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Association of Demographic Characteristics with Gestational Diabetes Mellitus and Gestational Hypertension in Ohio during 2012

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Association of Demographic Characteristics with Gestational Diabetes Mellitus and Gestational Hypertension in Ohio during 2012

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Master of Public Health Culminating Experience

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Abstract

Background: Gestational Diabetes Mellitus (GDM) and Gestational Hypertension (GHTN) are serious conditions that can arise during pregnancy. Both of these disorders can cause detrimental health effects to both mother and newborn. Methods: Ohio vital statistics birth data was obtained from Public Health- Dayton and Montgomery County. The data set included 137,268 women who gave birth during 2012 in Ohio and had a known GDM and/or GHTN status. IBM Statistical Package for Social Sciences for Mac Version 20 (SPSS, IBM, 2011) was used to analyze the data. Descriptive statistics, odds ratios, chi-square analyses, and multivariate logistic forward stepwise regressions were calculated from the data set. Results: GDM and GHTN were diagnosed in 6.2% and 5.5% of pregnant women respectively. Pre-pregnancy hypertension (OR = 2.783, p < .001) and increased pre-pregnancy BMI (OR = 2.889, p < .001) were the strongest predictors for GDM. Increased delivery BMI (OR = 2.921, p < .001) and pre-pregnancy Diabetes Mellitus (OR = 2.292, p < .001) were the strongest predictors for GHTN. After forward stepwise multiple logistic regression, pre-pregnancy BMI (OR = 1.095, p < .001), Asian race (OR = 0.642, p = .006) and marital status (OR = 0.507, p < .001) were significant factors for GDM. Pre-pregnancy BMI (OR= 1.101, p < .001), Medicaid (OR = 0.642, p < .001), and self-pay payment (OR= 0.433, p < .001) remained significant for GHTN. Conclusions: Physicians should continue to follow established guidelines, encourage pre-pregnancy weight loss, and screen individuals considered to be “high-risk” for GDM and GHTN.

Keywords: pregnancy, glucose, obesity, race, blood pressure
Association of Demographic Characteristics with Gestational Diabetes Mellitus and Gestational Hypertension in Ohio during 2012

Pregnancy is a very turbulent period in a woman’s life. The body undergoes physiological changes in order to sustain additional life. Consequently, one’s social life may drastically change. Pregnant women may need time off of work, extra burden may be placed on family members to accommodate for additional needs, and there is much financial strain to obtain healthy nutrition as well as attend prenatal appointments. While the majority of pregnancies are uneventful, certain pathologies may either be unmasked or developed during this time. Gestational Diabetes Mellitus (GDM) and Gestational Hypertension (GHTN) are two serious diseases that can drastically impact the health of both the mother and the newborn. Many of these outcomes may not be seen immediately; they may present in early childhood in the form of obesity or even later in life when the offspring becomes pregnant herself. Screening criteria and risk factors have been established for both GDM and GHTN, but like most literature pertaining to pregnant women, evidence is lacking. This study seeks to evaluate established and un-established risk factors in women giving birth in Ohio with the primary outcomes of GDM and GHTN.

Literature Review

Gestational Hypertension

Introduction.

Hypertensive disorders represent one of the most common complications of pregnancy, which affects 5-10% of pregnancies in the United States each year (Brown & Garovic, 2011; Danso & Opare-Addo, 2010; Jwa et al., 2011; Lee, Zhang, Wikman, Lindqvist, & Reilly, 2012; Leeman & Fontaine, 2008; Lykke et al., 2009; Mastrogiannis, Spiliopoulos, Mulla, & Homko,
About 50,000 women die from hypertensive disorders of pregnancy worldwide per year with the highest rates in black women, women who are older than 45 years old, and individuals who have Type 1 or Type 2 Diabetes Mellitus (DM1 and DM2) (Nij Bijvank & Duvekot, 2010; Vest & Cho, 2012). Gestational Hypertension (GHTN) is defined as the development of hypertension (HTN) after 20 weeks gestation with a return to normal blood pressure levels by 12 weeks postpartum (Ames, Rueda, & Caughey, 2012; Bulloch & Carroll, 2012; Fabry, Richart, Chengz, Van Bortel, & Staessen, 2010; Flack, Ferdinand, Nasser, & Rossi, 2010; Leeman & Fontaine, 2008; Lykke et al., 2009; Sullivan et al., 2011). GHTN falls in the category of “hypertensive disorders of pregnancy” which includes chronic HTN (CHTN), pre-eclampsia, eclampsia, and HELLP syndrome. Each disorder will be noted, but this literature review will primarily focus on GHTN.

**Blood pressure during pregnancy.**

Blood pressure (BP) typically decreases in the late first and early second trimester (Brown & Garovic, 2011; Jwa et al., 2011; Sullivan et al., 2011). Blood pressure then increases to pre-pregnancy levels or higher beginning in the third trimester (Jwa et al., 2011; Solomon et al., 1997). The mechanism behind GHTN is unknown, but it is likely multi-factorial with genetic, immune, vascular and placental factors each playing important roles (Cardwell, 2013; Sullivan et al., 2011). Each of these factors decreases placental blood supply, which in turn, causes fetal hypoxia and negatively impacts brain development (Heikura et al., 2013). The mechanism is likely similar to the etiology of CHTN in a non-pregnant individual since GHTN increases future post-pregnancy CHTN risk (Vest & Cho, 2012). GHTN affects between 5-10%

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1 >140 mmHg systolic blood pressure or >90 mmHg diastolic blood pressure
of all pregnancies (Ames et al., 2012; Fabry et al., 2010). In women who have DM1 or DM2, the underlying insulin resistance likely plays a roll as well (Sullivan et al., 2011). The diagnosis of GHTN may be difficult to differentiate from essential or CHTN as there may be no method to determine if a given pregnant woman’s rise in BP began previous or after 20 weeks gestation (Solomon & Seely, 2011; Sullivan et al., 2011). The diagnosis of GHTN can then only be established if the high BP levels resolve after 12 weeks postpartum.

**Workup of GHTN.**

There are different stages of HTN with a systolic BP of 140-159 mmHg and diastolic BP of 90-99 mmHg being classified as Stage I (Bulloch & Carroll, 2012; Yoder, Thornburg, & Bisognano, 2009). Systolic and diastolic BPs higher than the Stage I cutoffs are classified as Stage II. These stages can also be classified as “mild” and “severe”. Typically, HTN does not arise until the post-pregnancy years, however with the increasing prevalence of obesity and metabolic disorders in the general population, more and more pregnant women may develop CHTN (Vest & Cho, 2012; Yoder et al., 2009). This may cloud the diagnosis of GHTN as the physician must determine whether the increased blood pressure levels arose prior to pregnancy, which would lead to a diagnosis of CHTN, or due to a secondary cause, such as hypothyroidism (Wilson, Casey, McIntire, Halvorson, & Cunningham, 2012; Yoder et al., 2009). In order to determine if a given pregnant woman has GHTN or an even more serious condition, the physician should order a urinalysis, complete blood count, blood urea nitrogen and serum creatinine levels to assess kidney function, serum uric acid, and liver function enzymes (Vest & Cho, 2012).
Risk factors for GHTN.

Risk factors for the development of GHTN include high body mass index (BMI), excessive gestational weight gain, pre-pregnancy DM1 or DM2, Gestational Diabetes Mellitus (GDM) in any pregnancy, previous birth complicated by GHTN, barriers to seeking healthcare, poor nutrition, clinical depression, increased maternal age, and nulliparity (Cardwell, 2013; Flack et al., 2010; Fortner, Pekow, Solomon, Markenson, & Chasan-Taber, 2009; Hedderson, Darbinian, Sridhar, & Quesenberry, 2012; Jwa et al., 2011; Sullivan et al., 2011). In addition, about 20% of pregnant diabetic women will develop GHTN or an even more serious condition known as pre-eclampsia, which will be described later in this literature review (Sullivan et al., 2011). Consequently, the development of GHTN was twice as frequent in women with DMI compared to nondiabetic controls (Lee et al., 2012). Moreover, nulliparity, obesity, and excessive gestational weight gain was demonstrated to be strongly and positively associated with the development of GHTN in the Latina population (Fortner et al., 2009). For pregnant women in the workforce, one study observed that occupational exposures may have an impact as the number of consecutive work days without a day off was associated with the onset of GHTN (Haelterman, Marcoux, Croteau, & Dramaix, 2007). Jobs that involved pushing or pulling persons or objects was strongly associated with GHTN (Haelterman et al., 2007). One Swedish study found that women of the blood group AB had an increased risk of GHTN (Lee et al., 2012). Environment may also play a role in GHTN. One study found that pregnant women who live in neighborhoods with high levels of particulate matter, both PM10 and PM2.5, were more likely to develop GHTN (Vinikoor-Imler, Gray, Edwards, & Miranda, 2012). As for negative health consequences, GHTN is associated with fetal growth retardation, maternal intracerebral hemorrhage, renal and liver failure, small for gestational age infants, preterm delivery, abruption
of the placenta, and increased maternal cardiovascular disease risk later in life (Cardwell, 2013; Fabry et al., 2010; Flack et al., 2010; Fortner et al., 2009; Hedderson & Ferrara, 2008; Heikura et al., 2013; Sullivan et al., 2011; Vest & Cho, 2012; Vinikoor-Imler et al., 2012). Furthermore, effects on the fetus may not be seen immediately. One British cohort study found that maternal pregnancy related HTN disorders resulted in increased odds of their offspring developing HTN later on in life (Palmsten, Buka, & Michels, 2010). Cognitive development may be threatened as well. Heikura et al. (2013) demonstrated that GHTN may be a risk factor that might predispose offspring to impaired cognitive development in childhood compared to normotensive mothers. As for the mother, non-diabetic women who developed GHTN had a 3-fold elevated risk for developing DM2 in the future (Sullivan et al., 2011). In addition, another cohort study found that the risk to develop DM2 later in life after developing GHTN was increased three-fold (Lykke et al., 2009). This study also found a slight increase in the risk of maternal thromboembolism compared to normotensive pregnant women (Lykke et al., 2009).

**Pre-eclampsia.**

If an expectant mother develops GHTN, she is at risk to develop pre-eclampsia, a potentially severe pregnancy complication. Pre-eclampsia is defined as new onset HTN occurring after 20 weeks gestation with the presence of protein in the urine² (Ames et al., 2012; Leeman & Fontaine, 2008; Vest & Cho, 2012; Yoder et al., 2009). If CHTN is present and pre-eclampsia develops, then the condition is known as CHTN with super-imposed pre-eclampsia (Bulloch & Carroll, 2012; Vest & Cho, 2012). Pre-eclampsia occurs in 2-5% of all pregnancies, but the risk increases to 20-25% in pregnancies of women with CHTN (Vest & Cho, 2012). The mechanism for pre-eclampsia likely differs from GHTN since increased placental anti-

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² Must register 2+ on urine dipstick on 2 occasions 6 hours apart or urine protein measurement of >300mg/24 hours
angiogenic peptides and syncytiotrophoblast debris increase the risk of pre-eclampsia, but not GHTN (Bulloch & Carroll, 2012; Vest & Cho, 2012). Physical examination in suspected pre-eclampsia should include palpation of the liver, fundoscopy for retinal edema, assessment of mental status and reflexes, and a cardiorespiratory examination (Vest & Cho, 2012). This condition is associated with increased risks to the fetus which includes premature delivery, growth retardation, and death (Brown & Garovic, 2011; Haelterman et al., 2007). One study showed an increased odds ratio of developing GDM in pregnant women who had systolic BPs > 140/90 (Hedderson & Ferrara, 2008; Hedderson et al., 2012). Additional symptoms include multi-organ dysfunction such as elevated liver enzymes, kidney failure, and reduced fetal growth (Yoder et al., 2009). GHTN is differentiated from pre-eclampsia by an absence of protein in the urine (Leeman & Fontaine, 2008; Sullivan et al., 2011; Vest & Cho, 2012). About 50 percent of women who are diagnosed with GHTN during the 3rd trimester will develop pre-eclampsia (Leeman & Fontaine, 2008). The only definitive treatment for pre-eclampsia is delivery of the fetus (Leeman & Fontaine, 2008; Solomon & Seely, 2011; Sullivan et al., 2011). Blood pressure will typically improve within a few days of delivery and should return to baseline by 12 weeks postpartum (Vest & Cho, 2012).

**Eclampsia and HELLP syndrome.**

Pre-eclampsia can progress into an emergency condition known as eclampsia. Eclampsia follows the diagnostic criteria of pre-eclampsia with the addition of grand-mal seizures (Leeman & Fontaine, 2008). The estimated incidence of eclampsia is about 4 to 5 cases per 10,000 live births (Vest & Cho, 2012). Blood pressure may only be mildly elevated in women who develop eclampsia (Leeman & Fontaine, 2008). Pregnant women who are diagnosed with pre-eclampsia should immediately be given intravenous magnesium sulfate as the Magpie trial demonstrated
that risk for developing eclampsia is halved after the administration of this drug as well as likely
decreased risk of maternal death (Altman et al., 2002). In addition, HELLP syndrome is another
severe condition that can result from GHTN. HELLP syndrome is characterized by hemolysis,
elevated liver enzyme levels, and destruction of platelets (Leeman & Fontaine, 2008; Sullivan et
al., 2011). The clinical presentation may vary as blood pressure levels may normalize in 12-18%
of women, and 13% do not have proteinuria (Leeman & Fontaine, 2008). There is no benefit to
expectantly manage a pregnant patient with HELLP syndrome; fetal outcome will be poor with
delay in delivery occurring at the expense of the mother’s health (Sullivan et al., 2011).

**Goals of treatment.**

In terms of diagnosis, the clinician must carefully weigh the risk versus benefit ratio for
each individual patient, with the overall goal of improving both maternal and fetal outcomes
(Brown & Garovic, 2011). First and second trimester prenatal care and/or preconception care
provides opportunities for the early identification for hypertensive disorders of pregnancy as well
as appropriate intervention (Danso & Opare-Addo, 2010). A proper medical history must be
taken with each pregnant patient to determine if elevated blood pressure was present prior to
pregnancy. This may be difficult in low-income countries where pre-pregnancy BPs are often
not available (Danso & Opare-Addo, 2010). If pre-pregnancy BPs are unavailable, consistent
blood pressure monitoring must be employed at each prenatal appointment with an appropriate
sized cuff while the patient is in a seated position (Danso & Opare-Addo, 2010; Leeman &
Fontaine, 2008). If resources allow, serial urine protein measurement, usually by protein reagent
dipsticks, should be utilized to detect the presence of pre-eclampsia (Danso & Opare-Addo,
2010). Measurements of BP in the physician’s office may be elevated due to “white coat HTN”.
If so, ambulatory blood pressure monitoring should be encouraged to ensure the most accurate
BP measurements possible (Vest & Cho, 2012). In pregnant women with GHTN, fetal growth must be assessed and monitored with serial fundal height measurements and ultrasonography in 4-week intervals beginning at 28 weeks gestation (Leeman & Fontaine, 2008). Depending on the guidelines utilized, medical treatment may vary. Often, pregnant women with Stage I HTN do not require medical treatment other than diet modification and exercise therapy (Brown & Garovic, 2011; Leeman & Fontaine, 2008; Sullivan et al., 2011; Yoder et al., 2009). Guidelines for initiation of medical treatment of GHTN vary, but the American College of Obstetrics and Gynecology (ACOG) define a cutoff of a diastolic BP of > 105-110 mmHg or > 100 mmHg if end-organ damage is clinically apparent (Sullivan et al., 2011). Other guidelines, such as the Joint National Committee on Prevention, Detection, Evaluation, and Treatment of High Blood Pressure and the American Society of Hypertension use > 160 mmHg systolic BP as a treatment threshold (Sullivan et al., 2011; Vest & Cho, 2012). In addition, the report of the National High Blood Pressure Education Program recommends pharmacological treatment if systolic BP is > 150-160 mmHg, diastolic BP is > 100-110 mmHg, or if left ventricular hypertrophy or renal insufficiency is present (Brown & Garovic, 2011). Mild GHTN is less likely to produce end-organ damage compared to severe GHTN and treatment has not been shown to improve neonatal outcomes or prevent pre-eclampsia (Vest & Cho, 2012). Treatment at the “severe” level of HTN is often required to prevent arterial hemorrhagic events, such as cerebrovascular “strokes” and end organ damage, such as myocardial infarction (Danso & Opare-Addo, 2010; Leeman & Fontaine, 2008; Solomon & Seely, 2011; Sullivan et al., 2011; Yoder et al., 2009). Women with severe GHTN have worse perinatal outcomes compared to women with mild pre-eclampsia (Leeman & Fontaine, 2008). These higher pressures can also lead to decreased perfusion of the placenta, which may harm the fetus (Yoder et al., 2009). The immediate goal of medical anti-
hypertensive therapy is to achieve a 25% reduction of the Mean Arterial Pressure\textsuperscript{3} within 2 hours of presentation of “severe” HTN, with a goal of BP 160/110 over the next several hours (Vest & Cho, 2012; Yoder et al., 2009). Rapidly decreasing the BP could result in end organ damage in the mother as well as fetal ischemia (Yoder et al., 2009). The physician must also note additional signs and symptoms, such as proteinuria and elevated liver enzymes, which may indicate medical emergencies including pre-eclampsia and HELLP syndrome (Yoder et al., 2009).

**Treatment of GHTN.**

As for treatment, exercise and proper diet should be encouraged prior to pregnancy as lower BMIs have been associated with reductions in GHTN and a decreased risk to progress to pre-eclampsia or eclampsia (Solomon & Seely, 2011; Yoder et al., 2009). Bedrest is also an alternative non-pharmacological treatment for GHTN, but there is insufficient evidence in its effectiveness (Vest & Cho, 2012). The typical first line pharmacological agent for the treatment of severe GHTN is L-methyldopa (Solomon & Seely, 2011; Vest & Cho, 2012; Yoder et al., 2009). It is the only anti-HTN drug that has a Food and Drug Administration Class B\textsuperscript{4} rating. It is inexpensive, still available in pill form in the United States, and may be a useful first line treatment in lower income countries (Danso & Opare-Addo, 2010; Leeman & Fontaine, 2008).

Labetolol, an alpha and beta adrenergic receptor blocker, is also considered safe and efficacious during pregnancy (Danso & Opare-Addo, 2010). It is considered a first line treatment and may have fewer side effects of drowsiness, headache, and hypotension compared to methyldopa (Bulloch & Carroll, 2012). However, some studies have demonstrated intrauterine growth restriction (IUGR), exacerbation of asthma, neonatal hypotension,

\textsuperscript{3} Defined as \((\frac{1}{3})\text{Systolic BP} + (\frac{2}{3})\text{Diastolic BP}\)

\textsuperscript{4} Safe during pregnancy in animal studies, no evidence in human studies
bradycardia, and hypoglycemia due to the beta-adrenergic blockade (Nij Bijvank & Duvekot, 2010; Solomon & Seely, 2011; Yoder et al., 2009). This is the preferred beta-blocker used during pregnancy as atenolol is associated with IUGR, low birth weight, and neonatal bradycardia (Bulloch & Carroll, 2012; Sullivan et al., 2011; Vest & Cho, 2012). Labetolol is not widely used in low-resource countries, but is becoming more prevalent in the United States (Danso & Opare-Addo, 2010).

Angiotensin-converting enzyme inhibitors (ACEIs) and angiotensin receptor blockers (ARBs) are effective treatment choices for CHTN, but are contraindicated in pregnant women as they have been associated with teratogenic effects such as renal agenesis, fetal growth restriction, and fetal demise (Brown & Garovic, 2011; Leeman & Fontaine, 2008; Solomon & Seely, 2011; Sullivan et al., 2011). In fact, this drug category should be avoided in women of reproductive age, though if a woman becomes pregnant while taking ACEIs or ARBs, the medication should be stopped immediately.

Diuretics have an uncertain safety profile for the treatment of GHTN. There are theoretical concerns of electrolyte imbalances in the mother and decreased uteroplacental perfusion since the mechanism of diuretics is to decrease total blood volume (Bulloch & Carroll, 2012; Sullivan et al., 2011; Yoder et al., 2009). Also, the potassium-sparing diuretic spironolactone should not be used due to its anti-androgenic effects that can affect embryological development (Sullivan et al., 2011).

Calcium channel blockers (CCBs) are likely safe during pregnancy (Vest & Cho, 2012; Yoder et al., 2009). The typical drug used and most often evaluated is nifedipine (Nij Bijvank & Duvekot, 2010; Vest & Cho, 2012; Yoder et al., 2009). Nifedipine is often used with methyldopa in the short and long-term treatment of GHTN (Danso & Opare-Addo, 2010). There
is no evidence of major feto-maternal events with this medication (Danso & Opare-Addo, 2010). However, one side effect of nifedipine is severe headache which can mimic worsening hypertensive disease, though this effect is often ameliorated when administered with methyldopa (Danso & Opare-Addo, 2010). The dose of nifedipine must be taken into consideration as one meta-analysis found that use of a total nifedipine dose of > 60mg was associated with a significantly higher risk of side effects, including tachycardia and hypotension (Khan et al., 2010). As for additional CCBs, nicardipine may be preferred due its selectivity for blood vessels and lower risk of reflex tachycardia (Nij Bijvank & Duvekot, 2010; Vest & Cho, 2012; Yoder et al., 2009). Side effects of nicardipine occur in 4-7% of people taking this CCB and are related to vasodilation, which includes peripheral edema, flushing, and headache (Nij Bijvank & Duvekot, 2010). One meta-analysis found that nicardipine administration to pregnant women with a diastolic BP > 110mmHg resulted in a 87% success rate in studies which defined success as a 20% reduction in systolic or diastolic BP, demonstrating the high efficacy of this CCB during pregnancy (Nij Bijvank & Duvekot, 2010). There are theoretical concerns about CCB use during pregnancy since many of the embryologic growth processes are calcium dependent (Vest & Cho, 2012; Yoder et al., 2009). Another concern is if a given pregnant women is receiving prophylaxis or treatment for seizures, she would be given magnesium sulfate to increase the seizure threshold (Vest & Cho, 2012; Yoder et al., 2009). The issue is that there may be an interaction between the CCB and magnesium that could result in neuromuscular blockade or cardiac failure (Yoder et al., 2009).

Intravenous hydralazine or labetolol can be used in the event of a hypertensive emergency (Leeman & Fontaine, 2008; Nij Bijvank & Duvekot, 2010; Sullivan et al., 2011). Hydralazine can also be used in the long-term treatment for GHTN, but it must be given with
labetolol or methyldopa (Vest & Cho, 2012). Hydralyzone was shown to be less efficacious compared to nifedipine and is about equal to labetolol (Vest & Cho, 2012). One issue with hydralyzone is its side effect profile: severe headache, nausea, and vomiting. These side effects occur in approximately 50% of patients and may mimic symptoms of pre-eclampsia, possibly clouding its diagnosis (Nij Bijvank & Duvekot, 2010). Sodium nitroprusside is not typically used in hypertensive emergencies during pregnancy due to the risk of cyanide toxicity with prolonged use (Bulloch & Carroll, 2012).

**Gestational Diabetes Mellitus**

**Definition, negative health effects on the mother and fetus.**

GDM is defined as “any degree of glucose intolerance with onset or first recognition during pregnancy” (Anzaku & Musa, 2012; Carolan, Davey, Biro, & Kealy, 2012; Hedderson, Darbinian, Quesenberry, & Ferrara, 2011; Kwak, Jang, & Park, 2012; Murphy, 2010; Savona-Ventura, Vassallo, Marre, Karamanos, & MGSD-GDM study group, 2013; Torloni et al., 2009; Wong, Ross, Jalaludin, & Flack, 2013; Zhang & Ning, 2011). However, this diagnosis can be misleading as one could have undiagnosed DM1 and DM2 previous to pregnancy (Nolan, 2011). It is of great importance to diagnose this condition as early as possible as GDM has been associated with negative consequences in the mother, such as development of DMII later on in life, metabolic syndrome, delivery complications due to increased fetal size, and cardiovascular disease (Carolan, Gill, & Steele, 2012; Fraser, 2009; Malcolm, 2012; Reece, Leguizamon, & Wiznitzer, 2009; Savona-Ventura et al., 2013; Torloni et al., 2009). Depending on ethnicity, the rate of progression from GDM to DM2 ranges from 50-70% over a period of 5 to 10 years follow-up postpartum (Murphy, 2010). Expectant mothers with GDM are also at greater risk for developing GHTN, pre-eclampsia, preterm premature rupture of membranes, and having to
undergo caesarean section (Catalano et al., 2012; Kwak et al., 2012; Morisset et al., 2010; Xiong, Saunders, Wang, & Demianczuk, 2001). In addition, the fetus is put at risk to develop conditions such as macrosomia\(^5\), low birth weight due to preterm birth, and birth injuries such as shoulder dystocia that may lead to Neonatal Intensive Care Unit admissions as well as increased length of stay in the hospital (Carolan et al., 2012a; Carolan et al., 2012b; Evans, 2009; Fraser, 2009; Wong et al., 2013). As for future development, the fetus is at risk for developing childhood obesity, neurological and vascular abnormalities, and abnormal glucose intolerance, which could develop into DM2 (Akanji, 2003; Anzaku & Musa, 2012; Carolan et al., 2012; Carolan et al., 2012b; Evans, 2009; Hedderson et al., 2011; Kwak et al., 2012; Malcolm, 2012; Morisset et al., 2010; Murphy, 2010; Torloni et al., 2009; Wong et al., 2013; Zhang & Ning, 2011). Uncontrolled DM2 and GDM can also affect embryologic development including ventricular septal defects, transposition of the great vessels of the heart, spina bifida, and sacral agenesis (Evans, 2009; Fraser, 2009).

**Pathogenesis.**

During the course of pregnancy, insulin sensitivity decreases by 50-60% and levels of insulin resistance in the third trimester of pregnancy, usually between 24 and 28 weeks of gestation, are similar to those individuals with impaired glucose tolerance or newly diagnosed DM2 (Anzaku & Musa, 2012; Carpenter, 2007; Evans, 2009; Fraser, 2009; Kwak et al., 2012; Lacroix, Kina, & Hivert, 2013; Zhang & Ning, 2011). Women who develop GDM are thought to have a compromised capacity to adapt to this increased insulin demand (Fraser, 2009; Zhang & Ning, 2011). This results in impaired endothelial blood vessel cell function, vascular inflammation, and hemostasis, which leads to microvascular impairment that damages the

\(^5\) Neonatal weight at birth of > 4000 grams
placenta (Evans, 2009). Consequently, increased umbilical cord coiling and edematous placentae have been found in women with GDM and are associated with poor fetal outcomes (Evans, 2009). There is thought to be a strong genetic component to the development of GDM as certain variants of the CDKAL1 and TCF7L2 genes, along with many others, were found to be highly associated with GDM (Kwak et al., 2012). The mechanism of GDM is unlikely to be autoimmune in nature; this suggests a diagnosis of DM1 (Akanji, 2003). However, early DM1 can be unmasked during pregnancy, which may cloud the diagnosis of GDM (Fraser, 2009).

Pathogenesis II.

The overall additive effect of the factors listed above leads to the insulin secreting beta cells of the pancreas being unable to keep up with the increased demand of insulin and consequently, hyperglycemia results in both the mother and the fetus (Akanji, 2003; Evans, 2009; Kwak et al., 2012; Nolan, 2011). The majority of women who develop GDM have beta cell dysfunction against a background of chronic insulin resistance to which the insulin resistance of pregnancy is additive (Zhang & Ning, 2011). As glucose passes through the placenta, the fetus is forced to make more insulin to control the increased sugar load since maternal insulin is unable to be transferred to the fetus (Reece et al., 2009). The uptake of glucose contributes to excessive fetal growth leading to a large for gestational age fetus. This increases the likelihood that the newborn will have to be delivered by caesarean section, which has its own set of potential complications (Reece et al., 2009). Insulin resistance is multi-factorial, but is largely attributed to circulating placental hormones, which is why serum glucose concentrations often resolve during the postpartum period (Fraser, 2009). As for subsequent pregnancies, GDM is likely to recur at a frequency of 41-45% (Khambalia et al., 2013; Kwak et al., 2012). However, one must consider that GDM is multi-factorial and take into account
certain risk factors such as advanced maternal age, increased BMI, and peak serum glucose levels during previous pregnancies (Fraser, 2009).

**Risk factors.**

There have been numerous risk factors identified that may predispose expectant mothers for acquiring GDM, which include a previous history of unexplained intrauterine fetal death and previous birth with macrosomia (Akanji, 2003; Anzaku & Musa, 2012; Murphy, 2010). Other risk factors include a previous history of GDM, increased pre-pregnancy BMI, gaining excessive gestational weight, gross fetal malformations in previous births, Asian, African, Mediterranean, or Middle Eastern race, maternal age > 25, DM1 or DM2 in a first degree relative, any history of glucose intolerance or current glycosuria, and prior caesarean section (Akanji, 2003; Anzaku & Musa, 2012; Carolan et al., 2012; Carolan et al., 2012b; Carpenter, 2007; Catalano et al., 2012; Evans, 2009; Hedderson et al., 2011; Kwak et al., 2012; Morisset et al., 2010; Murphy, 2010; Savona-Ventura et al., 2013; Solomon et al., 1997; Xiong et al., 2001; Zhang & Ning, 2011).

One meta-analysis found that women with a low BMI (BMI < 20) had a risk of developing GDM 25% lower compared to women with a normal BMI (BMI 20-24) (Torloni et al., 2009). In addition, the odds ratio for developing GDM was 1.97, 3.76, and 5.55 for overweight (BMI 25-29), obese (BMI > 30), and severely obese women (BMI > 35) respectively compared with normal weight women (Torloni et al., 2009). Torloni et al found that for each 1 kg/m² increase in BMI, the prevalence of GDM increased by 0.92%. While BMI is one of the strongest predictors of GDM, the entire clinical overview of a given pregnant woman must be taken into account when screening for GDM (Fraser, 2009). Similarly, another study found that an adverse cardiometabolic risk profile, which includes hypercholesterolemia, advanced maternal age, pre-pregnancy hyperglycemia, increased BMI, and elevated pre-pregnancy blood pressure were
significant risk factors for the development of GDM (Hedderson et al., 2011). Cigarette smoking and tobacco usage have not been consistently identified as risk factors for GDM (Zhang & Ning, 2011). One study found maternal alcohol use decreased the onset of GDM, though the risks of drinking alcohol during pregnancy outweigh any potential benefit (Xiong et al., 2001).

**Epidemiology.**

The epidemiology of GDM is variable worldwide. This disease is more prevalent in non-White women, especially in Asians, who have a 5-10x higher rate of developing GDM compared to White women (Cheung, Wasmer, & Al-Ali, 2001; Fraser, 2009; Reece et al., 2009). In the USA, about 4-10% of pregnancies are affected by GDM and while rates of DM2 are increasing rapidly, the rates of GDM have increased at a slower pace through the 1990s (Carolan et al., 2012b; Fraser, 2009; Nolan, 2011; Reece et al., 2009; Savona-Ventura et al., 2013; Torloni et al., 2009; Zhang & Ning, 2011). Still, there are at least 200,000 cases of GDM in the USA per year (Morisset et al., 2010). This increase is likely due to improvements in In Vitro Fertilization leading to advanced maternal age at time of first pregnancy (Carolan et al., 2012b). In addition, the screening modality used can impact the reported prevalence of GDM (Anzaku & Musa, 2012; Fraser, 2009). The worldwide prevalence of GDM is between 1-14% though the accuracy of this estimate is difficult to determine based on whether screening is used, the modality of screening used, and differing blood glucose cutoffs used in each country (Anzaku & Musa, 2012; Fraser, 2009; Nolan, 2011).

**Screening.**

Screening for GDM has been a controversial topic over time (Fraser, 2009; Nolan, 2011). Certain organizations such as the Canadian Diabetes Association recommend universal screening, while other groups such as the American Diabetes Association recommend risk-
selective screening (Reece et al., 2009). The United States Preventative Services Task Force states that there are insufficient outcome data to recommend universal screening and the World Health Organization recommends screening in all but low-risk women in the 3rd trimester (Leary, Pettitt, & Jovanovic, 2010). The typical first screening test for GDM is known as the Oral Glucose Tolerance Test which occurs at 24-28 weeks gestation, though earlier screening may be instituted depending on whether an expectant mother is considered low risk\textsuperscript{6} or high risk\textsuperscript{7} with a random blood sugar or glycosylated hemoglobin measurement (Akanji, 2003; Leary et al., 2010; Savona-Ventura et al., 2013). Risk assessment for GDM should be undertaken by the clinician or midwife for all individuals at their first prenatal visit, particularly among “high-risk” populations (Savona-Ventura et al., 2013). However, the Hyperglycemia and Adverse Pregnancy Outcome (HAPO) Study Research Group found that abnormal Oral Glucose Tolerance Test results more strongly correlate with adverse fetal outcomes compared to glycosylated hemoglobin and should not be substituted for the Oral Glucose Tolerance Test at 24-28 weeks gestation (Lowe et al., 2012). Women with a previous diagnosis of GDM should be screened at 16-18 weeks gestation (Murphy, 2010).

**The Oral Glucose Tolerance Test.**

In order to undergo an Oral Glucose Tolerance Test, the pregnant woman ingests 50 grams of a glucose liquid and a blood glucose reading is drawn one hour after ingestion (Reece et al., 2009; Reichelt et al., 1998). If the woman registers higher than 130-140 mg/dL (or 7.2-7.8 mmol/L) then the next step is a 100 gram 3-hour Oral Glucose Tolerance Test where four blood

\textsuperscript{6} Must satisfy all criteria - Member of ethnic/racial group with low prevalence of DM1 or 2, no history of DM1 or DM2 in first degree relatives, age <25 yrs, normal pre-pregnancy weight and normal weight gain during pregnancy, no history of abnormal glucose metabolism, and no history of poor obstetric outcomes

\textsuperscript{7} Abnormal deviation of criteria of low risk category or current glycosuria
glucose measurements are taken: fasting, one hour, two hours, and three hours after ingestion. At least two values must be abnormal for the diagnosis of GDM to be given. However, this two step screening can be costly, so the World Health Organization has stated that a one-step 75 gram Oral Glucose Tolerance Test can be used in developing countries with glucose measurements being taken at fasting state and two hours after ingestion (Leary et al., 2010; Reece et al., 2009). This is also a typical modality of screening in European countries as well (Fraser, 2009). Depending on resources available, fasting blood sugars can possibly be used to diagnose GDM. One Brazilian study found that a fasting plasma glucose of 89 mg/dl or higher was able to reach a sensitivity of 88% and specificity of 78% in the detection of GDM (Reichelt et al., 1998). Savona-Ventura et al. (2013) performed a prospective non-interventional fasting blood sugar screening study in a Mediterranean population that produced similar results. This finding was also similar to results described for the 1 hour Oral Glucose Tolerance Test (Reichelt et al., 1998). However, one must note that there are many screening guidelines for GDM, which can alter sensitivities and specificities. Consequently, the HAPO study demonstrated a sensitivity of 85% for the 75-gram Oral Glucose Tolerance Test, so it is an accepted modality worldwide, though the efficacy of this test is still up for debate (Leary et al., 2010; Nolan, 2011). Additionally, genetic research may improve the prediction of onset of DM2 in women with a history of GDM (Kwak et al., 2012). Genotype testing for GDM and DM2 is not currently the norm; more research is needed. Looking toward the pregnant woman’s future, having an early diagnosis of GDM has long-term advantages for the individual as it provides an opportunity to intervene with targeted lifestyle modification and pharmacotherapy to prevent or delay the onset of DM2 and its associated complications (Savona-Ventura et al., 2013).
**Positive effects of glucose monitoring and treatment.**

The Australian Carbohydrate Intolerance Study performed a randomized clinical trial that demonstrated that women who were treated for GDM at 24-28 weeks gestation saw a decrease in perinatal complications from 4% to 1%, similar to the level of perinatal complications experienced by non-diabetic women (Anzaku & Musa, 2012; Fraser, 2009; Reece et al., 2009). These screening tests appeared to be cost-effective as for every 100 women screened, 2.2 fewer infants had perinatal complications and one less infant died (Moss et al., 2007). Therefore, it is of the upmost importance that this disease be diagnosed and treated in order to prevent complications. One recommended range for fasting blood glucose is 90-99mg/dL (5-5.5mmol/L), one hour post-prandial glucose < 140mg/dL (7.8mmol/L) and two hour post-prandial glucose < 120-127mg/dL (6.7-7.1mg/dL) (Reece et al., 2009). Pregnant women can constantly monitor their blood glucose using a home blood glucose monitor. This method has demonstrated reduced levels of glycosylated hemoglobin in pregnant women (Reece et al., 2009). On the other hand, glucose monitoring and subsequent nutritional management is controversial and studies have produced mixed results in their impact against adverse fetal outcomes such as macrosomia (Fraser, 2009; Reece et al., 2009).

**Diet as treatment for GDM.**

Dietary counseling has long been recommended for women who develop GDM (Zhang & Ning, 2011). The American Diabetes Association recommends restricting carbohydrate consumption to 35-40% of daily intake as it may decrease maternal glucose concentrations and improve fetal outcomes (Reece et al., 2009). Certain types of carbohydrates may be protective against GDM, especially dietary fiber and whole grains (Zhang & Ning, 2011). The elimination of unhealthy and “junk” foods, especially sugar sweetened beverages and foods high in saturated
fatty acids, is key in obtaining a normal fasting and post-prandial blood glucose (Carolan et al., 2012b; Morisset et al., 2010; Nolan, 2011; Zhang & Ning, 2011). The mechanism on the consumption of red meat and processed meats on the development of GDM has not yet been determined (Zhang & Ning, 2011). One hypothesis is that both of these food products are preserved with nitrates which have shown to be toxic to pancreatic beta cells in animal experiments (Zhang & Ning, 2011). In addition, the iron content of red meat may increase the risk of GDM as body iron overload has been hypothesized to promote insulin resistance and increase the risk of DM2 (Zhang & Ning, 2011). Conversely, consuming additional polyunsaturated fats may be protective against GDM (Zhang & Ning, 2011). Often, dietary changes can be difficult due to low socio-economic status and pre-existing cultural norms (Carolan et al., 2012b). Emphasizing fetal well-being as well as support from partners, family, and friends are seen as positive motivators toward dietary changes (Carolan et al., 2012b).

**Exercise as treatment for GDM.**

One method of preventing GDM is prenatal exercise (Zhang & Ning, 2011). Exercise is hypothesized to positively affect glucose homeostasis by increasing insulin sensitivity and secretion (Zhang & Ning, 2011). A study by Zhang, Solomon, Manson, and Hu (2006) demonstrated that women who spent < 2 hours per week watching television and performed “vigorous exercise” were less likely to develop GDM compared to women who watched > 20 hours of television per week and did not exercise. Another study showed that women who exercised throughout their pregnancies were less likely to deliver a large for gestational age infant (Snapp & Donaldson, 2008). This increase in exercise is encouraged to boost the woman’s metabolism and it may reduce post-gestational morbidity to the fetus among women with prior GDM (Carolan et al., 2012b; Carpenter, 2007). There may be long-term improvement
in glucose tolerance that may result from increased physical activity due to decreases in fat mass and increases in lean muscle mass (Zhang & Ning, 2011). While this is a useful preventative measure previous to pregnancy, it is not as practical as a short-term treatment as GDM is typically diagnosed during the second or third trimester (Lacroix et al., 2013; Nolan, 2011; Zhang & Ning, 2011).

**Insulin and barriers to treatment in GDM.**

Most pregnant women with GDM become normoglycemic with diet and exercise with only 10-20% requiring medical treatment (Murphy, 2010). Currently, the only Food and Drug Administration approved medical option for treatment of GDM is insulin (Nolan, 2011). The two rapid acting derivatives that are typically used are Lispro and Aspart, both of which have been shown to be safe and clinically effective during pregnancy (Reece et al., 2009). The lowest possible dose should be used as high levels of insulin may increase the woman’s risk for developing DM2 in the future (Carolan et al., 2012b). Barriers to effective treatment include failure to understand and make sense of the GDM diagnosis, failure to understand the urgency of immediate change of lifestyle during pregnancy, finding time to prepare healthy meals, failure to attend follow-up appointments with medical specialists, disruption to family routine, and lack of support from family or friends (Carolan et al., 2012b). There is controversy over when to deliver pregnant women who acquire GDM, but induction of labor at 38 weeks gestation may lead to better birth outcomes (Fraser, 2009).

**Oral medications in the treatment of GDM.**

Though there have been reports of adverse fetal outcomes when using oral anti-diabetic agents, metformin may still have protective benefits for the fetus and could decrease insulin resistance in the mother (Nolan, 2011). In addition, there is decreased burden on the mother as it
is less painful to take oral medications rather than receive insulin shots (Fraser, 2009). Metformin is also not associated with maternal hypoglycemia or weight gain (Murphy, 2010). Its benefit may be due to its vascular protective effects (Evans, 2009). More research on human subjects is needed to establish the safety of metformin during pregnancy as it can cross the placenta and possibly disrupt fetal growth (Fraser, 2009; Murphy, 2010; Nolan, 2011).

Conclusion

As GHTN and GDM appear to be rising problems, it is important to identify expectant mothers with identified risk factors and screen them appropriately in order to ensure the health and well-beings of the mother and newborn. In fact, the HAPO study demonstrated that blood glucose levels that were mildly elevated in the expectant mother, but did not meet the cutoff for GDM were still associated with poor birth outcomes such as shoulder dystocia and hyperinsulinemia in the newborn (Nolan, 2011). This study seeks to replicate previously identified demographic risk factors for GHTN and GDM in women giving birth in Ohio during 2012. The goal is to assist physicians in their risk assessment of high-risk pregnancies for the development of GHTN and/or GDM. The results will hopefully lead to increased awareness, screening, and treatment of both of these disorders.

Methods

Approval from Wright State University’s IRB committee was obtained through exempt review. Retrospective Ohio Birth data for 2012 was obtained from the Ohio Department of Health through Public Health-Dayton & Montgomery County. The primary outcomes assessed were binary responses, either yes or no, whether a given pregnant woman had developed GDM or GHTN. Data points where the mother’s GDM or GHTN status was unknown were excluded from the calculations. IBM Statistical Package for Social Sciences for Mac Version 20 (SPSS,
IBM, 2011) was used to analyze descriptive statistics including overall prevalence of GDM and GHTN, prevalence of each condition within independent variable groups, unadjusted odds ratios, and chi-square analyzes of each independent variable with the outcome of GHTN and GDM separately. Forward stepwise multiple logistic regression was performed for each independent variable with GDM or GHTN as the main outcome. The first step included the following variables: age, race, pre-pregnancy BMI, and delivery BMI being inputted using the “enter” function. The second step included the stepwise addition of other medical co-morbidities, which included pre-pregnancy HTN or DM1 and DM2, cigarettes smoked before and during pregnancy, and contracting a sexually transmitted infection (STI) during pregnancy. The third step included the addition of other demographic variables such as medical services payment method, usage of the Women, Infants, and Children (WIC) program, and maternal education. The definitions for each variable are described in Table 1.
Table 1. *Methods: Variables Analyzed*

<table>
<thead>
<tr>
<th>Variable Type</th>
<th>Variable</th>
<th>Measurement</th>
<th>Comments</th>
</tr>
</thead>
<tbody>
<tr>
<td>Dependent</td>
<td>Gestational Diabetes Mellitus</td>
<td>Y or N</td>
<td></td>
</tr>
<tr>
<td>Dependent</td>
<td>Gestational Hypertension</td>
<td>Y or N</td>
<td></td>
</tr>
<tr>
<td>Independent</td>
<td>Marital Status</td>
<td>Y or N</td>
<td></td>
</tr>
<tr>
<td>Independent</td>
<td>Pre-pregnancy Hypertension</td>
<td>Y or N</td>
<td>Not analyzed with GHTN</td>
</tr>
<tr>
<td>Independent</td>
<td>Pre-pregnancy Diabetes Mellitus</td>
<td>Y or N</td>
<td>Not analyzed with GDM</td>
</tr>
<tr>
<td>Independent</td>
<td>Maternal Education</td>
<td>1. Some college or higher vs. high school degree or lower&lt;br&gt;2. High school degree or lower, some college to bachelor’s degree, graduate education obtained</td>
<td>1. Chi-Square Analysis&lt;br&gt;2. Forward Stepwise Multivariate Logistic Regression</td>
</tr>
<tr>
<td>Independent</td>
<td>Cigarettes Smoked - 3 months Pre-Pregnancy - 1st Trimester - 2nd Trimester - 3rd Trimester</td>
<td>1. Y or N&lt;br&gt;2. Continuous</td>
<td>1. Chi-Square Analysis&lt;br&gt;2. Forward Stepwise Multivariate Logistic Regression</td>
</tr>
<tr>
<td>Independent</td>
<td>Sexually Transmitted Infections</td>
<td>Y or N</td>
<td>Includes Gonorrhea, Syphilis, Herpes Simplex Virus, Chlamydia, Hepatitis B, Hepatitis C</td>
</tr>
<tr>
<td>Independent</td>
<td>Race</td>
<td>White, Black, Native American, &quot;Asian&quot;</td>
<td>&quot;Asian&quot; group consists of Chinese, Japanese, Filipino, Hawaiian, and &quot;Other Asian&quot;</td>
</tr>
<tr>
<td>Independent</td>
<td>Payment type</td>
<td>Private Insurance/Tricare/CHAMPUS, Medicaid, Self-Pay</td>
<td>1. Medicaid vs. All other groups and Private Insurance vs. All other groups&lt;br&gt;2. All other groups vs. Private Insurance/Tricare/CHAMPUS as control</td>
</tr>
</tbody>
</table>
For the variable “Maternal Education”, groups were divided into having completed <8th grade, 9-12th grade, high school, some college, an associate’s degree, a bachelor’s degree, and a doctoral degree. These were combined into binary variables of “Less than a college degree” and “At least an associate’s degree” for the chi-square analysis. For the forward stepwise multivariate logistic regression, this variable was divided into three groups: High School degree or less, some college through Bachelor’s degree, and Attained Graduate Level Education.

For the maternal age category, age was initially recorded as a continuous variable. The group was recalculated into binary variables of ≤ 29 years of age and ≥ 30 years for the chi-square analysis. The continuous variable was used for the forward stepwise multivariate logistic regression.

Maternal pre-pregnancy and delivery BMI, calculated as Weight (kg)/ Height (meters)^2, was collapsed into the following groups: ≤ 19, 20-24, 25-29, 30-34, and ≥ 35. Binary groups were created (≤ 19 – 24 and ≥ 25) for the chi-square and odds ratio analysis. A logistic regression was used to compare each separate group with the 20-24 BMI group acting as the control. In the forward stepwise multivariate logistic regression:

- BMI was added as a continuous variable.
- Marriage was defined as whether a given pregnant women was married, denoted by yes or no.
- Cigarette usage was defined as a continuous variable denoting the number of cigarettes smoked three months pre-pregnancy and during each trimester. These groups were collapsed into categorical variables of whether a given pregnant woman had smoked at all during the given time period for the chi-square and odds
ratio analysis. For the forward stepwise multivariate logistic regression, “cigarettes smoked” was added as a continuous variable.

- The payment variable was defined as whether each pregnant woman had Medicaid, Private Insurance, Self Pay, CHAMPUS/Tricare, or “Other Government Insurance”. As access to medical care is similar for Private and Government insurances, these groups were combined. For the chi-square and odds ratio analysis, groups were combined into binary categorical variables of “Medicaid” versus “All Other Payment Types” as well as Private Health Insurance versus “All Other Payment Types”.

- Pre-pregnancy HTN and pre-pregnancy DM1 and DM2 were defined as binary responses as “yes” and “no”. Pre-pregnancy HTN was not analyzed with GHTN as an outcome and pre-pregnancy DM was not analyzed with GDM as an outcome because a given woman can only have one of these designations.

- The WIC program is a federal assistance program, which provides nutritional education and supplementation to pregnant or postpartum women and their children if they are 185% below the national poverty line. This variable was included to assess socioeconomic status (SES). This variable was defined as whether a given pregnant women had used this program, denoted by “yes” or “no”. Information about the percentage of women who qualified for WIC was not available.

- Maternal race was separated into White, African, Native American, and “Asian”, which is defined as Chinese, Japanese, Hawaiian, and Filipino.
STI was defined as whether a given pregnant woman contracted Gonorrhea, Syphilis, HSV, Chlamydia, Hepatitis B, or Hepatitis C during the time that they were pregnant. Each disease was combined into an “Any Infection” group for both the chi-square analysis and the multivariate forward stepwise logistic regression. Individual diseases were evaluated during the chi-square analysis as well.

Results

Gestational Diabetes Mellitus (GDM)

There were 137,268 women in our sample who had valid GDM data points. 8,454 women (6.2%) had GDM. Statistically significant odds ratios are shown in Figure 1. The chi-square and odds ratio results are shown in Table 2.

Figure 1. Statistically significant odds ratios for GDM risk factors. * p-value <.05, ** p-value < .01
Table 2. *Chi-Square and Odds Ratios for GDM*

<table>
<thead>
<tr>
<th>Variable</th>
<th>Chi-Square</th>
<th>Significance (p-value)</th>
<th>Odds Ratio (OR)</th>
<th>95% CI Lower</th>
<th>95% CI Higher</th>
</tr>
</thead>
<tbody>
<tr>
<td>Marital Status</td>
<td>169.089</td>
<td>&lt; .001**</td>
<td>1.354</td>
<td>1.293</td>
<td>1.417</td>
</tr>
<tr>
<td>Pre-pregnancy BMI</td>
<td>411.98</td>
<td>&lt; .001**</td>
<td>2.783</td>
<td>2.511</td>
<td>3.085</td>
</tr>
<tr>
<td>Delivery BMI</td>
<td>163.31</td>
<td>&lt; .001**</td>
<td>2.169</td>
<td>1.921</td>
<td>2.449</td>
</tr>
<tr>
<td>Cigarettes Smoked 3 Months before Pregnancy</td>
<td>0.005</td>
<td>0.943</td>
<td>0.998</td>
<td>0.948</td>
<td>1.051</td>
</tr>
<tr>
<td>Cigarettes Smoked during 1st Trimester</td>
<td>6.58</td>
<td>.01**</td>
<td>0.926</td>
<td>0.873</td>
<td>0.982</td>
</tr>
<tr>
<td>Cigarettes Smoked during 2nd Trimester</td>
<td>10.53</td>
<td>.001**</td>
<td>0.902</td>
<td>0.847</td>
<td>0.960</td>
</tr>
<tr>
<td>Cigarettes Smoked during 3rd Trimester</td>
<td>12.277</td>
<td>.001**</td>
<td>0.892</td>
<td>0.837</td>
<td>0.951</td>
</tr>
<tr>
<td>Pre-pregnancy HTN</td>
<td>411.984</td>
<td>&lt; .001**</td>
<td>2.783</td>
<td>2.511</td>
<td>3.085</td>
</tr>
<tr>
<td>Maternal College Education versus No College Education</td>
<td>21.816</td>
<td>&lt; .001**</td>
<td>1.113</td>
<td>1.064</td>
<td>1.164</td>
</tr>
<tr>
<td>Any Sexually Transmitted Infection</td>
<td>4.737</td>
<td>.03*</td>
<td>0.897</td>
<td>0.814</td>
<td>0.989</td>
</tr>
<tr>
<td>Gonorrhea</td>
<td>4.556</td>
<td>.033*</td>
<td>0.663</td>
<td>0.454</td>
<td>0.969</td>
</tr>
<tr>
<td>Chlamydia</td>
<td>25.217</td>
<td>&lt; .001**</td>
<td>0.653</td>
<td>0.552</td>
<td>0.772</td>
</tr>
<tr>
<td>Syphilis</td>
<td>0.188</td>
<td>.665</td>
<td>1.185</td>
<td>0.549</td>
<td>2.558</td>
</tr>
<tr>
<td>HSV</td>
<td>2.649</td>
<td>.104</td>
<td>1.114</td>
<td>0.978</td>
<td>1.268</td>
</tr>
<tr>
<td>Hepatitis B</td>
<td>6.907</td>
<td>.009*</td>
<td>1.695</td>
<td>1.138</td>
<td>2.524</td>
</tr>
<tr>
<td>Hepatitis C</td>
<td>1.033</td>
<td>.309</td>
<td>0.824</td>
<td>0.567</td>
<td>1.198</td>
</tr>
<tr>
<td>Maternal Race</td>
<td>1.799</td>
<td>.180</td>
<td>---</td>
<td>---</td>
<td>---</td>
</tr>
<tr>
<td>African Race</td>
<td>1.799</td>
<td>.180</td>
<td>0.955</td>
<td>0.909</td>
<td>1.024</td>
</tr>
<tr>
<td>Asian Race</td>
<td>10.906</td>
<td>.207</td>
<td>0.896</td>
<td>0.779</td>
<td>1.048</td>
</tr>
<tr>
<td>Maternal Age</td>
<td>0.294</td>
<td>0.588</td>
<td>1.013</td>
<td>0.967</td>
<td>1.060</td>
</tr>
<tr>
<td>WIC</td>
<td>0.439</td>
<td>0.507</td>
<td>0.931</td>
<td>0.754</td>
<td>1.150</td>
</tr>
<tr>
<td>Private Insurance vs. Other Types</td>
<td>52.337</td>
<td>&lt; .001**</td>
<td>1.176</td>
<td>1.126</td>
<td>1.229</td>
</tr>
<tr>
<td>Medicaid vs all other Payment types</td>
<td>35.911</td>
<td>&lt; .001**</td>
<td>0.868</td>
<td>0.829</td>
<td>0.909</td>
</tr>
</tbody>
</table>

*p < .05, ** p < .01

**Marital status.**

In our sample, 5,390 women with GDM were married compared with 3,041 women who were not. This difference was found to be statistically significant ($\chi^2 = 169.089, p < .001$). Women who were married had 1.354 odds of developing GDM (95% CI = 1.293 – 1.417) compared to non-married women.
Maternal education.

In our sample, 3,372 women (40%) of women with GDM had at least an associates degree or higher. Women who had attained higher education was found to be statistically significant ($\chi^2 = 21.816, p < .001$) and had 1.113 greater odds (95% CI = 1.064 – 1.164) of developing GDM compared to women who had not attained a college degree.

Pre-pregnancy HTN.

In our sample, 447 (5.2%) women with GDM also had pre-pregnancy HTN. Pre-pregnancy HTN was significantly associated with the development of GDM ($\chi^2 = 411.984, p < .001$). Women with pre-pregnancy HTN had 2.783 odds (95%CI = 2.511 – 3.085) of developing GDM compared to pregnant women who were normotensive.

Race.

Race was found to be a non-significant factor in the development of GDM ($\chi^2 = 10.906, p = .207$). Even when the Chinese, Japanese, Hawaiian, Filipino, and Other Asian categories were combined into one group and compared against the White, Black, and Native American groups, no significant differences were found ($\chi^2 = 1.799, p = .180$). There were no significant differences between the African group ($\chi^2 = 1.378, p = .240$) and the Asian group ($\chi^2 = 1.378, p = .240$) compared to the other races in the development of GDM.

STI’s.

In our sample, 453 (5.3%) women who developed GDM reported that they had contracted an STI during their pregnancy. This finding was statistically significant in a negative association with the development of GDM ($\chi^2 = 4.737, p = .03$)(OR = .897, 95% CI = .814 - .989). When broken down into individual diseases, women who had contracted Gonorrhea ($\chi^2 = 4.566, p = .033$)(OR = .663, 95% CI = .454 -.969), and Chlamydia ($\chi^2 = 25.217, p < .001$)(OR =
.653, 95% CI = .552 - .772) were less likely to develop GDM. Women who contracted Hepatitis B were more likely to develop GDM ($\chi^2 = 6.907, p = .009$) (OR = 1.695, 95% CI = 1.138 – 2.524). There was no significant association with GDM and Syphilis, HSV, and Hepatitis C.

**Cigarette usage.**

In our sample, 1,983 (23.4%) women who developed GDM had smoked cigarettes prior to getting pregnant. There was no statistically significantly association between these two factors ($\chi^2 = 0.005, p = .943$). Women who smoked during the first trimester of pregnancy were significantly less likely to develop GDM ($\chi^2 = 6.580, p = .01$)(OR = .926, 95%CI = .873 - .982). Women who smoked during the second trimester of pregnancy were significantly less likely to develop GDM ($\chi^2 = 10.530, p = .001$)(OR = .902, 95%CI = .847 - .960). Women who smoked during the third trimester of pregnancy were significantly less likely to develop GDM ($\chi^2 = 12.277, p < .001$)(OR = .892, 95%CI = .837 - .951)

**WIC.**

In our sample, 2,783 women (44%) utilized WIC. There was no significant association between WIC usage and GDM ($\chi^2 = 0.439, p = 0.507$).

**Pre-pregnancy BMI.**

The mean pre-pregnancy BMI in our sample was 26.47. When analyzed as binary groups, there was a significant difference in the development of GDM when one’s BMI was ≥ 25 ($\chi^2 = 1664.424, p < .001$)(OR = 2.899, 95%CI = 2.749 – 3.057). When compared to the 20-24 BMI group, the ORs for developing GDM were 0.717 (95%CI = 0.620 – 0.828), 1.748 (95% CI = 1.625 – 1.879), 2.736 (95%CI = 2.533 – 2.954), and 4.334 (95%CI = 4.043 – 4.645) for the ≤ 19, 25-29, 30-34, and ≥35 groups respectively. These results are displayed in Figure 2.
**Delivery BMI.**

The mean delivery BMI in our sample was 31.91. When analyzed as binary groups, there was a significant difference in the development of GDM in women who had a BMI $\geq 25$ ($\chi^2 = 163.610, p < .001$)(OR = 2.169, 95%CI = 1.921 – 2.449). When compared to the 20-24 BMI group, the ORs for developing GDM were 1.180 (95% CI = 1.033 – 1.348), 1.813(95%CI = 1.591 – 2.066), and 3.638 (95%CI = 3.208 – 4.125) for the $\leq 19$, 25-29, 30-34, and $\geq 35$ groups respectively.

**Maternal age.**

The mean age of our sample was 27.32. When analyzed as binary variables of $\leq 29$ years of age and $\geq 30$ years of age, there were no significant differences between age groups in developing GDM ($\chi^2 = 0.294, p = 0.558$).
Payment type.

The majority of our sample had private insurance/Tricare/CHAMPUS (n = 65565, 46.3%) or Medicaid (n = 52703, 37.2%). Women with Medicaid were less likely to develop GDM compared to women with other payment types (\(\chi^2 = 35.911, p = < .001\)) (OR = 0.868, 95% CI = 0.829 – 0.909). Women with private insurance/Tricare/CHAMPUS had higher odds of developing GDM compared to Medicaid and Self-Pay (\(\chi^2 = 42.75, p < .001\)) (OR = 1.176, 95% CI = 1.126 – 1.229).

Forward stepwise multivariate logistic regression.

After forward stepwise multivariate logistic regression, three variables remained significant. Pre-pregnancy BMI (B = 0.091, S.E. = 0.017, p < .001, OR = 1.095, 95%CI = 1.059 – 1.133) was determined to confer higher odds of developing GDM. Asian race was found to be protective against the development of GDM (B = -0.44, S.E. 0.0162, p = 0.006, OR = 0.642, 95%CI = 0.467 – 0.881). In addition, unmarried women were less likely to develop GDM (B = -0.689, S.E. = 0.137, p < .001, OR = 0.507, 95%CI = 0.386 – 0.661). These results are shown in Table 3. These variables explained 8.1% of the total variance. All other independent variables were not statistically significant.

Table 3. Forward Stepwise Multivariate Logistic Regression for GDM- Significant Variables

<table>
<thead>
<tr>
<th>Characteristic</th>
<th>Slope (B)</th>
<th>Standard Error (S.E.)</th>
<th>Significance (p-value)</th>
<th>OR</th>
<th>95% CI (Lower)</th>
<th>95% CI (Higher)</th>
</tr>
</thead>
<tbody>
<tr>
<td>Pre-Pregnancy BMI</td>
<td>0.091</td>
<td>0.017</td>
<td>&lt; .001**</td>
<td>1.095</td>
<td>1.059</td>
<td>1.133</td>
</tr>
<tr>
<td>Asian Race (as compared to White)</td>
<td>0.444</td>
<td>0.0162</td>
<td>.006*</td>
<td>0.642</td>
<td>0.467</td>
<td>0.881</td>
</tr>
<tr>
<td>Marital Status (Married as Control)</td>
<td>0.689</td>
<td>0.137</td>
<td>&lt; .001**</td>
<td>0.507</td>
<td>0.386</td>
<td>0.661</td>
</tr>
<tr>
<td>Constant</td>
<td>4.084</td>
<td>0.413</td>
<td>&lt; .001**</td>
<td>0.017</td>
<td></td>
<td></td>
</tr>
</tbody>
</table>

* p-value < .05 ** p-value < .001
Gestational Hypertension (GHTN)

There were 137,270 women in our sample who had valid GHTN data points; 7,568 women in our sample (5.5%) had GHTN. The statistically significant variables are shown in Figure 3. The chi-square and odds ratio results are shown in Table 4.

*Figure 3. Statistically significant odds ratios for GHTN risk factors.*

* p-value < .05,  ** p-value < .01
Table 4. *Chi-Square and Odds Ratios for GHTN*

<table>
<thead>
<tr>
<th>Variable</th>
<th>Chi-Square</th>
<th>Significance (p-value)</th>
<th>Odds Ratio (OR)</th>
<th>95% CI (Lower)</th>
<th>95% CI (Higher)</th>
</tr>
</thead>
<tbody>
<tr>
<td>Marital Status</td>
<td>14.638</td>
<td>&lt; .001**</td>
<td>0.913</td>
<td>0.871</td>
<td>0.957</td>
</tr>
<tr>
<td>Pre-pregnancy BMI</td>
<td>1067.517</td>
<td>&lt; .001**</td>
<td>2.404</td>
<td>2.277</td>
<td>2.537</td>
</tr>
<tr>
<td>Delivery BMI</td>
<td>225.410</td>
<td>&lt; .001**</td>
<td>2.921</td>
<td>2.522</td>
<td>3.382</td>
</tr>
<tr>
<td>Cigarettes Smoked 3 Months before Pregnancy</td>
<td>42.129</td>
<td>&lt; .001**</td>
<td>0.827</td>
<td>0.781</td>
<td>0.876</td>
</tr>
<tr>
<td>Cigarettes Smoked during 1st Trimester</td>
<td>74.703</td>
<td>&lt; .001**</td>
<td>0.747</td>
<td>0.700</td>
<td>0.799</td>
</tr>
<tr>
<td>Cigarettes Smoked during 2nd Trimester</td>
<td>90.206</td>
<td>&lt; .001**</td>
<td>0.719</td>
<td>0.670</td>
<td>0.772</td>
</tr>
<tr>
<td>Cigarettes Smoked during 3rd Trimester</td>
<td>92.116</td>
<td>&lt; .001**</td>
<td>0.703</td>
<td>0.654</td>
<td>0.756</td>
</tr>
<tr>
<td>Pre-pregnancy DM</td>
<td>0.800</td>
<td>&lt; .001**</td>
<td>2.292</td>
<td>1.925</td>
<td>2.728</td>
</tr>
<tr>
<td>Maternal College Education versus No College Education</td>
<td>27.038</td>
<td>.371</td>
<td>1.022</td>
<td>0.974</td>
<td>1.072</td>
</tr>
<tr>
<td>Any Sexually Transmitted Infection</td>
<td>16.669</td>
<td>&lt; .001**</td>
<td>1.268</td>
<td>1.159</td>
<td>1.388</td>
</tr>
<tr>
<td>Gonorrhea</td>
<td>33.459</td>
<td>&lt; .001**</td>
<td>1.722</td>
<td>1.322</td>
<td>2.243</td>
</tr>
<tr>
<td>Chlamydia</td>
<td>6.327</td>
<td>&lt; .001**</td>
<td>1.449</td>
<td>1.277</td>
<td>1.644</td>
</tr>
<tr>
<td>Syphilis</td>
<td>3.182</td>
<td>.022*</td>
<td>2.194</td>
<td>1.171</td>
<td>4.111</td>
</tr>
<tr>
<td>HSV</td>
<td>0.056</td>
<td>0.074</td>
<td>1.131</td>
<td>0.988</td>
<td>1.296</td>
</tr>
<tr>
<td>Hepatitis B</td>
<td>1.744</td>
<td>.813</td>
<td>0.937</td>
<td>0.547</td>
<td>1.606</td>
</tr>
<tr>
<td>Hepatitis C</td>
<td>15.031</td>
<td>0.187</td>
<td>0.760</td>
<td>0.504</td>
<td>1.144</td>
</tr>
<tr>
<td>Maternal Race</td>
<td>7.222</td>
<td>.059</td>
<td>---</td>
<td>---</td>
<td>---</td>
</tr>
<tr>
<td>African Race</td>
<td>0.013</td>
<td>.007*</td>
<td>1.087</td>
<td>1.023</td>
<td>1.155</td>
</tr>
<tr>
<td>Asian Race</td>
<td>1.648</td>
<td>.091</td>
<td>1.009</td>
<td>0.869</td>
<td>1.171</td>
</tr>
<tr>
<td>Maternal Age</td>
<td>0.164</td>
<td>0.199</td>
<td>0.969</td>
<td>0.923</td>
<td>1.017</td>
</tr>
<tr>
<td>WIC</td>
<td>0.164</td>
<td>.686</td>
<td>0.969</td>
<td>0.923</td>
<td>1.017</td>
</tr>
<tr>
<td>Private Insurance vs. Other Types</td>
<td>42.75</td>
<td>&lt; .001**</td>
<td>1.167</td>
<td>1.114</td>
<td>1.223</td>
</tr>
<tr>
<td>Medicaid vs all other Payment types</td>
<td>0.083</td>
<td>0.774</td>
<td>1.007</td>
<td>0.960</td>
<td>1.056</td>
</tr>
</tbody>
</table>

*p < .05, ** p < .01

**Marital status.**

In our sample, 4,142 (55%) women with GHTN were married compared with 3,386 women with GHTN who were not. This difference was found to be statistically significant ($\chi^2 = 14.638, p < .001$). Women who were married had 0.913 odds of developing GHTN (95% CI = 0.871 -0.957) compared to non-married women.
Maternal education.

In our sample, 2,875 women (38%) of women with GHTN had at least an associate’s degree or higher. Having higher education was not found to be statistically significant in developing GHTN compared to women who had not attained a college degree ($\chi^2 = 0.800, p = 0.371$).

Pre-pregnancy DM.

In our sample, 146 (2%) women who had pre-pregnancy DM developed GHTN. This factor was statistically significant in the development of GHTN ($\chi^2 = 92.116, p < .001$). Women with pre-pregnancy DM had 2.292 greater odds of developing GHTN (95%CI = 1.925- 2.728) compared to women without pre-pregnancy DM.

Race.

As a whole, race was found to be a non-significant factor in the development of GHTN ($\chi^2 = 15.031, p = .059$). During individual analysis, Africans were more likely to develop GHTN compared to other races ($\chi^2 = 7.222, p = .007$)(OR = 1.087, 95%CI = 1.023 – 1.155). Asians were not significantly more likely to develop GHTN compared to other races ($\chi^2 = 0.013, p = 0.910$)(OR = 1.009, 95%CI = 0.869-1.171).

STI’s.

In our sample, 550 (7.3%) women who developed GHTN reported that they had contracted an STI at some point in their life. This finding was statistically significant in the development of GHTN ($\chi^2 = 27.038, p < .001$)(OR = 1.268, 95% CI = 1.159 – 1.388). When broken down into individual diseases, women who contracted Gonorrhea ($\chi^2 = 16.669, p < .001$)(OR = 1.722, 95%CI = 1.322 -2.243), Syphilis ($\chi^2 = 6.327, p = .012$)(OR = 2.194, 95%CI = 1.171 – 4.111), and Chlamydia ($\chi^2 = 33.459, p < .001$)(OR = 1.449, 95%CI = 1.277 – 1.644)
were more likely to develop GHTN. There was no significant association with GHTN and HSV, Hepatitis B, and Hepatitis C.

**Cigarette usage.**

In our sample, 1,545 (20.5%) women who developed GHTN had smoked cigarettes prior to getting pregnant. Women who smoked prior to pregnancy were significantly less likely to develop GHTN ($\chi^2 = 42.129, p < .001$) (OR = 0.827, 95%CI = 0.781 -0.876). Women who smoked during the first trimester of pregnancy were significantly less likely to develop GDM ($\chi^2 = 74.703 p < .001$)(OR = .747, 95%CI = 0.700 - 0.799). Women who smoked during the second trimester of pregnancy were significantly less likely to develop GDM ($\chi^2 = 83.445, p < .001$)(OR = 0.719, 95%CI = 0.670 – 0.772). Women who smoked during the 3rd trimester of pregnancy were significantly less likely to develop GDM ($\chi^2 = 90.206, p = < .001$)(OR = 0.703, 95%CI = 0.654 – 0.756).

**WIC.**

In our sample, 2,783 women (44%) utilized WIC. There was no significant association between WIC usage and GHTN ($\chi^2 = 0.164, p = 0.686$)(OR = 0.954, 95%CI = 0.761 – 1.197).

**Pre-pregnancy BMI.**

When analyzed as binary groups, there was a significant difference in the development of GHTN when one’s BMI was $\geq 25$ ($\chi^2 = 1067.517, p < .001$)(OR = 2.404, 95%CI = 2.277 – 2.537). When compared to the 20-24 BMI group, the ORs for developing GHTN were 0.676 (95%CI = 0.584-0.783), 1.645 (95% CI = 1.529 -1.770), 2.254 (95%CI = 2.080-2.442), and 3.214 (95%CI = 2.987 -3.457) for the $\leq 19$, 25-29, 30-34, and $\geq 35$ groups respectively.
**Delivery BMI.**

When analyzed as binary groups, there was a significant difference in the development of GHTN when one’s BMI was ≥ 25 ($\chi^2 = 225.410$, $p = < .001$)(OR = 2.921, 95%CI = 2.522 – 3.382). When compared to the 20-24 BMI group, the ORs for developing GDM were 1.394 (95% CI = 1.187 -1.636), 2.578 (95%CI = 2.206 - 3.012), and 5.046 (95%CI = 4.337-5.871) for the 25-29, 30-34, and ≥ 35 groups respectively. There were no significant differences in developing GHTN in the ≤ 19 BMI group compared to the 20-24 group. These results are shown in Figure 4.

![Figure 4. Delivery BMI vs. Odds of developing GHTN.](image)

**Maternal age.**

When analyzed as binary variables of ≤ 29 years of age and ≥ 30 years of age, there were no significant differences between age groups in developing GHTN ($\chi^2 = 1.648$, $p = 0.199$).
Payment type.

There were no significant differences between women with Medicaid in the development of GHTN compared to women who utilized other payment types ($\chi^2 = 0.083, p = 0.774$). Women with private insurance/Tricare/CHAMPUS had higher odds of developing GHTN compared to women with Medicaid or Self-pay ($\chi^2 = 42.750, p < .001$)(OR $= 1.167, 95\% CI = 1.114-1.223$)

**Forward Stepwise Multiple Logistic Regression.**

After forward stepwise multiple logistic regression, only three variables remained statistically significant. Women with increased delivery BMI had higher odds of developing GHTN ($B = 0.096, S.E. = 0.019, p < .001, OR = 1.101, 95\% CI = 1.061-1.141$). Women with Medicaid ($B = -0.604, S.E. = 0.142, p = .001, OR = 0.642, 95\% CI = 0.413 - 0.723$) and who self-paid for medical services ($B = -0.836, S.E. = 0.425, p = .049, OR = 0.433, 95\% CI = 0.188 - 0.996$) were significantly less likely to develop GHTN compared to women with private insurance. These results are displayed in Table 5. Approximately 7.5% of the variance was explained by these variables. All other independent variables were not statistically significant after the logistic regression.

<table>
<thead>
<tr>
<th>Characteristic</th>
<th>Slope (B)</th>
<th>Standard Error (S.E.)</th>
<th>Significance (p-value)</th>
<th>OR</th>
<th>95% CI (Lower)</th>
<th>95% CI (Higher)</th>
</tr>
</thead>
<tbody>
<tr>
<td>Delivery BMI</td>
<td>0.096</td>
<td>0.019</td>
<td>&lt; .001**</td>
<td>1.101</td>
<td>1.061</td>
<td>1.141</td>
</tr>
<tr>
<td>Medicaid as compared to Private Insurance</td>
<td>-0.604</td>
<td>0.142</td>
<td>.001*</td>
<td>0.642</td>
<td>0.413</td>
<td>0.723</td>
</tr>
<tr>
<td>Self-Pay as compared to Private Insurance</td>
<td>-0.836</td>
<td>0.425</td>
<td>&lt; .049*</td>
<td>0.433</td>
<td>0.188</td>
<td>0.996</td>
</tr>
<tr>
<td>Constant</td>
<td>-5.196</td>
<td>0.413</td>
<td>&lt; .001**</td>
<td>0.006</td>
<td></td>
<td></td>
</tr>
</tbody>
</table>

* p-value < .05  ** p-value < .001
Discussion

GDM and GHTN are serious disorders of pregnancy that can result in major health consequences for the mother and child. Risk factors and screening criteria have been established for both of these disorders (Akanji, 2003; Hedderson et al., 2011). The findings of this study both supported and refuted the literature.

Findings

Pre-pregnancy BMI and delivery BMI.

Pre-pregnancy and delivery BMIs were both found to be significant in the development of GHTN and GDM. Hedderson, Darbinian, Quesenberry, and Ferrara (2011) had similar findings in their study evaluating risk factors of GHTN. Obesity contributes to cellular insulin resistance and is listed as one of the high-risk criteria for GDM (Akanji, 2003). In addition, while blood pressure decreases in early pregnancy, it may become increased past baseline in the 2nd and 3rd trimesters (Jwa et al., 2011; Solomon & Seely, 2011). Our study demonstrated a significant association with increased pre-pregnancy BMI in developing both GHTN and GDM. In regards to GHTN, this finding replicates Fortner et al.’s study findings where they found that obese women (BMI > 29) had 2.5x odds of developing GHTN compared to controls (Fortner et al., 2009). In addition, Hedderson, Darbinian, Sridhar, and Quesenberry (2012) also found that obese women were found to have a higher incidence of GHTN. As for GDM, Hedderson et al. (2011) found increased pre-pregnancy BMI to be independently associated with GDM. While our study was unable to assess interventions for increased pre-pregnancy BMI, studies have demonstrated that exercise can decrease the risk of comorbidities during pregnancy (Zhang et al., 2006; Zhang & Ning, 2011). In consideration of different cultures, this may pose a problem as physician screening and nutritional advice may not be readily available as noted by Danso and
Opare-Addo (2010) in their review on challenges of diagnosis and treatment of pregnant women in low-income countries.

When controlling for age and race during the multiple logistic regression, the odds of the development of GDM and GHTN decreased. This suggests that race and advanced maternal age may add to the prevalence and impact of obesity on the development of these disorders. As stated previously, both of these disorders are multi-factorial (Flack et al., 2010; Fraser, 2009). Overall, these findings reiterate Xiong, Saunders, Wang, and Demianczuk’s (2001) findings that physicians should promote maternal weight loss before becoming pregnant in order to decrease their likelihood of developing GDM and GHTN.

**Age.**

Advanced maternal age has been established as a risk factor for both GHTN and GDM (Akanji, 2003; Fabry et al., 2010; Flack et al., 2010; Savona-Ventura et al., 2013). Depending on the diagnostic criteria used, the age cutoff may either be 25 or 30 years (Akanji, 2003). As the body ages, blood vessels thicken and develop plaques which results in decreased regulation of blood pressure. In addition, cells become less sensitive to insulin, which can develop into DM2. This study did not find a significant relationship between increasing maternal age and the primary outcomes during chi-square analysis or multiple logistic regression. While this refutes the current literature, it was notable that the mean age of our sample was only 27.32 years (Akanji, 2003; Fabry et al., 2010). Therefore, it is possible our sample was skewed toward younger women who were less likely to develop either outcome. With the developments in *In vitro* fertilization, the ability to freeze ova, and the increasing number of women pursuing higher education as well as careers before first pregnancy, the maternal age of first pregnancy is on the
rise (Carolan et al., 2012b). Doctors must be aware of this fact as they screen women for GDM and GHTN.

**Cigarette smoking.**

While cigarette smoking has not been consistently identified as a risk factor for GDM, we still felt that it was necessary to include this variable as a demographic factor since cigarette smoking is associated with many comorbid conditions (Zhang & Ning, 2011). During the chi-square analysis, we found that cigarette smoking appeared to be protective against GDM and GHTN. The clinical significance of this finding is uncertain as cigarette smoking causes vasoconstriction, which can lead to harmful consequences such as abruption of the placenta and decreased fetal growth (Magee et al., 2009). Of note, one cannot be diagnosed with GHTN if they already have CHTN, which is associated with cigarette smoking (Ponciano-Rodriguez, Paez-Martinez, Villa-Romero, Gutierrez-Grobe, & Mendez-Sanchez, 2014). This difference in development of GDM and GHTN became non-significant during the multiple logistic regression. While smoking is addictive, the physician must provide counseling and options for cessation. Nicotine replacement therapy and Varenicline (Chantix) are effective smoking cessation pharmacotherapies, though neither is approved during pregnancy. Consequently, there is insufficient evidence whether either medication causes significant developmental defects in fetuses and newborns (Coleman, Chamberlain, Davey, Cooper, & Leonardi-Bee, 2012; Kaplan, Olgaç Dundar, Kasap, & Karadas, 2014).

**WIC.**

The WIC program was included in our analysis as a measure of SES as one must be 185% below the national poverty line in order to qualify. While low SES is not an official risk factor for developing GHTN or GDM, it is associated with minority race groups, lesser
likelihood of seeking prenatal care, and poor nutrition (Akanji, 2003; Cardwell, 2013; Carolan et al., 2012b). There were no significant differences found between WIC usage and the development of GHTN or GDM in the chi-square analysis or forward stepwise multivariate logistic regression. As stated previously, in order to be diagnosed with either of these conditions, one must first be screened. If using WIC is associated with fewer prenatal visits, it is possible that neither condition was diagnosed in the majority of WIC-using participants in our sample. It is also possible there was a confounding variable that was not analyzed in our study. This is not meant to disparage the use of WIC; it is a very useful nutritional program that helps mothers in need provide for their current and future children.

**Payment type.**

Payment type was used in this analysis as a surrogate for one’s SES. Government (CHAMPUS or Tricare) insurance was added into the private insurance category as it allows similar increased access to healthcare. Those with lower SES are more likely to have Medicaid insurance or pay out-of-pocket. Our study found that there were significant differences between payment types in the development of GHTN and GDM in the chi-square analysis. Women with Medicaid were less likely to develop GDM in the chi-square analysis, though this difference became non-significant in the multivariate logistic regression. When compared against private insurance during the multivariate logistic regression, mothers with Medicaid and who paid out-of-pocket for their medical care were significantly less likely to develop GHTN. As stated above, women with lower SES are less likely to have access to medical care and treatments prior to and during their pregnancies (Cardwell, 2013). While global access to healthcare in second and third-world countries is an ongoing problem, newer screening criteria may be implemented for lower income countries, such as the 1 step 75-gram Oral Glucose Tolerance Test that will
allow for earlier diagnosis and treatment for GDM (Goldenberg, McClure, Macguire, Kamath, & Jobe, 2011; Leary et al., 2010; Reece et al., 2009). As for GHTN, domestically, methyldopa is available on the Walmart *Four Dollar* prescription list, making this medication very affordable (Wal-Mart Stores Incorporated, 2013). Though labetolol and nifedipine are not yet available on this list, physicians must work with their patients to find affordable and medically sound methods to screen and treat women GHTN in women with lower SES.

**STI’s.**

Contracting an STI during pregnancy was included in our analysis as another co-morbidity. We hypothesized that if one were to contract an STI during pregnancy, they would be at risk for other detrimental conditions such as GHTN and GDM. Since literature is lacking in this variable’s association with other diseases, we felt it necessary to include it in our analysis. This variable was found to be significant and protective in our chi-square analysis, though this finding became non-significant during the stepwise multiple logistic regression. It is unlikely that contracting an STI has any protective benefit against developing GDM or GHTN. It is possible that if these individuals were considered to be high risk, they were more likely to visit a hospital and be able to be counseled into making better life decisions. More research is needed in this area.

**Marital status.**

Marital status was analyzed as a binary variable of whether a woman was married during the time of pregnancy. Literature is also lacking in this area. Marital status can be double-edged; while it can be protective and supportive, there are also stresses that come with marriage. This variable was found to be statistically significant during chi-square analysis. However, married women had lesser odds of developing GHTN, but were more likely to develop GDM.
This variable continued to be significant during the stepwise logistic regression. As this was one of the few variables that remained significant during the logistic regression, more studies are need to determine if this was an incidental finding or truly clinical significant.

**Race.**

Race has been established as a risk factor for GDM as those of Asian and African race are significantly more likely to develop this disease (Akanji, 2003; Cheung et al., 2001). Cheung, Wasmer, and Al-Ali (2001) found that screening guidelines are often inadequate to identify at-risk Asian women because they may not have any risk factors other than their race. In addition, while race is not a definitive risk factor for GHTN, minority groups will likely have decreased access to health care making this group more at risk to develop detrimental health conditions (Bener & Saleh, 2013; Cardwell, 2013; Flack et al., 2010; Fortner et al., 2009). Our study found no differences in the development of GDM between racial groups in the chi-square analysis. African Americans were more likely to develop GHTN compared to other races in the chi-square analysis. It is surprising that our study found that Asians had lesser odds of developing GDM compared to Caucasians in the forward stepwise multivariate logistic regression. This refutes the findings in Carolan, Davey, Biro, and Kealy’s (2012) study on Australian women that found that women who were born in Asian countries and emigrated to Australia were at the highest risk of developing GDM. This discrepancy across studies demonstrates multiple definitions of “Asian”. In our study, we were unable to determine whether “Asian” women were born in the United States or abroad. While our findings cannot suggest screening based on race alone, the physician must consider this factor in his or her overall clinical impression to determine whether earlier or additional screening is needed for either GDM and GHTN.
**Education.**

Education was considered to be a measure of SES. We hypothesized that women with higher education would be less likely to develop GHTN or GDM as they would be more likely seek out prenatal and emergency care. Our study found that women with less than a high school education were less likely to develop GDM compared to women with college and graduate education. This finding is likely related to screening and age. One must attend prenatal appointments in order to be diagnosed with GDM. In addition, women with graduate education are likely to be older than women with less than a high school degree and may be more prone to develop diseases of pregnancy. Educational status was not significant in the forward stepwise multiple logistic regression suggesting a confounding factor for these findings. While the literature has evaluated age in developing GDM and GHTN, more research is needed to determine which aspects of SES may contribute to these diseases.

**Limitations**

Like any research project, our study had some limitations. First, the data points for GDM and GHTN were listed as “yes” or “no”. There was no method of determining to what extent each woman had GDM or GHTN. This would likely alter a given woman’s treatment plan if they had, for example, severe GHTN compared to mild GHTN. Second, this study’s results can be generalized to Ohio, but may not be as applicable to other states. Third, we were unable to analyze other identified risk factors for GDM or GHTN, such as Hispanic race or family history of either condition. Fourth, like every data set, our data is subject to coding errors and omissions. Fifth, we were unable to analyze pre-eclampsia incidence in our sample due to small sample size. Last, no evaluation could be performed on treatment method for either disease.
Conclusion

GDM and GHTN are serious disorders of pregnancy that continue to impact the well-beings of both mother and newborn. It is of the upmost importance that at-risk pregnant women be screened and treated in order to attain the best perinatal outcome. While there are established screening criteria for GDM, less so for GHTN, more evidence is needed to determine which risk factors are the strongest predictors and whether there are risk factors that are missing from the screening criteria. Our study re-emphasized the effects of increased BMI and refuted the literature on race in the development of GDM and GHTN. Our study also explored new avenues with un-established risk factors of marital status and STI’s, suggesting a possible risk of GDM with marital status. Overall, our study represents a contribution to the literature pertaining to pregnant women to help guide further research on GDM and GHTN.
References


Appendix 1 – IRB Approval

Office of Research and Sponsored Programs
201 University Hall
3640 Col. Glenn Hwy.
Dayton, OH 45435-0001
(937) 775-2425
(937) 775-3781 (FAX)
e-mail: rsp@wright.edu

DATE: April 14, 2014
TO: Jared Klein, PI, Graduate Student
    Public Health
    Sara Paton, Ph.D., Faculty Advisor
FROM: B. Laurel Elder, Chair
      WSU Institutional Review Board

SUBJECT: SC# 5507
  'An Analysis of the Demographic Factors Impacting the Development of Gestational Diabetes and Gestational Hypertension'

At the recommendation of the IRB Chair, your study referenced above has been determined to meet Federal exemption criteria 45 CFR 46.101(b). Please note that any change in the protocol must be reviewed by the IRB, as the project may no longer be exempt.

This action will be reported to the Full Board at their next scheduled meeting.

If you have any questions or require additional information, please call Jodi Blacklidge, Program Facilitator at 775-3974.

Thank you!

Enclosure
RESEARCH INVOLVING HUMAN SUBJECTS

SC# 5507

ACTION OF THE WRIGHT STATE UNIVERSITY
EXEMPT DETERMINATION
Assurance Number: FWA00002427

Title: 'An Analysis of the Demographic Factors Impacting the Development of Gestational Diabetes and Gestational Hypertension'

Principal Investigator: Jared Klein, Ph. Graduate Student
Public Health
Sara Paton, Ph.D., Faculty Advisor

The Institutional Review Board Chair has determined that your project is exempt from IRB oversight per 45 CFR 46.101(b4).

Signed: [Signature]
Chair, WSU-IRB

Approval Date: April 11, 2014
IRB Mtg. Date: May 19, 2014
### Appendix 2 – List of Competencies Met in CE

#### Tier 1 Core Public Health Competencies

<table>
<thead>
<tr>
<th>Domain #1: Analytic/Assessment</th>
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<tbody>
<tr>
<td>Identify the health status of populations and their related determinants of health and illness (e.g., factors contributing to health promotion and disease prevention, the quality, availability and use of health services)</td>
</tr>
<tr>
<td>Describe the characteristics of a population-based health problem (e.g., equity, social determinants, environment)</td>
</tr>
<tr>
<td>Use variables that measure public health conditions</td>
</tr>
<tr>
<td>Use methods and instruments for collecting valid and reliable quantitative and qualitative data</td>
</tr>
<tr>
<td>Identify sources of public health data and information</td>
</tr>
<tr>
<td>Recognize the integrity and comparability of data</td>
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<tr>
<td>Identify gaps in data sources</td>
</tr>
<tr>
<td>Adhere to ethical principles in the collection, maintenance, use, and dissemination of data and information</td>
</tr>
<tr>
<td>Describe the public health applications of quantitative and qualitative data</td>
</tr>
<tr>
<td>Use information technology to collect, store, and retrieve data</td>
</tr>
<tr>
<td>Describe how data are used to address scientific, political, ethical, and social public health issues</td>
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<thead>
<tr>
<th>Domain #2: Policy Development and Program Planning</th>
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<tbody>
<tr>
<td>Gather information relevant to specific public health policy issues</td>
</tr>
<tr>
<td>Describe how policy options can influence public health programs</td>
</tr>
<tr>
<td>Explain the expected outcomes of policy options (e.g., health, fiscal, administrative, legal, ethical, social, political)</td>
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<tr>
<td>Demonstrate the use of public health informatics practices and procedures (e.g., use of information systems infrastructure to improve health outcomes)</td>
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<tr>
<th>Domain #3: Communication</th>
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<tbody>
<tr>
<td>Participate in the development of demographic, statistical, programmatic and scientific presentations</td>
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<tr>
<th>Domain #4: Cultural Competency</th>
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<tbody>
<tr>
<td>Recognize the role of cultural, social, and behavioral factors in the accessibility, availability, acceptability and delivery of public health services</td>
</tr>
<tr>
<td>Describe the dynamic forces that contribute to cultural diversity</td>
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<tr>
<th>Domain #5: Community Dimensions of Practice</th>
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<tbody>
<tr>
<td>Recognize community linkages and relationships among multiple factors (or determinants) affecting health (e.g., The Socio-Ecological Model)</td>
</tr>
<tr>
<td>Identify stakeholders</td>
</tr>
<tr>
<td>Collaborate with community partners to promote the health of the population</td>
</tr>
<tr>
<td>Describe the role of governmental and non-governmental organizations in the delivery of community health services</td>
</tr>
<tr>
<td>Identify community assets and resources</td>
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<tr>
<th>Domain #6: Public Health Sciences</th>
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<tbody>
<tr>
<td>Describe the scientific foundation of the field of public health</td>
</tr>
<tr>
<td>Identify prominent events in the history of the public health profession</td>
</tr>
<tr>
<td>Relate public health science skills to the Core Public Health Functions and Ten Essential Services of Public Health</td>
</tr>
<tr>
<td>Identify the basic public health sciences (including, but not limited to biostatistics, epidemiology, environmental health sciences, health services administration, and social and behavioral health sciences)</td>
</tr>
<tr>
<td>Describe the scientific evidence related to a public health issue, concern, or, intervention</td>
</tr>
<tr>
<td>Retrieve scientific evidence from a variety of text and electronic sources</td>
</tr>
<tr>
<td>Discuss the limitations of research findings (e.g., limitations of data sources, importance of observations and interrelationships)</td>
</tr>
<tr>
<td>Describe the laws, regulations, policies and procedures for the ethical conduct of research (e.g., patient confidentiality, human subject processes)</td>
</tr>
<tr>
<td>Partner with other public health professionals in building the scientific base of public health</td>
</tr>
</tbody>
</table>
### Domain #7: Financial Planning and Management
- Describe the local, state, and federal public health and health care systems
- Adhere to the organization’s policies and procedures

### Domain #8: Leadership and Systems Thinking
- Incorporate ethical standards of practice as the basis of all interactions with organizations, communities, and individuals
- Describe how public health operates within a larger system
- Identify internal and external problems that may affect the delivery of Essential Public Health Services

### Concentration Competencies

#### Health Promotion and Education:

<table>
<thead>
<tr>
<th>Area 1: Assess needs, assets and capacity for health education</th>
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</thead>
<tbody>
<tr>
<td>Analyze factors that foster or hinder the learning process</td>
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<tr>
<td>Identify factors that foster or hinder skill building</td>
</tr>
<tr>
<td>Analyze factors that foster or hinder skill building</td>
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<tr>
<td>Synthesize assessment findings</td>
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<tr>
<th>Area 2: Plan health education programs</th>
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<tbody>
<tr>
<td>Use assessment results to inform the planning process</td>
</tr>
<tr>
<td>Formulate specific, measurable, attainable, realistic, and time-sensitive objectives</td>
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<tr>
<td>Organize health education into a logical sequence</td>
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<tr>
<th>Area 4: Conduct evaluation and research related to health education</th>
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<tbody>
<tr>
<td>Create purpose statement</td>
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<tr>
<td>Develop evaluation/research questions</td>
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<tr>
<td>Assess the merits and limitations of qualitative and quantitative data collection for research</td>
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<tr>
<td>Critique existing data collection instruments for research</td>
</tr>
<tr>
<td>Develop data analysis plan for research</td>
</tr>
<tr>
<td>Write new items to be used in data collection for research</td>
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<tr>
<td>Disseminate research findings through professional conference presentations</td>
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<tr>
<th>Area 5: Administer and manage health education</th>
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<tbody>
<tr>
<td>Use communication strategies to obtain program support</td>
</tr>
<tr>
<td>Synthesize data for purposes of reporting</td>
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<tr>
<th>Area 6: Serve as a health education resource person</th>
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<tbody>
<tr>
<td>Use a variety of resources and strategies</td>
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<tr>
<th>Area 7: Communicate and advocate for health and health education</th>
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</thead>
<tbody>
<tr>
<td>Evaluate advocacy efforts</td>
</tr>
<tr>
<td>Use evidence-based research to develop policies to promote health</td>
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