pregnancy-0

World Health Organization [WHO]. (2012). *WHO global report: mortality attributable to tobacco*. https://www.who.int/tobacco/publications/surveillance/rep_mortality_attributable/en/

Wung, S. F., & Lin, P. C. (2011). Shared genomics of type 2 and gestational diabetes mellitus. *Annual Review of Nursing Research, 29*, 227-260. https://doi.org/ 10.1891/0739-6686.29.227

Xiang, A. H., Wang, X., & Martinez, M. P. (2015). Association of maternal diabetes with autism in offspring. *Journal of American Medical Association, 313*(14), 1425-1434. https://doi.org/10.1001/jama.2015.2707

Xiang, A. H., Wang, X., Martinez, M. P., Walthall, J. C., Curry, E. S., … Getahun, D. (2015). Association of maternal diabetes with autism in offspring. *Journal of American Medical Association, 313*(14), 1425-1434. https://doi.org/10.1001/jama.2015.2707

Xiao, L., Zhao, J. P., Nuyt, A. M., Fraser, W. D., & Luo, Z. C. (2014). Female fetus is associated with greater maternal insulin resistance in pregnancy. *Diabetic Medicine, 31*(12), 1696-1701. https://doi.org/10.1111/dme.12562

Xiong, X., Saunders, L. D., Wang, F. L., & Demianczuk, N. N. (2001). Gestational diabetes mellitus: Prevalence risk factors, maternal and infant outcomes. *International Journal of Gynaecology and Obstetrics, 75*, 221-228. https://doi.org/10.1016/S0020-7292(01)00496-9

Xu, G., Jing, J., Bowers, K., Liu, B., & Bao, W. (2014). Maternal diabetes and the risk of autism spectrum disorders in the offspring: A systematic review and meta-analysis. *Journal of Autism and Developmental Disorders, 44*(4), 766-775. https://doi.org/10.1007/s10803-013-1928-2

Yamamoto, J. M., Kellett, J. E., Balsells, M., Garcia-Patterson, A., Hadar, E., … Corcoy, R. (2018). Gestational diabetes mellitus and diet: A systematic review and meta-analysis of randomized controlled trials examining the impact of modified dietary interventions on maternal glucose control and neonatal birth weight. *Diabetes Care, 41*(7), 1346-1361. https://doi.org/10.2337/dc18-0102

Yuen, L., & Wong, V. W. (2015). Gestational diabetes mellitus: Challenges for different ethnic groups. *World Journal of Diabetes, 6*(8), 1024-1032. https://doi.org/10.4239/wjd.v6.i8.1024

Zaren, B., Lindmark, G., Wilbell, L., & Folling, I. (2000). The effect of smoking on glucose homeostasis and fetal growth in pregnant women. *Upsala Journal of Medical Sciences, 105,* 41-56. https://doi.org/10.1517/03009734000000046

Zhang, C., Liu, S., Solomon, C. G., & Hu, F. B. (2006). Dietary fiber intake, dietary glycemic load, and the risk for gestational diabetes mellitus. *Diabetes Care, 29*(10), 2223-3220. https://doi.org/ 10.2337/dc06-0266

Zhang, C., & Ning, Y. (2011). Effect of dietary and lifestyle factors on the risk of gestational diabetes: review of epidemiologic evidence. *The American Journal of Clinical Nutrition, 94*(suppl 6), 1975S-1979S. https://doi.org/10.3945/ajcn.110.001032

Zhang, C., Qiu, C., Hu, F. B., David, R. M., van Damn, R. M., Bralley, A., & Williams, M. A. (2008). Maternal plasma 25-hydroxyvitamin D concentrations and the risk for gestational diabetes mellitus. *PLoS ONE, 3*(11), e3753. https://doi.org/10.1371/journal.pone.0003753

Zhang, C., Schulze, M. B., Solomon, C. G., & Hu, F. B. (2006). A prospective study of dietary patterns, meat intake and the risk of gestational diabetes mellitus. *Diabetologia, 49*(11), 2604-2613. https://doi.org/ 10.1007/s00125-006-0422-1

Zhang, C., Solomon, C. G., Manson, J. E., & Hu, F. B. (2006). A prospective study of pregravid physical activity and sedentary behaviors in relation to the risk for gestational diabetes mellitus. *Archives of Internal Medicine, 166*(5), 543-548. https://doi.org/10.1001/archinte.166.5.543

Zhang, C., Tobias, D., Chavarro, J. E., Bao, W., Wang, D., Ley, S. H., & Hu, F. B. (2014). Adherence to healthy lifestyle and risk of gestational diabetes mellitus: prospective cohort study. *British Medical Journal, 349*, g5450. https://doi.org/10.1136/bmj.g5450

Zhao, L. P., Sheng, X. Y., Zhou, S., Yang, T., Ma, L. Y., Zhou, Y., & Cui, Y. M. (2015). Metformin versus insulin for gestational diabetes mellitus: A meta-analysis. *British Journal of Clinical Pharmacology, 80*(5), 1224-1234. https://doi.org/10.1111/bcp.12672

Zhao, Z., Li, S., Liu, G., Yan, F., Ma, X., Huang, Z., & Tian, H. (2012). Body iron stores and heme-iron intake in relation to risk of type 2 diabetes: A systematic review and meta-analysis. *PLoS One, 7*, e41641. https://doi.org/ 10.1371/journal.pone.0041641

Zheng, J., Feng, Q., Zheng, Z., & Xiao, X. (2018). The effects of probiotics on metabolic health in pregnant women: An evidence based meta-analysis. *PLoS One, 13*(5), e0197771. https://doi.org/10.1371/journal.pone.0197771

Zhong, C., Chen, R., Zhou, X., Xu, S., Li, Q., … Yang, N. (2018). Poor sleep during early pregnancy increases subsequent risk of gestational diabetes mellitus. *Sleep Medicine, 46*, 20-25. https://doi.org/10.1016/j.sleep.2018.02.014

Zhou, Q., Zhang, K., Li, W., Liu, J. X, Hong, J., Qin, S. W, ... Nie, M. (2009). Association of KCNQl gene polymorphism with gestational diabetes mellitus in a Chinese population. *Dietologia, 52*(11), 2466-2468. https://doi.org/10.1007/s00125-009-1500-y