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Maternal Obesity and Incidence of Depression

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A Capstone Project Submitted in Partial Fulfillment of the Requirements of the Renée Crown University Honors Program at Syracuse University

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Abstract

Obesity is a national epidemic in the United States, which both directly and indirectly affects the social productivity of individuals, the American macro economy and individuals' personal health and well being. Depression often interferes with an individual's ability to work, sleep, study, eat, and enjoy life. A maternal state of both obesity and depression may cause serious adverse medical conditions in the mother's child. Taking steps to treat depression and obesity are critical in the construct of modern medicine. This project yields results that have the potential to make treatment options better tailored, more efficient, effective and economically-sound for the obese, pregnant population. This study was conducted using a collaborative approach, combining retrospectively collected patient data from Syracuse University and SUNY Upstate Medical Center. Our data shows that morbid obesity is positively correlated with higher incidence of depression. This suggests that health care practioners should screen morbidly obese perinatal women for depression more frequently and assist these women in accessing psychological treatment and weight management options.

Executive Summary

The purpose of this study is to screen women in the peripartum stage of pregnancy using the Edinburgh Postnatal Depression Scale. This information will be used in combination with retrospectively collected patient data to match de-identified patients' presence or abstence of depression with her body mass index. The retrospectively collected data will allow us to collect the body mass index (BMI). The goal of this research is to discern whether obesity (a BMI of >30) correlates with greater incidence of depression.

Body mass index can be calculated by dividing (weight in pounds x 703) by (height in inches squared) or by dividing (weight in kilograms) by (height in meters squared). The ratio of BMI is a fairly accurate indicator of an individual's fatty tissue mass and body mass index is used to assess obesity. Obesity is defined as a physiological state where an excess of adipose tissue results in a body mass index (BMI) of greater than 30.

The DSM-V defines perinatal or peripartum depression as major depression that occurs during pregnancy or in the period immediately subsequent to delivery [Lara et al., 2015]. Depression is typically characterized by reduced interest and pleasure in self, others and the environment (anhedonia) and a decrease in general appetite and sexual activity [Weinstock et al., 2001]. Clinicians characterize the peripartum stage as occuring during pregnancy and up to one year postpartum [O'Hara et al., 2013]. Identification of perinatal depression is imperative because it may have links to post partum depression (PPD), which can in turn impair the affected women's ability to care for herself and her baby [Lara et al., 2015]. Perinatal and post partum depression are associated with high usage of emergency rooms, malnutrition and developmental delay of the child and lower quality interactions between the mother and her child [Murray and Cooper, 1997; Field 2010].

The significance of this study cannot be understated. Depression often interferes with an individual's ability to work, sleep, study, eat, and enjoy life. From a social and economic perspective, individuals who are depressed will not function at their optimal level as members in the community. Obesity is a national epidemic in the United States, which both directly and indirectly affects the social productivity of individuals, the American macro economy and individuals' personal health and happiness. A depressed state compounded with obesity is not optimal from any perspective. Individuals who are obese and depressed are categorized as being at a severe risk for multiple diseases including diabetes, sleep apnea and chronic hypertension [Linne, 2004; Garbaciak et al., 1985; Cnattingius et al., 2002]. Furthermore, a maternal state of both obesity and depression may also lead to proliferation of serious health conditions in the mother's child [Weinstock et al., 2011]. Taking steps to treat depression and obesity are critical in the management of patients. This project will make treatment options better tailored, more efficient, effective and economically-sound for the obese, pregnant population.

The clinical relevance of this study is based on the hypothesis that if there is an increased incidence of depression in obese obstetric patients, these patients can be monitored and treated for depressive symptoms prior to the postpartum period. This intervention may help to accelerate both the mother's recovery from depression and may aid in fostering the health of the newborn. If our hypothesis that a greater BMI (Body Mass Index) correlates with a higher incidence of depression is correct, this can impact obstetrical care and aid in the treatment of patients. If significant findings are found, it would indicate that obese maternal patients should receive mental health screenings throughout pregnancy and psychological treatment should be made readily available to this population. Even if our hypothesis is incorrect, we will still have discovered an important finding; that a greater BMI does not correlate with depression in

obstetric patients. This can help improve the efficiency and effectiveness of obstetrical care, by avoiding the added expense of mental health screening and treatment for this group of women.

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Preface

I wish to be a Physician Assistant in the near future and as a medical practitioner, I want to be able to provide the highest possible quality care to my patients. Studies like this one provide health care providers valuable information about how they can tailor their treatment to specific patients. I also wish to contribute to medical research prior to becoming a practitioner. The joined, collaborative research efforts at Syracuse University and SUNY Upstate Medical Center have certainly increased the quality of this project and this project is a statement about the importance of collaborative medical science research.

Acknowledgements

I wish to express my deepest gratitude to my advisor, Dr. Robert Silverman and Christine Rydelek at SUNY Upstate Medical Center for their time, kindness, expertise and professional guidance and for helping me see this project through to completion. My greatest thanks to Karen Davis who helped make the collection of the patient information possible and for helping to guide and clarify matters throughout the extent of the project. Thanks to Martha Wojtowycz for contributing her superior biostatistical analysis expertise and supervision. Thanks to Meghan Hall and Dr. Sandra Lane for making this collaborative research possible. Also, my deepest gratitude to the Renee Crown Honors staff at Syracuse University and to my Biotechnology, medical and biological science professors at Syracuse who have helped mentor, teach, guide and encourage me throughout my career at Syracuse, especially Surahabi and Ramesh Raina. Thanks to the Syracuse Athletic Department and Syracuse Women's Rowing Team for sponsoring my studies. A special thanks to my family and friends for their continued unconditional support and encouragement. A special thanks to all those not named but who have helped me during my time as a Renee Crown Honors student at Syracuse University. I so cherish the time I have spent here; the experience has been priceless.

Advice to Future Honors Students

I would advise future Honors Students to brainstorm about Capstone topics early on in their collegiate career and to choose their ultimate Capstone topic based purely on passion and interest in the project material. No matter what hurdles you encounter in your work, you will be willing to work through them so long as you are passionate about your project! Don't be afraid to ask for help! Its suprising how many people will be interested in supporting you with your work! Good luck!

Chapter 1

Introduction

Syracuse University's College of Arts and Sciences notes on their website that, "Biotechnology is a rapidly growing, interdisciplinary field that seeks to solve today's most challenging problems in such areas as human and animal health, agriculture, and the environment " [Syracuse University College of Arts and Sciences, Biotechnology webpage 2010]. My capstone project seeks to understand one of America's most pressing health care issues using biotechnological tools. Specifically, this project will determine whether there is an increased prevalence of depression in increasingly obese pregnant patients versus normal weight pregnant patients. I will use an interdisciplinary approach and will draw from retrospectively collected clinically measured physiological data and from previous data collected by highly trained leaders in the fields of depression and obstetrics (The Department of Obstetrics and Gynecology at Upstate Medical University: Correlates and Consequences of Prenatal Depression-an exploratory study). I will review primary data about various levels of obesity and its correlation to incidence of depression. Our goal is to draw a conclusion about the effects of obesity, an extraordinarily pressing American public health issue, and correlate it to the incidence of depression.

The broad spectrum, interdisciplinary approach that is characteristic of biotechnology, will be particularly useful when evaluating the problem of obesity as it relates to pregnant women especially in the face of the medical and social consequences that demand varied evaluative methods. Depression often interferes with an individual's ability to work, sleep,

study, eat, and enjoy life. From a social and economic perspective, individuals who are depressed will not function at their optimal level as members in the community. Obesity is a national epidemic in the United States, which both directly and indirectly affects the social productivity of individuals, the American macro economy and individuals' personal health and happiness. A depressed state compounded with obesity is not ideal from any perspective. Individuals who are obese and depressed are categorized as being at a severe risk for multiple diseases including diabetes, sleep apnea and chronic hypertension [Linne, 2004; Garbaciak et al., 1985; Cnattingius et al., 2002]. Furthermore, a maternal state of both obesity and depression may also lead to proliferation of serious health conditions in the mother's child [Weinstock et al., 2011]. Taking steps to treat depression and obesity are critical in modern medical care. This project will make treatment options better tailored, more efficient, effect and economically-sound for the obese, pregnant population.

The clinical relevance of this study is based on the hypothesis that if there is an increased incidence of depression in obese obstetric patients, these patients can be monitored and treated for depressive symptoms prior to the postpartum period. This intervention may help to accelerate both the mother's recovery from depression and may aid in fostering the health of the newborn. If our hypothesis that a greater BMI (Body Mass Index) correlates with a higher incidence of depression is correct, this can impact obstetrical care and aid in the treatment of patients. If significant correlation is found, it would indicate that obese maternal patients should receive mental health screenings throughout pregnancy and psychological treatment should be made readily available to this population. Even if our hypothesis is incorrect, we will still have discerned a significant finding that a greater BMI does not correlate with depression in obstetric

patients. This can help improve the efficiency and effectiveness of obstetrical care, by avoiding the added expense of mental health screening and treatment for this group of women.

The New York Statewide Perinatal Data System's 2013 database, was used to correlate BMI with perinatal depression (See Appendix A for the New York State Birth Certificate and Statewide Perinatal Data System Work Booklet). Using the parameters, they found that for obese and normal weight women, 64.0% and 71.4% are not depressed at all, 23.1% and 18.4% are a little depressed, 7.1% and 4.7% are moderately depressed, 0.9% and 0.7% are very depressed and 1.3% and 0.6% are very depressed, respectively, and needed psychological help [New York Statewide Perinatal Data System, 2013]. This information was collected from 18,000 deidentified Central New York expectant mothers in 2013 and clearly shows that perinatal obesity has a strong correlation with depression during pregnancy. The depression scale used to screen the mothers and develop these statistics was a validated assessment (PRAMS, the Pregnancy Risk Assessment Monitoring System), developed by the CDC, Center for Disease Control. This statistical analysis powers our study with high clinical relevance. The goal of this study will be to find whether *increasing levels* of obesity correlate with increasing incidence of depression during the perinatal period.

Chapter 2

Literature Search

Obesity is a condition where the adipose tissue mass of an individual is beyond the physical requirement for such tissue [Singh, 2014; Linne 2004]. Obesity is the result of a surplus of energy consumed (energy intake) as compared to energy expended [Singh, 2014]. The accumulation of triglycerol, rather than accumulation of carbohydrate glycogen or protein in the liver or muscle, is the only way the body can become "excessive" [Sikaris, 2004]. As a tissue, adipose tissue is an organ system that can vary enormously between individuals and perhaps more so than other tissues in the body [Linne, 2004]. The variations in adipose morphology and location in the body can have tremendous implications for health and disease. Adipose tissue acts as an endocrine organ in the body, as it can release adipokines that can affect and regulate the functioning of other tissues in the body. These functions are amplified in the case of obesity [Kershaw et al, 2004]. It is important to note that women in general, have a greater proportion of subcutaneous fat and lower bone and muscle mass compared to men. Also notable, is that bone density, bone mass and muscle mass all decrease with age in both sexes. The World Health Organization has redefined body morphologies as a BMI >25 as overweight and >30 as obese [World Health Organization, 1997].

There are a variety of metabolic genes and gene products that are generally agreed upon as associated with causation and proliferation of obesity [Snyder et al., 2003]. Leptin is a hormone that communicates with the central nervous system and binds in the hypothalamic

arcuate nucleus thereby stimulating production of pro-opiomelanocortin (POMC). Two active products of POMC are alpha-melanocyte stimulating hormome (alpha-MSH) and adrenocortioctropin (ACTH). Alpha MSH binds to its respective receptors in the paraventricular nucleus to signal satiety and decrease food intake. [Sikaris, 2004]. Mutations with the leptin signaling pathway are rare but are thought to be linked to obesity [Proietto et al, 2004]. Beta- 3 adrenergic receptor (ADRB3) is expressed in adipose tissue and in normal cases, is involved with lipid metabolism and thermogenesis. A mutation of ADRB3 is associated with obesity [Widen et al., 1995; Clement et al., 1995; Kurokawa et al., 2001]. Peroxisome proliferator activated peptide receptor gamma (PPAR-gamma) is a nuclear receptor that is essential for adipogenesis and regular insulin signaling [Sikaris, 2004]. The Pro12A1a mutation can decrease the binding affinity of PPAR-gamma. However, it has been found that Pro12A1a mutations seem to have a compounded, more severe effect in individuals already afflicted with a genetic proclivity to obesity, and the Pro12A1a mutation has less of an effect on individuals predisposed to have a normal body weight [Ek et al., 1999]. Adiponectin is typically associated with maintenance of energy homeostasis, macromolecule metabolism and anti-inflammation. High levels of adiponectin are associated with weight loss [Stumvoll et al, 2002]. A deficit of adiponectin may be linked to obesity and polymorphisms of adiponectin are associated with obesity, increased weight gain and insulin sensitivity [Stumvoll et al., 2002].

There are many physiological costs to obesity. In this study, the physiological costs of obesity that have the greatest consequences for pregnancy and maternity will be analyzed. Obstructive sleep apnea is characterized by absence of airflow in spite of thoraco-abdominal muscular movement [Sikaris, 2004]. Seventy percent of patients with obstructive sleep apnea are obese, likely due to fatty deposits in the neck and pharyngeal area [Malhotrra et al., 2002;

Fleetham 1992]. This is very problematic, especially in consideration of fetal development since there is a lowering in physiological oxygen saturation in maternal blood during resting hours [Series et al., 1993]. This may lead to decreased oxygen delivery to the developing fetus. Obstructive sleep apnea is also associated with hypertension and coronary artery disease (CAD) [Sikaris, 2004]. The increased incidence of hypertension that is linked to obesity, is also associated with increased sympathetic nervous activity. This increased activity in turn can cause hyperinsulinaemia, increased intrarenal pressure and increased levels of hepatic free fatty acids, angiotensin II, leptin, central chemoreceptor sensitivity. In addition, it can reduce baroreceptor reflex and the ability to respond to changes in pressure in blood vessels [Sikaris, 2004; Grassi et al., 1998]. These conditions also have the potential of effecting oxygenation and nutrient availability to the growing fetus in a negative manner. There is also a greatly increased risk of insulin resistance when individuals are obese. Upwards of 90% of diabetic patients are overweight or obese [Albu et al., 1998]. This may be due to the fact that preceding the onset of diabetes, weight gain and insulin resistance usually occur [Sikaris, 2004]. Increased levels of free fatty acids in the circulation of individuals with increased adipose mass can produce insulin resistance in the skeletal muscle and liver and can reduce beta cell functioning and resultant release of insulin in the pancreas. This reduction in physiological functioning due to excessive fatty acids is called lipotoxicity [Schaffer, 2003]. Poor regulation of insulin and glucose may also negatively affect the developing fetus. Also notable, growth hormone (GH) levels and vitamin D levels are lower in obese populations. Lower vitamin D levels results in higher levels of parathyroid hormone (PTH) which in turn affects calcium metabolism [Sikaris, 2004]. Sikaris suggests that social isolation and indoor dwelling may somehow be connected to these vitamin deficiencies. Additionally, it should not be overlooked that obesity is associated with a

significant community stigma. Public disapproval and rejection can affect education, employment, income, marital status and health care [Sikaris, 2004]. These are important clinical factors that are believed to be associated with adversely impacting the person's quality of life and increasing the incidence of depression [Sikaris, 2004].

Obesity is a world-wide epidemic. In the United States, it has been reported that approximately 44% of women aged 18-49 were obese, 40% of married women in the United Arab Emirates are obese and in Sweden, the prevalence of overweight BMIs among the female population aged 16-44 doubled in recent years. Obesity is a threat to the health of childbearing-aged women and their offspring [Linne, 2004].

During pregnancy it is normal for the female body to create a buffer of approximately 30,000 kcal to ensure that there is a sufficient energy resource for the last trimester when fetal metabolic demands are high, for anticipated extended lactation and to shield the pregnancy from the possibility that there will be food scarcity [Linne, 2004]. However, the weight gain recommendations from the Insitute of Medicine (IOM) vary depending on pre-pregnancy BMI. It is recommended that obese women, or those women with a BMI >29, gain approximately 6.8kg, which metabolically is significantly less than 30,000 kCal buffer most women gain during pregnancy [Linne, 2004].

Obesity during pregnancy places a pregnancy into the high risk for complications category. Obese women as compared to normal weight women have a higher rate of hypertensive diagnoses, gestational diabetes, delivery complications, caesarean sections, prolonged labor and delivering children with poor health [Linne, 2004; Garbaciak et al., 1985; Cnattingius et al., 2002]. Obese women of child-bearing age have a decreased choice of contraceptive methods and an increased risk of hyperandrogonadism, amenorrhoea,

dysovulation, infertility, hyperinsulinaemia, polycystic ovary syndrome, depression, edometrial cancer and breast cancer [Linne, 2004]. During a pregnancy, obese women have an increased risk of developing gestational diabetes, hypertension, dyslipidaemia, impaired endothelial function leading to preclampsia, back pain, preterm delivery, induced labor, prolonged labor, vulvar or perineal lacerations, cesarean section and related complications including infection and thromboembolism [Linne, 2004]. It is generally accepted that the average weight women has a 1-3% chance of developing gestational diabetes. Obese women have an approximately 17% chance of developing gestational diabetes [Linne, 2004]. Obese pregnant women have an increased risk of babies to develop spina bifida, macrosomia, head trauma, shoulder dystocia, brachial plexus injury, fracture of the clavicle, increased perinatal mortality and hemorrhage after delivery [Linne, 2004]. For these reasons, it is clear why obesity places women in a "high risk" category during pregnancy.

Pregnancy is also characterized by increased psychological vulnerability. This vulnerability can be amplified or minimized by certain factors such as age, health, socio-economic status and obesity [Della Vedova et al, 2011]. A high risk pregnancy is a pregnancy in which a medical factor pertaining to either the mother or the fetus, may adversely affect the ultimate outcome of the pregnancy [Levy-Shiff et al., 2002]. When a pregnancy is categorized as high risk, the psychological stress of the mother regarding the pregnancy is affected in a variety of ways [Levy-Shiff et al., 2002]. Maternal reactions may include depression (as analyzed by the present study), regressive behavior, injured self-esteem, reduced confidence and self-efficacy, anger directed at low-risk pregnancies, and/or self-recrimination [Levy-Shiff et al., 2002]. Increased doubt and uncertainty compound the physiological threats of the pregnancy. Psychological threats can interfere with the normal adjustment to pregnancy and may actually

work to emotionally and psychologically separate the mother from her unborn child [Levy-Shiff et al., 2002]. For practitioners who care for women with high risk pregnancies, it is useful to monitor the stress-related responses of the expectant mothers. Maternal psychological state has the capacity to affect the immediate situation in the pregnancy and the long term outcome of the mother's ability to care for her child.

Previous psychological research has been performed on high risk pregnancies, including those where mothers had pre-gestational diabetes or gestational diabetes. Cognitive appraisal of pregnancy as a challenge and threat, cognitive appraisal of control, ways of coping, social support, depression (via the Beck Depression Inventory), state-trait anxiety, well-being and distress (via Pines and Aronson 1981 Burnout Questionnaire), and other psychological symptoms have been measured [Levy et al., 2002]. These factors are all relevant and will be considered in this study. Specifically the mother's primary and secondary cognitive appraisals, coping strategies and availability of coping resources are extremely important to monitor [Levy-Shiff et al., 2002]. Cognitive appraisal is how the mother assesses her social and physical situations and her pregnancy. Pregnancy may be appraised as a threat, a challenge or a miracle. Coping strategies are considered the cognitive and behavioral efforts in managing the stressors related to pregnancy. Lastly, coping resources refer to the social support system available to the mother during pregnancy [Levy-Shiff et al., 2002].

Lack of support throughout pregnancy can increase depression and anxiety symptoms. Partner and/or spousal support and family support factor significantly in the presence or absence of depressive and anxiety-related symptoms. Familial support has been shown to be protective against mood disorders. Mothers given a high level of social support during stress-inducing stages of their pregnancy had lower levels of circulating stress hormones than did mothers

without support [Wadhwa et al., 1996]. This association suggests that there is an relationship between the hypothalamic-pituitary-adrenal axis activation, hormone and neurotransmitter emission and perceived stress. Sufficient social support also resulted in reduced negative -affect emotions and reduced the likelihood of preterm labor and depression [Oakley et al., 1990]. Likewise, this suggests that there is a potential connection between self perceived or assessed stress and incidence of preterm birth. The absence of familial support is a risk factor for mood disorders [Alfaraj et al., 2009; Hammarberg et al., 2013; Pajulo et al., 2001; Rubertsson et al., 2003; Whisman et al., 2011]. It is also important that the patient's partner other also have a well managed psychological state. If an adverse psychological state is discovered in the significant other, it is important to have a clear understanding of the basis of the problem. It is also important to consider that the clinician having a relationship with the significant other may aid in identification of anxiety or depression in their partner (the mother) [deMontigny et al., 2013; Forsyth et al., 2011]. These factors may be important for future studies, but this study will focus on depressive symptoms of the mother.

There are a multitude of factors that relate to a mother's ability to cope with stress and depression. Low educational level reduces the ability to cope with stress [Kubzansky et al., 1999]. Poverty is also an underlying factor for pregnancy stressors as it seems to correlate with lack of access to quality health services, infant care and information [Lara et al., 2015]. In Mexico and other resource-restricted countries, there is a greater probability that perinatal depression will go undetected and untreated [Lara et. al., 2005]. This is probably due to total underutilization of mental health care services [Halbreich and Karkun, 2006; Borges et al, 2006; Matijasevich et al, 2009; Fisher et al., 2012]. Awareness of the social stigma correlated with depression in combination with reduced awareness in under-resourced areas may also prevent

women from seeking treatment [Lara et al., 2014]. One way to help close this prenatal healthcare gap is to increase awareness of pre and post natal depression. Educating health care providers and expectant mothers and providing options to train health care providers to screen successfully for depression may help close this gap. It is also important to assess more vulnerable mothers, such as those who are less educated, single, unemployed or impoverished to provide support networks [Lara et al, 2015].

Another important variable, relevant to our patient sample in Syracuse, is that residing in an urban environment is known to be more stressful than a rural community, perhaps due to the urban prevalence of noise, pollution, crime and overcrowding [Weinstock et al., 2001]. This conclusion has been drawn because schizophrenia and depressive symptoms in individuals have been more highly correlated with cities and areas with high population density [Marcelis et al., 1998]. The concept of 'urban stress' simply exemplifies how even minor, seemingly insignificant stressors may actually have a significant impact on depression and pregnancy. Stressors may include everything from everyday life events, to daily hassles, to pre-existing medical conditions. Divorce, serious illness or death of a loved one, domestic affairs, financial problems, young age, poor preparation for pregnancy or delivery, low socioeconomic status, sexual abuse, unwanted pregnancy, lack of partner, psychiatric disorders and depressive symptoms are all classified as stressors that have the potential to affect pregnancy. Major psychological and physiological disturbances associated with depression may have a significant impact on the pregnancy as well [Teixeira et al., 1999].

The DSM-V defines perinatal or peripartum depression as major depression that occurs during pregnancy or in the period immediately subsequent to delivery [Lara et al., 2015]. Depression is typically characterized by reduced interest and pleasure in self, others and the

environment (anhedonia) and a decrease in general appetite and sexual activity [Weinstock et al., 2001]. Clinicians often characterize the peripartum stage as ranging up to one year postpartum [O'Hara et al., 2013]. Identification of perinatal depression is imperative because it may have links to post partum depression (PPD), which may impair the affected women's ability to care for herself and her baby [Lara et al., 2015]. Perinatal and post partum depression are associated with high usage of emergency rooms, malnutrition, developmental delay of the child and lower quality interactions between the mother and her child [Murray and Cooper, 1997; Field 2010]. Perinatal depression has significant clinical value because it has detrimental effects on women's health as well as pregnancy outcomes [Marcus et al., 2003]. Self reporting scales are useful as screening tools for further diagnostic assessment and for quick and accurate clinical measurements of depression [Lara et al., 2015].

Between ten and twenty percent of women experience perinatal depression [Lee et al., 2007]. Kwan et al. have found that the Edinburgh Postnatal Depression Scale (EPDS) can be used as a screening tool for depression and can additionally be used to measure dysphoria (Appendix B). It can screen for a range of symptoms including depression, anxiety and anhedonic symptioms in many different settings. The Edinburgh scale can be administered by any level health care professional, trained in interviewing patients (Kwan et al., 2015). Recent studies exploring high risk pregnancies showed that EPDS was able to successfully identify depression and anxiety [Thiagayson et al., 2012]. The present study will use the Edinburgh Postnatal Depression scale to analyze the incidence of depression in obese mothers during the perinatal period.

Depression has found to be associated with premature births, miscarriages, longer hospital stays within the perinatal period, lower infant Apgar scores and smaller head

circumferences [Campagne, 2004; Orr et al., 2007; Lancaster et al., 2010; Chen et al., 2004; Stanton et al., 2002]. Pregnant women who are depressed are at an increased risk for delivering prematurely. Premature infants are at a greater risk for low birth weights and having smaller size relative to their respective gestational age [Field et al., 2006]. The occurrence of preterm labor is also strongly associated with the stress-induced release of hypothalamic, pituitary and placental hormones [McClean et al, 1995; Wadhwa et al., 1998]. Therefore, early detection of possible depression is critical in preventing potential future complications in the mother and fetus. Clinician observation of the mother's cognitive appraisal and coping strategies and access to resources is imperative for this reason [Arnal-Remon et al., 2014]. Because stress in pregnancy can influence the development of birth complications, it is important that early identification of mood disorders takes place to ensure successful, positive-outcome pregnancies [Arnal-Remon et al., 2014]. The American Psychological Association considers treatment for depression that is occurring during pregnancy to be a priority so that pospartum depression can be avoided [Spinelli et al., 2003; Heron et al., 2004].

Neurotransmitters are essentially the modulators of depression. Suboptimal regulation of the HPA axis is generally linked to anxiety disorders and depression, where higher concentrations of cortisol can be found in both urinary and salivary samples [Arborelius et al., 1999; Nemeroff et al., 1984]. Not surprisingly, depressed subjects show higher plasma and urinary levels of ACTH and catecholamines in response to a stressful situation [Gold et al., 1988; Holsboer et al., 1994, Heim et al., 2000]. Also, through blood sampling experimentation, beta-endorphin concentrations (especially in chronic rather than episodic cases) have a positive correlation with stress and anxiety. These hormones activate the HPA axis during pregnancy [Demyttenaiere et al., 1989; Wadhwa et al., 1996]. This physiological linkage between the

body's stress systems and female reproductive tract has significant consequences for a pregnancy.

Normal growth and development of a fetus can be significantly altered by a magnitude of factors including complications of pregnancy, maternal or fetal infections and teratogens. Teratogenic agents can disturb the normal development of the fetus [Mulder et al., 2002]. Hormones and neurotransmitters released in excess by the body during depression or times of extreme stress are considered potential teratogens.

Stressors activate the hypothalamus-pituitary-adrenal cortex system (HPA axis) and the sympathetic nervous system, or the adrenal medulla system. Activation of these stress-systems releases excess corticotropin-releasing hormone (CRH), adrenocorticotropin-releasing hormone (ACTH), cortisol and (nor)adrenaline in the blood [Mulder et al., 2002]. These hormones deriving from the HPA axis in particular have a strong inhibiting effect on HPG and CRH. Cortisol receptors are numerous in the endometrium, ovaries and myometrium [Mulder et al., 2002].

Corticosteroids and catecholamines are known to strongly affect peripheral blood flow. Doppler blood flow studies using ultrasound to measure blood flow in vessels have shown that there is increased resistance of the uterine artery in women that experience high levels of anxiety thus altering the blood flow to the uterus [Teixeira et al., 1999]. Gestational stress can also cause an increase in CRH from the placenta, specifically catecholamines and cortisol and cause fetal hypoxia. This is because hormonal shifts cause fluctuations in the mother's blood vessel tone and heart contractions. This demonstrates that gestational stress can affect fetal development [Petraglia et al, 1996; Sug-Tang et al., 1992].

Maternal cortisol levels are linked to concentrations of fetal cortisol [Gitau et al., 1999]. Cortisol levels are transmitted through the placenta, thereby affecting the fetus [Chrousos et al., 1998; Weinstock et al., 2011]. Activation of the fetal hypothalamus-pituitary-adrenal (HPA) axis in response to the mother's psychological stress could have a significant impact on the offspring's behavioral pathology later in life [Weinstock et al., 2011]. Specifically, maternal stress hormones may interfere with fetal levels of testosterone and aromatase at critical periods which may alter some masculine behaviors or morphologies later in life and cause cell loss in the fetal brain by disregulating the fetal HPA axis and altering normal development of the limbic system [Weinstock et al., 2011].

It is generally assumed that maternal stress may impact the fetus through communication of stress hormones via the female reproductive system. Possible transmission of maternal stress can include stress-induced release of CRH from the placenta to the intrauterine environment where the fetus is developing, transplacental transport of maternal hormones, and reduction of blood flow to the uterus and fetus [Mulder et al., 2002].

The concept of 'fetal programming' relates to the concept that the fetal exposure to nutrients, proteins, hormones, temperature and infection will play a critical role in the development of the fetus. Stress hormones have the capacity to participate in fetal programming (See Appendix C). Programming in-vitro, is related to the incidence of cardiovascular and allergic diseases, hypertension, diabetes and schizophrenia [Mulder et al., 2002]. It is also important to note that when the mother experienced significant stress in early as opposed to mid-gestation, the offspring had lower birth weights and more significant motor impairments [Schneider et al., 1999]. Damage inflicted on the developing fetus will depend on the timing of the given maternal (physiological or psychological) stressor relative to the development of the

fetus [Weinstock et al., 2011]. Studies have shown that gestational stress at 5 to 7 weeks gestation may affect the development of the superoptic and paraventricular neucli and hypthalamus and regulation of HPA axis [Weinstock et al., 2011]. Therefore it is very important for clinicians and patients alike to be aware of physiological and psychological stress of pregnancy, due to the potential to induce a fetal complication.

Proving that perinatal maternal stress and depression is linked to an offspring's neurological and behavioral pathology is difficult because of the possibility that there is a genetic component in the mediation of these neuropathies and behavioral traits. It is also important to note that the maternal stress may persist after birth and the infant's development and behavior may be influenced by this latter behavioral influence of the mother [Weinstock et al, 2001]. It has been noted that, some individuals adopt passive behaviors when faced with unpleasant events. This lack of control over aversive environmental events leads to the expectancy that future stimuli will be uncontrollable. This in turn leads to a phenomenon known as 'learned helplessness'. Prenatal stress can, in some cases, exacerbate learned helplessness in the mother. Learned helplessness can clearly lead to poor child development and may strain early familial relationships between the mother and her child. [Seligmean, 1972; Secoli et al., 1998]

However, regardless of the precise way in which maternal stress affects the newborn, it is important to minimize levels of stress. Through genetics, placental development or parenting, the mother's psyche will affect her newborn. Additionally, the high risk associated with being obese while pregnant has the capacity to establish hazardous health conditions for the mother and her developing infant. This area of medicine involving depression in obese maternal patients is critically important to understand.

Chapter 3

Methods

Design

Taking advantage of the collaborative research ties between Syracuse University and Upstate Medical University, this project integrates research from principle investigator, Dr. Renee Mestad's living study, "Correlates and Consequences of Prenatal Depression: An exploratory Study" with independently collected data for this study, "Maternal Obesity and Incidence of Depression". The current study has been exempt from IRB approval through association with Dr. Mestad's study (IRB exemption #389664-1). The current research team and Dr. Mestad's research team both stand to benefit from the information collected, and society at large will benefit as well. The research practice of collaborative efforts and data collection is perhaps one of the most efficient methods of research. It is cost effective and can lead to more accurate and clinically applicable research outcomes. Combined, the breadth and depth of the information about prenatal and perinatal depression will provide compelling evidence for practioners to alter their patient treatment methods. The statistical significance of our study is reinforced by the New York Statewide Perinatal Data System from 2013. This project will provide information that can have an immediate applied benefit in medicine. It is an example of "incremental research". A stepping stone that will advance researchers and medical practitioners alike with progression toward precise, personalized medicine. The collaborative approach to

clarification and discovery of principles about human physiology and medical science is valuable in clinical medicine and therefore it is also pertinent and applicable to the field of Biotechnology.

The following statements reflect the goals of Dr. Mestad's research team's studies and are from her Internal Review Board (IRB) exemption statement:

> "Dr. Mestad's study aims to investigate the correlates and consequences of prenatal depression in the patient population served by Upstate University's UHCC Women's Health Clinic. Principal Investigators, Dr. Carrie Smith, Dr. Bruce Carter, and Dr. Sandra Lane, whom are all faculty professors within the David B. Falk College of Sport and Human Dynamics at Syracuse University, are recipients of the college's SEED (Supporting Effective Educator Development) Grant amounting in $5000.00. These funds will act as the primary sponsor of this study. Dr. Renee Mestad, within the department of Obsterics and Gynecology at Upstate Medical University is also acting as a faculty Principal - 2 - Generated on IRBNet Investigator overseeing the chart review process at UHCC Clinic and Crouse Hospital… Per the 45 Code of Federal Regualtions, Part 46 101.B, our intended study falls under the exemption category of research because it involves the collection and study of existing data, documents, and records. The study Principal Investigator, Renee Mestad, is an attending physician in the department of Obstetrics and Gynecology and has available access to patient antenatal and delivery charts at Upstate University Hospital and Crouse Hospital. The study design is a retrospective cohort design that will include the review of 350 antenatal and delivery charts within the following timeframe: 01/01/2010-12/31/2010… all data will be collected in a de-identified manner.

The collected data will not be linked directly nor indirectly to a specific patient record. Upstate Medical University Women's Health Services conducts prenatal screening of all pregnant patients three times during the pregnancy. This proposed study will involve review of the prenatal and hospital delivery charts of 350 patients. The study is a retrospective cohort design that involves chart reviews of the antenatal and delivery charts of 350 pregnant Syracuse residents who delivered during the timeframe of 01/01/2010-12/31/2010. All data abstracted will be collected in a de-identified manner within the chart review room at Upstate Medical University Hospital and Crouse Hospital. All chart reviews are listed on the de-identified form that is attached to this application. A quality control method that has been previously established in studies conducted by the CDC will be used during the chart review process. All chart reviewers are either staff or faculty of Upstate Medical University or students and volunteers associated with Syracuse University and have been granted the appropriate Upstate Medical access to review these charts.

All chart reviewers are individuals familiar with Upstate University Hospital's privacy policies as well as federal HIPPA guidelines. All have completed the appropriate CITI training as required by Upstate Medical University and Syracuse University. The variables that will be extracted for this study include the Edinburgh depression scores, maternal social and economic demographics, antecedent health and social conditions that might influence mood and emotional status, pregnancy related problems, and conditions that might affect stress and coping, and birth outcomes. The data abstraction form is attached to this application. This exemption application is also being simultaneously submitted to the Syracuse University and Crouse Hospital for IRB exemption.

Although the American College of Obstetricians and Gynecologists "strongly encourages" prenatal screening for depression, it is not routinely part of prenatal care in most clinical settings. UHCC/ WHS at Upstate Medical University is unusual in conducting this routine screening. UHCC/WHS also serves a very diverse group of low socioeconomic status patients. This chart review would allow us to collect quantitative data on a diverse group of pregnant women who were screened three times during the pregnancy as part of routine prenatal care. Prenatal screening of all pregnant patients occurs three times during the pregnancy with the Edinburgh assessment tool. Women who screen "positive" are interviewed by the on-site social worker to determine the extent and type of their emotional and psychological distress. The social worker then refers women who need additional therapeutic services. We are interested in answering the following questions:

1.) What proportion of women screen positive for depression at once, twice, or three times during pregnancy?

2.) What proportion of those who screen positive are referred for further care by the social worker?

3.) What appeared to be the barriers to referral for additional care? Were any barriers associated with limitations of available services? Were any barriers due to refusal of Medicaid or other insurance denial of approval for the referral? Were any barriers due to the patient refusal to seek services?

4.) What clinical and/or social conditions of the woman were associated with positive screens for depression?

5.) What birth outcomes were associated with positive screens for depression?

6.) What proportion of women who were previously taking prescribed antidepressant medication prior to the pregnancy stopped the medication during the pregnancy? What proportion of women who stopped antidepressant medication during pregnancy screen positive for depression?

The submitted data form will be used for data collection. A single master list (key) linking the subject's unique study number to medical record number will be secured in a locked location within Upstate, and always kept separate from the data forms. They will not contain any other information and will be destroyed immediately after medical record abstraction."

Dr. Mestad's original research, combined with the present study's data can be viewed in Appendix D. The unique nature of the current study is that obesity will be studied in a graded manner. BMIs of patients will be categorized in to ranges of >18.5 for underweight, 18.5-24.49 for normal weight, 24.5-29.9 for overweight, 30-39.9 for obese and >40 for morbidly obese. The incidence of depression among these groups of patients will be quantified and analyzed for statistically significant differences. The current research team will then analyze how BMI correlates with the incidence of depression. We hypothesize, based on the Central New York Database, that an increase in BMI will correlate with an increase in incidence of depression.

To join the research objectives of the present group and Dr. Mestad's research group, one of Dr. Mestad's primary researchers, Meghan Hall, created an IRB addendum that added our research team's objectives to Dr. Mestad's current research initiative (see Appendix E). Our research is not supported by the research grant that was awarded to Dr. Mestad's team, although our research outcomes will benefit her team as well. Karen Davis, secretary to Dr. Alex Spadola and administrative assistant for the Upstate Department of OB/GYN Center for Maternal and

Child Health lead the data collection for the current study in place of Dr. Keller who had to leave the study due to professional obligations. Dr. Keller is a resident physician in the obstetrics and gynecology department at Upstate Medical University. Ms. Davis will assist with the collection of retrospective data. These patient data points are already included in the patient charts that have been examined by Dr. Mestad's team. The current research team will be able to determine the BMI of the patients previously examined by Dr. Mestad's team. Using the patient's Edinburg Postnatal Depression Scale, Dr. Mestad's team has depression scores for the patients that will be analyzed for this study.

In this project, the Edinburgh Postnatal Depression Scale (EDPS) will be used to assess patients for depression in the antepartum stage of pregnancy, prior to delivery of the baby. This scale has ten multiple choice questions that can be filled out by patients in approximately two minutes. The result of the survey will be linked to the demographic and pre and post natal information for each patient to link the Edinburgh depression score with the physicality of patients. The incidence of depression will indicate if there is a correlation between obesity and depression in pregnant women. The clinical relevance of this information is that knowledge of an increased incidence of depression will help indicate populations where additional mental health preventive care is needed. Ultimately, this will improve the effectiveness and personalization of medicine.

The patients in this study have been asked by the nurse or physician responsible for the patient to respond to the questions on the Edinburgh scale. The patient will be asked by their nurse or physician to be assessed during one of their routine pre-natal visits. The patients will be told "Would you like to respond to a few questions regarding your mood during your pregnancy?" Using this information, health care providers will be better able to care for all of the

needs of women during pregnancy; not just the physical needs of pregnant patients, but the mental and psychological needs as well. This study will also draw data from previously collected medical records to look at demographic information as well as the gestational age the baby was born, weight, APGAR score, # of days in the NICU if any, risk factors and BMI. These demographic analyses were primarily conducted by Dr. Mestad's team. There is no risk for the patient, except for the potential psychological cost associated with responding to a survey. The basic methods and goals of the research is easily conveyed to the patients in a few sentences, which ensures that the patients have a complete understanding of the research they may choose to get involved with.

Subject Characteristics

The patient population served by Upstate University's UHCC Women's Health Clinic was used in this study. These patients represent a populous from Syracuse, NY an urban community with a population of 145, 169 in 2010 (U.S. Census). According to the 2010 US Census, Syracuse is 56.0% White, 29.5% Black, 8.3% Hispanic or Latino, 5.5% Asian, 5.1% Two or more races and 1.1 % American Indian or Alaska Natives.

Specifically the current project will involve the review of prenatal and hospital delivery charts of 157 UHCC/WHS patients who were residents of Syracuse, New York and gave birth in 2010.

Instrumentation

The Edinburgh Postnatal Depression Scale (EPDS) will be used (see Appendix B). The EPDS was developed for postnatal depression, but it has been validated in antenatal women as well (Gibson et al., 2009). The EPDS items were adapted from from the Irritability, Depression and Anxiety scale and the Hospital Anxiety and Depression Scale (Cox et al. 1987). It has been proposed that dysphoria, or a state of poor emotional well-being that encompasses depressive and anxiety symptoms, rather than just depression alone, has been identified in perinatal mothers using the Edinburgh depression scale (Green 1998). For the ultimate purpose of this study, it would be quite helpful if the EPDS screens for additional psychological maladies because this study aims to identify which patients need extra psychological screening to allow for a more enjoyable pregnancy and motherhood.

The Edinburgh Postnatal Depression Scale is a 10 question administered questionnaire that rates symptoms from 0 to 3 (Kwan et al., 2014). This rating scale is done using a 4-point Likert scale (Kwan et al., 2014). Total score ranges from 0 to 30 with higher scores correlating with greater severity of psychological distress. Several different cutoff scores for depression have been established in different studies. Some studies have used the cutoff 12/13 out of 20, others have used 14/15 out of 20 and still others have used a score of 1 or more on item 10 as indicators for perinatal depression (Gibson et al., 2009, Swalm et al., 2010, Kwan et al., 2014). This study is using a Edinburgh score of 10 as a cutoff for possibility of depression.

Experimental Testing and Procedure

The data was collected for this project in a retrospective fashion. This means that a physician or health care provider gathered the information in the past. This research team was

authorized by the IRB to access the medical records and the data subsequently. This subsequent, retrospective data collection is what took place in this study.

A Collaborative Institutional Training Initiative (CITI) certified individual well trained in patient interviewing previously collected the BMI and Edinburgh data. The mission of CITI is "To promote the public's trust in the research enterprise by providing high quality, peer reviewed, web based, research education materials to enhance the integrity and professionalism of investigators and staff conducting research" *(*CITI Program Mission Statement**).** Essentially, CITI certified individuals have additional ethics, confidentiality and patient interaction training.

The CITI certified health trained interviewer then retrospectively collected BMI, Edinburgh perinatal depression score along with other demographic data. The data was collected from paper charts from the UHCC (University Health Care Clinic, associated with Upstate Medical University). Karen Davis ultimately retrospectively accessed the data for this study.

Statistical Analysis

Our model of data analysis was adoped from Lara et al.'s study that found the prevalence and incidence of perinatal depression and depressive symptoms among Mexican women. In our study, frequencies were calculated for categorical data and means and standard deviations for continuous variables were calculated. Period prevalence was defined as the proportion of total cases of depression in the first, second and third trimester. In our data set, Edinburgh 1 correlates to the Edinburgh scale administered in the first trimester, Edinburgh 2 correlates to the Edinburgh scale administered in the second trimester and Edinburgh 3 correlates to the Edinburgh scale administered in the third trimester. Final analysis compared incidence of depressive symptoms to patient body mass index. Edinburgh response rates for the various BMI

categories previously listed were also calculated to identify if individuals within a certain BMI category responded more or less frequently to the Edinburgh scale. Statistical analyses were conducted using Statistical Package for the Social Sciences (SPSS).

Limitations

A limitation for this study is that all self report scales including the Edinburgh Postnatal Depression Scale, yield higher depression rates than those based on clinical interviews [Halbreich et al., 2006]. It is important to note that the scale is administered by a health care professional but the patient uses her own discretion to answer the Edinburgh questions. The higher depression rates yielded may be due to the rigidity in evaluative methods of the scale. Patients can't verbalize specifically how they feel, they instead have to pick a number on the scale that most closely reflects their feelings. Only psychometric properties are evaluated. This could lead to faulty, inaccurate results because patient's psychological anomalies that are not detectable will not be found.

Women are typically given the diagnosis of depression and anxiety if they fulfill a set of criteria (Kwan et al., 2015). Specifically tailored diagnosis cannot be discerned from the EPDS, patients can only be assigned to a generalized diagnostic category.

Additionally, we do not have information regarding why certain women refused to be tested. We also do not have the demographics of the women that refused to complete the survey. Although this is not a common limitation, knowing the psychological proclivities of the women who did not respond to the survey would help establish a more accurate data set and conclusion. Behaviors such as avoidance, antisocial interactions, lack of openness have been indicators of depression. These behaviors, if they are partially exhibited by patients who refused to answer the survey, could indicate that some of the women who refused to answer the survey may in fact

have had some degree of depression. The fact that this suject subset is not incorporated in the study may actually understate the results and the conclusions about obesity and perinatal depression. It could also lead to us overstating our results and conclusions if these women were in fact not experiencing depression.

We also have only tested women in the clinic. This data reflects an urban population and does not include an accurate representation of suburban or rural communities.

Delimitations

The estimates based on the self-reported questionnaires are extremely cost efficient and do not required specially trained clinicians to administer the assessment [Robertson et al., 2003]. Self reports still have very clinically significant information regarding their depression screening ability and it is important to utilize this technique because especially during the perinatal period, depressive symptoms may be very debilitating for the mother and may result in less desirable pregnancy outcomes [Marcus et al, 2003].

The EPDS provides quick and simple information about mothers' psychological state that does not need to be collected by a physician. This makes it possible for more patients to be screened for depression, in situations where they may not have been screened at all due to the lack of accessibility or availability of a psychological health professional. Using a simple survey screening tool like the EPDS is the first step in completely eliminating perinatal depression and preventing postpartum depression in the obese population.

Chapter 4

Results

This study analyzed 157 patients. These 157 deidentified patients were drawn from the S.E.E.D. dataset that contained 259 individuals. The 157 individuals that were analyzed had a complete data set, including BMI and Edinburgh results. For the 157 patients, the mean BMI was 30.8685. The median BMI was 29.9500. The BMI standard deviation was 7.30493. The 25^{th} percentile for BMI in the patient population was 25.5750, the 50^{th} percentile was 29.9500, the 75^{th} percentile was 35.1000. The range of BMIs in the study was 16.97(extremely underweight) to 54.86 (extremely morbidly obese). This is fairly surprising since the 50^{th} percentile is on the verge of developing obesity. The frequency of each BMI is also listed on pages 1-5 of Appendix F.

Underweight mothers (BMI >18.5) composed 0.6% of the subject population, normal weight mothers (BMI 18.5-24.49) composed 19.1% of the subject population, overweight mothers (BMI 24.5-29.99) composed 30.6% of the population, obese mothers (BMI 30-39.99) consisted of the largest amount of the subject population, representing 38.3% of mothers and morbidly obese mothers (BMI ≥40) composed 11.5% of the subject population.

75.2% of all mothers scored less than 10 on the Edinburgh during all three trimesters, indicating that 75.2% of the population was not depressed. 24.2% of all mothers scored 10 or

greater on the Edinburgh scale at least once during all three trimesters, indicating that 24.2% of the subject population was depressed at some point during the study. This study had a 99.4% response rate, meaning that 99.4% of the mothers given the survey responded from the subject population.

The median score for Edinburgh 1, administered during the first trimester, is 13.00 out of 30, which is a score that indicates depression. The median score for Edinburgh 2, administered during the second trimester, is 12.50 out of 30, which indicates presence of depression. For Edinburgh 3, the vast majority of mothers did not have the Edinburgh administered, so no average score could be calculated for this trimester. (Please see Appendix F, page 8 and the Table titled "Frequencies" for this information.) Appendix F, pages 6-8, show the frequency of the various scores on the Edinburgh scale for each trimester. Interestingly, the highest Edinburgh score recorded in this study, which was recorded in Edinburgh 2 in the second trimester is a score of 26/30 (extremely depressed), which was reported for the subject who had the highest BMI in the study, 54.86 (morbidly obese) (Appendix F, page 9).

The most important data in this study is the crosstabulation of the BMI categories with incidence of depression. 0% of the underweight patients (BMI >18.5) were depressed. (However, there was only one underweight individual in this study.) 30.0% of the normal weight patients (BMI 18.5-24.99) were depressed. 29.2% of the overweight patients (BMI 25.00-29.99) were depressed. 13.6% of the obese patients (BMI 30-39.99) were depressed. 38.9% of the morbidly obese patients (BMI ≥40) were depressed. This data can be seen in the crosstabulation table on page 16 of Appendix F.

The mean BMI for not depressed individuals is 30.6614 and the mean BMI for possibly depressed individuals is 31.2989, seen on page 17 of Appendix F. This difference is not statistically significant.

The response rate was also calculated for the separate BMI categories. For Edinburgh 1, 0.0% of underweight individuals did not respond, 30.0% of normal weight individuals did not respond, 43.8% of overweight individuals did not respond, 26.7% of obese individuals did not respond and 11.1% of morbidly obese individuals did not respond. Fore Edinburgh 2, 0.0% of underweight individuals did not respond, 60.0% of normal weight individuals did not respond, 43.8% of overwight individuals did not respond, 40.0% of obese individuals did not respond and 11.1% of morbidly obese individuals did not respond. This shows that underweight and morbidly obese individuals tended to respond to the Edinburgh scale more frequently than the other weight classes. However, the underweight population only composed 0.6% of the subject population and the morbidly obese population only composed 11.5% of the subject population. Response rates for Edinburgh 3 were not calculated because the response rate was exceptionally low for the in this trimester.

Lastly, we ran a morbid obesity and possibility of depression crosstabulation. Individuals who were not morbidly obese had a 22.5% incidence of depression. Individuals who were morbidly obese had a 38.9% incidence of depression.

Chapter 5

Discussion

Our study has indicated that a BMI of ≥40 during the perinatal stage of pregnancy is correlated with a higher incidence of depression. Our study also indicates that a BMI of <40 during the perinatal stages of pregancy correlates to a moderate risk of depression in women. According to our data, women who are normal weight, overweight and mildly to moderately obese have a similar risk of developing depression during the perinatal stages of pregnancy. On average, about 22.5% of underweight, normal weight, overweight and mildly to moderately obese women had depression, whereas 38.9% of morbidly obese women developed depression.

This is partially consistent with previous studies which have found that depression and anxiety are co morbidities of obesity (Novick et al., 2005; Simon et al., 2006; Kloiber et al., 2007; Singh, 2014 The significance of the present study is that the health of two individuals is found to be affected by perinatal obesity and depression: the mother and child. Epigeneitics has shown us that risk factors such as maternal nutrition and stresses alter the risk of similar (obesity-related) pathologies in her offspring from in-utero exposure (Singh, 2014). Maternal obesity is associated with a increased risk of congenital defects (Arabin et al., 2014). In cases of perinatal depression, not only can developing infants' health be affected by the mother's abnormal neurotransmitter and hormone levels in utero, the mother-child bond postpartum may be at risk as well. A poor post-partum relationship between the child and mother can create an environment for the child that can accentuate any (epi)genetic predispositions to depression or

obesity (Singh, 2014). Prior to this study, research projects on clinical correlations as they relate to treatment options have been lacking. These types of studies are urgently needed in the field of high risk pregnancies in obstetrics and gynecology to reduce the metabolic, cardiovascular and psychological risks for women and children and to minimize the consequential costs for society (Arabin et al., 2014). The outcome of this study has relevance to not only to obstetrics and gynecology but also to pediatrics and child and family welfare. On an individual basis, physiological and psychological wellness often precedes social productivity. Since obesity is an increasing healthcare, social and even political issue in the United States, the issues of perinatal maternal obesity and depression are of critical imporatance.

There has been sufficient physiological data collected that supports this study's finding that exceptionally high BMI (morbid obesity; BMI ≥40) in the perinatal stage is correlated with depression. Excess food and energy supply to the brain will result in abnormal brain physiology. According to Singh, foods have a direct influence on the brain and neurotransmitter systems (Singh 2014). Excess food consumption can cause a shift in neurochemistry, which has a direct effect on brain structure, chemistry and physiology. Mood can influence food choices and food choices can effect mood (Singh 2014). The physiological connection between depression and food consumption can lead to raised plasma levels of CRH, ACTH and cortisol in the mother which may lead to increased likelihood of preterm birth, developmental delays and behavioral abnormalities in children (Weinstock, 2001).

Maternal obesity can cause methylation of cytidine based in cytosine-guanosine nucleotide dimmers (CpG) (DNA methylation). This may lead to an epigenetic change that can be passed transgenerationally (Bird, 2002). It has been found that total methylation in the placentas of obese women is significantly higher than in normal-weight women (Nomura et al.,

2014). This placental methylation can change the regulation of metabolism in the offspring and can also trigger development of chronic metabolic disorders and other physiological disorders potentially involving the brain (Nomura et al., 2014).

There have been several trials, both randomized and non-randomized, that support providing women with evidence-based recommendations (Rasmussen et al., 2004; Dyson et al., 2005). There are a number of treatment protocols that health care providers should consider for women with a BMI of ≥30 (See Appendix G). Maternity services need to identify what resources and services are locally available and accessible for specific patients. According to Arabin (2014), the specific assistance that women with abnormal BMI should receive involves counseling with regards to diet, supplements, exercise and weight gain throughout the pregnancy. Arabin et al., state that women should be counseled to be moderately active (for 30-60 minutes per day) so that any dietary alteration made can be paralleled with activity so that fat-free tissue is not lost (Arabin et al., 2015). Health practitioners should also help women set weight gain goals based on their current BMI (see Appendix H) (Davies et al., 2003). Women with a BMI of >27 have lower serum folate levels even after controlling for folate intake, which suggests that overweight and obese women should possibly receive higher supplemental doses of folate supplementation in order to minimize the risk of fetal neural tube defects (Arabin 2014).

One issue that health care providers may discover in their counseling of patients is a lack of patient compliance or motivation. Patients may be motivated by discussing with them that following dietary and exercise guidelines could reduce their risk for caesarean section, hypertension, abnormal birth weight in their children and birth defects in their children (Davies et al., 2003). Another challenge ties in the fact that that low income and insufficient education are often associated with maternal obesity (Sutherland et al., 2013). This makes clinical

interventions more complex. Health care providers should take care to avoid stigmatizing patients, while identifying and communicating with patients about the consequences of further weight gain (Arabin et al., 2014).

The period of time surrounding and encompassing pregnancy, including the perinatal stage of pregnancy, presents many opportunities for healthcare providers to maximize the health of the mother and child. Maternal obesity is associated with the highest risk of miscarriages and stillbirths and even in the absence of these obstetric catastrophes, the mother and child continue to be at risk for multiple physiological complications (Arabin et al., 2014). A concerted effort between politicians, media, general practitioners, midwives and obstetricians should be made to improve the health care outcome for women who experience perinatal obesity and depression. A practice called strategic litigation by government and private attorneys is beginning to play a role in the obesity prevention movement by monitoring food marketing tactics (Graff et al., 2012). Strategic litigation can bring about significant public policy changes by taking carefully selected cases to court. This type of political and legal involvement serves to draw public attention to the problem of obesity and food marketing; it fuels policy development and spurs industry to change its practices voluntarily (Graff et al., 2012). Health care professionals should also consider engaging in the initiatives previously stated regarding preventive medicine practices as these tactics are essential in the fight against maternal obesity and depression.

Chapter 6

Conclusion

The purpose of the current study is to use biotechnological and clinical measurements to develop a greater understanding of how weight, body mass index (BMI) and obesity can influence perinatal depression. This heightened understanding of perinatal depression can be used as a tool that can tailor medical care more precisely to an individual patients' physiology. We have found that women with a BMI over 40 (whom are classified as morbidly obese) have an increased incidence of depression in pregnancy. Clinically, this information is useful because mothers with BMIs >40 can be identified as being at greater risk for mental dysphoria, specifically depression. Clinical knowledge of the increased risk of depression can allow for greater preventive action such as additional screening procedures, more health care provider inquiry about the mother's mental health status and engagement in more preventive medicine practices.

Moving forward is it important to consider how the screening procedures for obese and other potentially depression-inclined individuals can be made even more precise and accurate. One idea for further research may involve using an expanded scale called the Sheehan-Roland-Moris Disability Scale (see Appendix I). This scale is designed for implementation of scientific modeling to improve treatment outcomes. Instead of providing patients with four choices to choose from to describe their feelings about a particular question, the Sheehan-Roland-Moris Disability Scale has ten choices for patients to choose from to describe their feelings about a given question. The ten-option questions will likely lead to greater accuracy in assessment of patient psychological state.

In healthcare and in society, it is our responsibility to cost-effectively use all the tools at our disposal, especially those of biotechnologically-developed origin which have been developed from physiological and statistically derived data, to better the health and medical treatment of all individuals.

6 Chapters have been redacted

Works Cited

1. Alfaraj I, Spada M, Nikcevic AV, Puffet A, Meer S. 2009. Positive beliefs about rumination in depressed and non-depressed pregnant women: A preliminary investigation. Journal of Reproductive and Infant Psychology, 27, 54-60.
2. Arborelius L, Owens MJ, Plotsky PM, Nemfroff CB. 1999. The role of corticotropin-releasing factor in depression and anxiety disorders. J. Endocrinol. 160, 1-12.
3. Arabin B and Stupin J H. 2014. Overweight and Obesity before, during and after Pregnancy Part 2: Evidence-based Risk Factors and Interventions. Geburtshilfe Frauenheilkd. 74(7); 646-655.
4. Arnal-Remon B, Moreno-Rosset C, Ramirez-Ucles I, Antequera-Jurado R. 2014. Assessing depression, anxiety and couple psychological well-being in pregnancy: a preliminary study. Journal of Reproductive and Infant Psychology.
5. Bird A. 2002. DNA methylation patterns and epigenetic memory. Genes Dev. 16; 6-21.
6. Borges G, Medina-Mora ME, Wang PS, Lara C, Berglund P, Walters E. 2006. Treatment and adequacy of treatment of mental disorders among respondents to the Mexico National Comorbidity Survey. Am J Psychiatry. 163(8); 1371-1378.
7. Bruch, H. 1985. Four Decades of Eating Disorders. New York, NY: Guilford Press.
8. Clement K, Vaisse C, Manning BS et al. Genetic variation in the beta 3-adrenergeic recetpro and an increased capacity to gain weight in patients with morbid obesity. N Engl J Med 1995; 333: 352-4.

9. Chen H, Chan YH, Tan KH, Lee T. 2004. Depressive symptomatology in pregenancy. A Singaporean perspective. Social Psychiatry and Psychiatry Epidemiology, 39; 975-979.
10. Cnattingius S, Lambe M. Trends in smoking and overweight during pregnancy: prevalence, risks of pregnancy complications and adverse pregnancy outocmes. Simin Perinatol 2002; 26: 286-295.
11. Collaborative Institutional Training Initiative (CITI). 2015. University of Miami.
12. Davies G A, Wolfe L A, Mottola M F. et al. 2003. Exercise in pregnancy and the postpartum period. J Obstet Gynaecol Can. 25; 516-529.
13. Della Vedova AM, Ducceschi B, Cesana B, Imbasciati A. 2011. Maternal bonding and risk of depression in late pregnancy: A survey of Italian nulliparous women. Journal of Rreproductive and Infant Psychology, 29; 208-222.
14. deMontigny P, deMontigny F. 2012. Conceiving a first child: Fathers' perceptions of contributing elements to their decision. Journal of Reproductive and Infant Psychology. 31; 274-284.
15. Demyttenaiere K, Nijs P, Evers-Kiebooms F, Koninckx PR. 1989. The effect of a specific emotion stressor on prolactin, cortisol and testosterone concentrations in women varies with their trait anxiety. Fertil Steril. 52; 942-948.
16. Dyson L, McCormick F, Renfew M J. 2005. Interventions for promoting the initiation of breastfeeding. Cochrane Database Syst Rev.
17. Ek J, Urhammer SA, Sorensen TI, Anderson I, Auwerx J, Pedersen O. Homozygosity of the Pro12Ala variant of the peroxisome proliferation-activated receptor-gamma 2 (PPAR-

gamma2): divergent modulating effects on body mass index in obese and elan Caucasian med. Diabetologia 1999; 42: 892-5.

18. Field T, Diego M, Hernandex-Reif M. 2006. Prenatal depression effects on the fetus and newborn: a review. Infant Behav Dev. 29; 1-6.
19. Field T. 2010. Postpartum depression effects on early interactions, parenting, and saftey practices: a review. Infant Behav Dev. 33; 1-6.
20. Fisher J, Carbral de Mello M, Patel V, Rahman A, Ran T, Holton S, Holmes W. 2012. Prevalence and determinants of common perinatal mental disorders in women in low- and lower-middle-income countries: a systemic review. Bull World Health Organ. 90; 139-149.
21. Fleetman JA. Upper airway imaging in relation to obstructive sleep apnea. Clin Chest Med 1992; 13: 399-416.
22. Forsyth C, Skouteris H, Wertheim EH, Paxton SJ, Milgrom J. 2011. Mens's emotional responses to their partner's pregnancy and their views on support and information recieved. Australian and New Zealand Journal of Obstetrics and Gynacology. 51; 53-56.
23. Garbaciak JA Jr, Richter M, Miller S, Baron JJ. Maternal weight and pregnancy complications. Am J Obstet Gynecol 1985; 152: 238-245.
24. Gold PW, Goodwin FK, Chrousos GP. 1988. Clinical and biochemical manifestations of depression: Relation ot hte neuro-biology of stress. N Engl J Med. 319; 348-352.
25. Graff S K, Kappagoda M, Wooten H M, McGowan A K and Ashe M. 2012. Policies for Healthier Communities: Historical, Legal, and Practical Elements of the Obesity Prevention Movement. Annu. Rev. Public Health. 33; 307-24.

26. Grassi G, Seravalle G, Colombo M et al. Body weight reduction, sympathetic nerve traffic, and arterial baroreflex in obese normotensive humans. Circulation 1998; 97: 2037-42.

27. Halbreich U, Karkun S. 2006. Cross-cultural and social diversity of prevalence of postpartum depression and depressive symptoms. J Affect Disord. 91; 97-111.

28. Hammarberg K, Wynter K, Fisher J, McBain J, Gibson F, Boivin J et al. 2013. the experience of pregnancy: Does age or mode of conception matter? Journal of Reproductive and Infant Psychology. 31; 109-120.

29. Heim C, Newport DJ, Heit S, Graham YP, Wilcox M, Bonsall R, Miller AH, Nemroff CB. 2000. Pituitary-adrenal and autonomic responses to stress in women after sexual and physical abuse in childhood. J Am Med Assoc. 284; 595-597.

30. Heron J, O'Connor TG, Evans J, Golding J, Glover V. 2004. What impact does pregnancy have on anxiety about health? Journal of Psychosomatic Obstetrics & Gynecology. 30; 223-230.

31. Holsboer F, Grasser A, Friess E, Wiedemann K. 1994. Steroid effects on central neurons and implications for psychiatric and neurological disorders. Ann NY Acad Sci. 746; 345-359.

32. Kaplan H I and Kaplan H S. The psychosomatic concept of obesity. 1957. J. Nerv. Ment. Dis. 125; 181-201.

33. Koren O, Goodrich JK, Cullender TC, Spor A, Laitinen K, Backhed HK, Gonxaex A, Werner JJ, Angenent LT, Knight R, Backhed F, Isolauri E, Salminen S, Ley RE. Host Remodeling of the Gut Microbiome and Metabolic Changes during Pregnancy. 2012. Cell. 150; 470-480.

34. Kershaw EE, Flier JS. 2004. Adipose tissue as an endocrine organ. J Clin Endocrinol Metab. 89; 2548-56.

35. Kloiber S, Ising M, Reppermund S, Horstmann S, Dose T, Majer M, et al. 2007. Overweight and obesity affect treatment response in major depression. Biol. Psychiatry 62; 321-326.

36. Kurokawa N, Nakai K, Kameo S, Liu ZM, Satoh H. Association of BMI with the beta3-adrenergic receptor gene polymorphism in Japanese: meta-analysis. Obes Res 2001; 9: 741-5.

37. Kwan R, Bautista D, Choo R, Shirong C, Chee C, Saw SM, Chong YS, Kwek K, Meaney MJ, Rush AJ, Chen H. 2015. The Edinburgh Postnatal Depression Scale as a measure for antenatal dysphoria. Journal of Reproductive and Infant Psychology. 33(1); 28-41.

38. Lancaster CA, Flynn HA, Johnson T, Marcus SM, Davis MM. 2010. Periartum length of stay for women with depressive symptoms during pregnancy. Journal of Women's Health. 19; 31-37.

39. Lara MA, Navarrete L, Nieto L, Martin JPB, Navarro JL, Lara-Tapia H. 2015. Prevalence and incidence of perinatal depression and depressive symptoms among Mexican women. J Affect Disord. 175; 18-24.

40. Lee D, Chung T. 2007. Postnatal depression: An update. Best Practice and Research Clinical Obstetrics and Gynaecology. 21; 183-191.

41. Levy-Shiff R, Lerman M, Har-Even D, Hod M. Maternal Adjustment and Infant Outcome in Medically Defined High-Risk Pregnancy. Developmental Psycholgy 2002; 38-1; 93-103.

42. Linne Y. Effects of obesity on women's reproduction and complications during pregnancy. The International Association for the Study of Obesity. obesity reviews. 2004; 5: 137-143.

43. Malhotra A, White DP. Obstructive sleep apnoea. Lancet 2002; 360: 237-45

44. Marcelis M, Navarro-Mateu F, Murray R, Selten JP, Van Os J. 1998. Urbanization and pscyhosis: A study of 1942-1978 birth cohorts in the Netherlands. Psycol Med. 28; 871-879.

45. Marcus SM, Flynn HA, Blow FC, Barry K 2003. Depressive symptoms among pregnant women screened in obstetrics settings. J Womens Health. 12(4); 373-380.

46. Matijasevich A, Golding J, Smith GD, Santos IS, Barros AJ, Victoria CG. 2009. Differentials and income-related inequalities in maternal depression during the first two years after childbirth: birth cohort sudies from Brazil and the UK. Clin Pract Epidemiol Ment Health. 5; 1-12.

47. Mulder EJH, Robles de Medina PG, Huizink AC, Van den Bergh BRH, Buitelaar JK, Visser GHA. 2002. Prenatal maternal stress: effects on pregnancy and the (unborn) child. Early Human Development. 70; 3-14.

48. Murray L, Cooper PJ. 1997. Postpartum depression and child development. Psychol Med. 27(2): 253-260.

49. Nemeroff CB, Widerlov E, Bissette G, Walleus H, Karlsson I, Eklund K, Kilts CD, Loosen PT, Vale W, 1984. Elevated concentrations of CSF corticotropin-releasing factor-like immunoreactivity in depressed patients. Science. 226; 1342-1344.

50. New York Statewide Perinatal Data System, 2013

51. Nomura Y, Lambertini L, Rialdi A. et al. 2014. Global methylation in the placenta and umbilical cord blood from pregnancies with maternal gestational diabetes, preeclampsia, and obesity. Reprod Sci. 21: 131-137.

52. Novick J S, Stewart J W, Wisniewski S R, Cook I A, Manev R, Nierenberg AA, et al. 2005. Clinical and demographic features of atypical depression in outpatients with major depressive disorder: preliminary findings from STAR*D. J. Clin. Psychiatry. 66; 1002-1011.

53. Oakley A, Rajan L, Grant A. 1990. Social support and pregnancy outcome. Br J Obstet Gynaecol. 97; 155-162.

54. O'Hara MW, McCabe JE. 2013. Postpartum depression: current status and future directions. Annu Rev Clin Psychol. 9; 379-407.

55. Orr ST, Blazer DG, James SA, Reiter J. 2007. Depressive symptoms and indicators of maternal health status during pregnancy. Journal of Women's Health. 16; 535-542.

56. Pajulo M, Savonlahti E, Sourander A, Helenius H, Piha J. 2001. Antenatal depression, substance dependency and social support. Journal of Affective Disorders. 65; 9-17.

57. Proietto J, Baur LA. 2004. Management of obesity. Med J Aust. 180; 474-80.

58. Rasmussen K M, Kjolhede C L. 2004. Prepregnant overweight and obesity diminish the prolactin response to suckling in the first week postpartum. Pediatrics.

59. Robertson C, Waldenstrom U, Wickberg B. 2003. Depressive mood in early pregnancy: Prevalence and women at risk in a national Swedish sample. Journal of Reproductive and Infant Psychology. 21; 113-123.

60. Robertson E, Celasun N, Stewart D E. 2003. In: Stewart D E, Robertson E, Dennis C L, Grace S L, Wallington T (Eds), Postpartum Depression: Literature Review of Risk

Factors And Interventions. University Health Network Women's Health Program, Toronto, pp. 71-196.

61. Schacter, S. 1968. Obesity and eating: Internal and external cues differentially affect the eating behavior of obese andnormal subjects. Science. 161; 751-756.
62. Schaffer JE. Lipotoxicity: when tissues overeat. Curr Opin Lipidol 2003; 14: 281-7.
63. Schneider ML, Roughton EC, Koehler AJ, Luback GR. 1999. Growth and devleopment following prenatal stress in primates: an examination of ontogenetic vulnerability. Child Dev. 70; 263-274.
64. Secoli, SR. Teixeira NA. 1998. Chronic prenatal stress affects development and behavioral depression in rats. Stress. 2; 273-280.
65. Seligman, ME. 1972. Learned helplessness. Annu Rev Med. 23; 407-412.
66. Series F, Marc I, Cormier Y, La Forge J. Ulility of nocturnal home oximetry for case finding in patients with suspected sleep apnea hypopnea syndrome. Ann Intern Med 1993; 199: 449-53.
67. Sikaris, KA. The Clinical Biochemistry of Obesity. Clin Biochem Rev 2004; 25: 165-181.
68. Simon G E and Von Korff M. 2006. Medical co-morbidity and validity of DSM-IV depression criteria. Psychol Med. 36; 27-36.
69. Simon G E, Von Korff M, Saunders K, Miglioretti D L, Crane P K, Van Belle G, et al. 2006. Association between obesity and psychiatric disorders in the US adult population. Arch. Gen. Psychiatry. 63; 824-830.
70. Singh, M. Mood, food, and obesity. 2014. Frontiers in Psychology. 5; 1-20.

71. Snyder EE, Walts B, Perusse L et al. 2004. The human obesity gene map: the 2003 update. Obes Res. 12; 369-439.

72. Spinelli MG, Endicott J. 2003. Controlled clinical trial of interpersonal psychotherapy versus parenting education program for depressed pregnant women. The American Journal of Psychiatry. 160; 555-562.

73. Stumvoll M, Tschritter O, Fritsche A et al. Association of the T-G polymorphism in adiponectin (exon 2) with obesity and insulin sensativity: interaction with family history of type 2 diabetes. Diabetes 2002; 51: 37-41.

74. Stanton AL, Lobel M, Sears S, Stein DeLuca R. 2002. Psychosocial aspects of selected issues in women's reproductive health: Current status and future directions. Journal of Consulting and Clinical Psychology. 70; 751-770.

75. Stupin J H and Arabin B. 2014. Overweight and Obesity before, during and after Pregnancy Part I: Pathophysiology, Molecular Biology and Epigenetic Consequences. Geburtshilfe Frauenheilkd. 74(7): 639-645.

76. Sutherland G, Brown S, Yelland J. 2013. Applying a social disparities lens to obesity in pregnancy to inform efforts to intervene. Midwifery.

77. Syracuse University College of Arts and Sciences, Biotechnology webpage 2010

78. Teixeira JMA, Fisk NM, Glover V. Association between maternal anxiety in pregnancy and increased uterine artery resistance index: cohort based study. 1999. BMJ. 318: 153-7.

79. Thiagayson P, Krishnaswamy G, Lim ML, Sung SC, Haley CL, Fung DSS et al. 2012. Depression and anxiety in Singaporean high-risk pregnancies- prevalence and screening. General Hospital Psychiatry. 35; 112-116.

80. Wadhwa, PD, Dunkel-Schetter C, Chicz-DeMet A, Porto M, Sandman CA. 1996. Prenatal pscychosocial factors and the neuroendocrine axis in human pregnancy. Psychosomat Med. 58; 432-446.

81. Wadhwa, PD, Porto M, Garite TJ, Chicz-DeMet A, Sandman CA. 1998. Maternal corticotropin-releasing hormone levels in the early third trimester predict length of gestation in human pregnancy. Am J Obstet Gynecol. 179; 1079-1085.

82. Weinstock M. 2001. Alterations induced by gestational stress in brain morphology and behavior of the offspring. Progress in Neurobiology. 65; 427-451.

83. Whisman MA, Davila J, Goodman SH. 2011. Relationship adjustment, depression and anxiety during pregnancy and the postpartum period. Journal of Family Psychology. 25; 375-383.

84. Widen E, Lehto M, Kanninen T, Walston J, Shuldiner AR, Groop LC. Association of a polymorphism in the beta 3-adrenergic-receptor gene with features of the insulin resistance syndrom in Finns. N Engl J Med 1995; 333 348-51.

85. World Health Organization. The global epidemic of obesity. Geneva, Switzerland: World Health Organization. 1997.

Appendices

Mother's Name:	Mother's Med. Rec. Number:

New Birth Registration

Parents – Mother

Mother's First Name:	Mother's Middle Name:
Mother's Current Last Name :	Last Name on Mother's Birth Certificate:
Social Security Number: – –	Mother's Date of Birth: *(MM/DD/YYYY)* / /
Infant's First Name:	Infant's Middle Name:
Infant's Last Name:	Infant's Name Suffix *(e.g. Jr., 2nd, III)*:

Infant

Sex: ☐ Male ☐ Female ☐ Undetermined	Plurality:	Birth Order:	Medical Record No.:
Date of Birth: *(MM/DD/YYYY)* / /	Time of Birth: *(HH:MM)* :	☐ am ☐ pm ☐ military (24-hour time)	

Parents – Infant

Was child born in this facility? ☐ Yes ☐ No If child was **not** born in this facility, please answer the following questions:

In what type of place was the infant born?

☐ Freestanding Birth Center (regulated by DOH)
☐ Home (intended)
☐ Home (unintended)
☐ Home (unknown intent)
☐ Clinic / Doctor's Office (not regulated by DOH)
☐ Other

If New York State Birthing Center, enter its name:

In what county was the child born?

Parents – Birthplace

Institution

Site of Birth, If **Other** Type of Place:	Street Address – if other than Hospital / Birthing Center:

If place of infant's birth was other than Hospital or Birthing Center:

City, town or village where birth occurred:	Zip / Postal Code:

Infant's Pediatrician/Family Practitioner: **NBS**

Attendant

Attendant's Information:

License Number: | Name: *First* *Middle* *Last*

Title: *(Select one)*

☐ Medical Doctor ☐ Doctor of Osteopathy ☐ Licensed Midwife (CNM) ☐ Licensed Midwife (CM) ☐ Other

Certifier

Certifier's Information:

☐ Check here if the Certifier is the same as the Attendant *(otherwise enter information below)*

License Number: | Name: *First* *Middle* *Last*

Title: *(Select one)*

☐ Medical Doctor ☐ Doctor of Osteopathy ☐ Licensed Midwife (CNM) ☐ Licensed Midwife (CM) ☐ Other

Parents – Payor

Primary Payor for this Delivery:

Select one:

☐ Medicaid / Family Health Plus
☐ CHAMPUS / TRICARE
☐ Self-pay
☐ Private Insurance
☐ Other Government / Child Health Plus B
☐ Indian Health Service
☐ Other

If Medicaid is not the primary payor, is it a secondary payor for this delivery? ☐ Yes ☐ No

Is the mother enrolled in an HMO or other managed care plan? ☐ Yes ☐ No **QI**

Mother's Name: *First*	*Middle*	*Last*	Mother's Med. Rec. Number:
Father / Second Parent Name: *First*	*Middle*	*Last*	*Suffix*
Infant's Name: *First*	*Middle*	*Last*	*Suffix* Date of Birth

To the hospital:

1. Obtain the parent(s) signature(s).
2. File the original Release Form in the mother's hospital record.
   Note: It is not necessary to file the remainder of the Work Booklet.
3. Provide a copy to the parent(s).
4. Do **not** send copies to the New York State Department of Health or to any Social Security office, unless specifically requested by such agency.

To the parent(s):

1. Please read the following notice about the collection and use of Social Security Numbers on your child's birth certificate.
2. Please check "Yes" or "No" to indicate if you wish to participate in the Social Security Administration's Enumeration at Birth program.

NOTICE REGARDING COLLECTION OF PARENTS' SOCIAL SECURITY NUMBERS: The collection of parents' Social Security Numbers on the New York State Certificate of Live Birth is mandatory. They are required by Public Health Law Section 4132(1) and may be used for child support enforcement, public health related purposes, when requested by State, federal and municipal governments for official purposes, when required by Public Health Law Section 4173 or 4174, and when otherwise required or authorized by law.

Social Security Release

The Social Security Administration offers the parents of newborns an opportunity to apply for a Social Security Number for their child through the birth certificate registration process. This is referred to by the Social Security Administration as Enumeration at Birth (EAB). If you participate in the EAB, the New York State Department of Health will forward to the Social Security Administration information from your child's birth certificate. Please note that the Social Security Administration will not process your EAB request unless, the birth certificate includes your child's full name. If you participate in the EAB, disclosure of parents' Social Security Numbers is mandated by 42 U.S.C. 405(c)(2). The Social Security Number(s) will be used by the Internal Revenue Service (IRS) solely for the purpose of determining Earned Income Tax Credit compliance. If you wish to participate in the Social Security Administration EAB program check "Yes" below.

May the Social Security Administration be furnished with information from this form to issue your child a social security number?

Yes ☐

No ☐

Mother's Signature ▶ _____ **Date** _____

Father's or Second Parent's Signature ▶ _____ **Date** _____

Either parent's signature applies to the above release.
If neither box is checked for the release, a 'No' response will be assumed.

Hospital Name:	
Signature of Hospital Representative ▶	Date:

Mother's Name:	Mother's Med. Rec. Number:

Mother

Medical Record Number:

Parents

Mother's Demographics

Mother's Education: *(select one)*

- ☐ 8th grade or less
- ☐ 9th – 12th grade; no diploma
- ☐ High school graduate; or GED
- ☐ Some college credit, but no degree
- ☐ Associate's degree
- ☐ Bachelor's degree
- ☐ Master's degree
- ☐ Doctorate degree

City of Birth:	State/Terr./Province of Birth:	Country of Birth, if not USA:

Hispanic Origin:

Select all that apply

- ☐ No, not Spanish/Hispanic/Latina
- ☐ Yes, Cuban
- ☐ Yes, Mexican, Mexican American, Chicana
- ☐ Yes, Other Spanish/Hispanic/Latina
  Specify:
- ☐ Yes, Puerto Rican

Mother's Demographics

Race:

Select all that apply

- ☐ White/Caucasian
- ☐ Chinese
- ☐ Korean
- ☐ Guamanian or Chamorro
- ☐ American Indian or Alaska Native **Tribe:**
- ☐ Other Asian **Specify:**
- ☐ Other Pacific Islander **Specify:**
- ☐ Other **Specify:**
- ☐ Black or African American
- ☐ Filipino
- ☐ Vietnamese
- ☐ Samoan
- ☐ Asian Indian
- ☐ Japanese
- ☐ Native Hawaiian

Mother's Residence

Residence Address

Street Address:

State/Terr./Province:	County:	City, Town or Village:

Zip/Postal Code:	Mother's Country of Residence, if not USA:	U.S./Canadian Phone Number: ( ) –

Mother's Mailing Address

Mailing Address – Most Recent

☐ Check here if the mailing address is the same as the residence address *(otherwise enter information below)*

Mailing Address:

City, Town or Village:	State/Terr./Province:	Country, if not USA:	Zip/Postal Code:

Employment

Employment History

Employed while Pregnant: ☐ Yes ☐ No	Current / Most Recent Occupation:	Kind of Business / Industry:

Name of Company or Firm:	Address:

City:	State/Territory/Province:	Zip / Postal Code:

Mother's Name:	Mother's Med. Rec. Number:

Father or Second Parent

Will the mother and father be executing an Acknowledgement of Paternity? ☐ Yes ☐ No ☐ Not required

What type of certificate is required? ☐ Mother / Father ☐ Mother / Mother

Parents

Father's or Second Parent's Demographics

Parent's First Name:	Parent's Middle Name:
Parent's Current Last Name:	Last Name on Parent's Birth Certificate:
Parent's Name Suffix *(e.g. Jr., 2nd, III)*:	Social Security Number: – –

Demographics

Parent's Date of Birth: *(MM/DD/YYYY)* / /

Education: *(select one)*

- ☐ 8th grade or less
- ☐ 9th – 12th grade; no diploma
- ☐ High school graduate; or GED
- ☐ Some college credit, but no degree
- ☐ Associate's degree
- ☐ Bachelor's degree
- ☐ Master's degree
- ☐ Doctorate degree

City of Birth:	State/Terr./Province of Birth:	Country of Birth, if not USA:

Hispanic Origin:

Select all that apply

- ☐ No, not Spanish/Hispanic/Latino
- ☐ Yes, Cuban
- ☐ Yes, Mexican, Mexican American, Chicano
- ☐ Yes, Other Spanish/Hispanic/Latino — **Specify:**
- ☐ Yes, Puerto Rican

Race:

Select all that apply

- ☐ White/Caucasian
- ☐ Chinese
- ☐ Korean
- ☐ Guamanian or Chamorro
- ☐ American Indian or Alaska Native **Tribe:**
- ☐ Other Asian **Specify:**
- ☐ Other Pacific Islander **Specify:**
- ☐ Other **Specify:**
- ☐ Black or African American
- ☐ Filipino
- ☐ Vietnamese
- ☐ Samoan
- ☐ Asian Indian
- ☐ Japanese
- ☐ Native Hawaiian

Parent's Residence

Residence Address

☐ Check here if the parent's residence address is the same as the mother's address *(otherwise enter information below)*

Street Address:	
City, Town or Village:	State / Territory / Province:
Parent's Country of Residence, if not USA:	Zip / Postal Code:

Employment

Employment History

Current / Most Recent Occupation: Kind of Business / Industry:

Name of Company or Firm:	Address:	
City:	State / Territory / Province:	Zip / Postal Code:

Mother's Name:	Mother's Med. Rec. Number:

Interview/Records QI

Parents

Survey of Mother (in hospital)

Survey of Mother (in hospital)

Did you receive prenatal care? ☐ Yes ☐ No *(If 'Yes' please answer question 1. Otherwise skip to question 2.)*

1. During any of your prenatal care visits, did a doctor, nurse, or other health care worker talk with you about any of the things listed below?

	Yes	No
a. How smoking during pregnancy could affect your baby?	☐	☐
b. How drinking alcohol during your pregnancy could affect your baby?	☐	☐
c. How using illegal drugs could affect your baby?	☐	☐
d. How long to wait before having another baby?	☐	☐
e. Birth control methods to use after your pregnancy?	☐	☐
f. What to do if your labor starts early?	☐	☐
g. How to keep from getting HIV (the virus that causes AIDS)?	☐	☐
h. Physical abuse to women by their husbands or partners?	☐	☐

2. How many times per week during your current pregnancy did you exercise for 30 minutes or more, above your usual activities? — Times per week:

3. Did you have any problems with your gums at any time during pregnancy, for example, swollen or bleeding gums? ☐ Yes ☐ No

4. During your pregnancy, would you say that you were: *(select one)*
   ☐ Not depressed at all ☐ A little depressed
   ☐ Moderately depressed ☐ Very depressed
   ☐ Very depressed and had to get help

5. Thinking back to just before you were pregnant, how did you feel about becoming pregnant?
   ☐ You wanted to be pregnant sooner ☐ You wanted to be pregnant later
   ☐ You wanted to be pregnant then ☐ You didn't want to be pregnant then or at any time in the future

Chart Review (Prenatal and Medical)

Chart Review (Prenatal and Medical)

1a. Copy of prenatal record in chart?
   ☐ Yes, Full Record ☐ Yes, Prenatal Summary Only
   ☐ No

1b. Was formal risk assessment in prenatal chart?
   ☐ Yes, with Social Assessment ☐ Yes, without Social Assessment
   ☐ No

1c. Was MSAFP / triple screen test offered?
   ☐ Yes ☐ No
   ☐ No, Too Late

1d. Was MSAFP / triple screen test done?
   ☐ Yes ☐ No

2. How many times was the mother hospitalized during this pregnancy, not including hospitalization for delivery?

Admission & Discharge

Admission and Discharge Information

Mother

Admission Date for Delivery *(MM/DD/YYYY)* / / — Discharge Date *(MM/DD/YYYY)* / /

Infant

Discharge Date *(MM/DD/YYYY)* / /

☐ Discharged Home ☐ Infant Died at Birth Hospital
☐ Infant Still in Hospital ☐ Infant Discharged to Foster Care/Adoption
☐ Infant Transferred Out ☐ Unknown

Edinburgh Postnatal Depression Scale[1] (EPDS)

Postpartum depression is the most common complication of childbearing.[2] The 10-question Edinburgh Postnatal Depression Scale (EPDS) is a valuable and efficient way of identifying patients at risk for "perinatal" depression. The EPDS is easy to administer and has proven to be an effective screening tool.

Mothers who score above 13 are likely to be suffering from a depressive illness of varying severity. The EPDS score should not override clinical judgment. A careful clinical assessment should be carried out to confirm the diagnosis. The scale indicates how the mother has felt ***during the previous week***. In doubtful cases it may be useful to repeat the tool after 2 weeks. The scale will not detect mothers with anxiety neuroses, phobias or personality disorders.

Women with postpartum depression need not feel alone. They may find useful information on the web sites of the National Women's Health Information Center <www.4women.gov> and from groups such as Postpartum Support International <www.chss.iup.edu/postpartum> and Depression after Delivery <www.depressionafterdelivery.com>.

SCORING

QUESTIONS 1, 2, & 4 (without an *)
Are scored 0, 1, 2 or 3 with top box scored as 0 and the bottom box scored as 3.

QUESTIONS 3, 5-10 (marked with an *)
Are reverse scored, with the top box scored as a 3 and the bottom box scored as 0.

Maximum score: 30
Possible Depression: 10 or greater
Always look at item 10 (suicidal thoughts)

Instructions for using the Edinburgh Postnatal Depression Scale:

1. The mother is asked to check the response that comes closest to how she has been feeling in the previous 7 days.

2. All the items must be completed.

3. Care should be taken to avoid the possibility of the mother discussing her answers with others. (Answers come from the mother or pregnant woman.)

4. The mother should complete the scale herself, unless she has limited English or has difficulty with reading.

[1]Source: Cox, J.L., Holden, J.M., and Sagovsky, R. 1987. Detection of postnatal depression: Development of the 10-item Edinburgh Postnatal Depression Scale. *British Journal of Psychiatry* 150:782-786.

[2]Source: K. L. Wisner, B. L. Parry, C. M. Piontek, Postpartum Depression N Engl J Med vol. 347, No 3, July 18, 2002, 194-199

Edinburgh Postnatal Depression Scale[1] (EPDS)

Name: _____ Address: _____

Your Date of Birth: _____ _____

Baby's Date of Birth: _____ Phone: _____

As you are pregnant or have recently had a baby, we would like to know how you are feeling. Please check the answer that comes closest to how you have felt **IN THE PAST 7 DAYS**, not just how you feel today.

Here is an example, already completed.

I have felt happy:
- ☐ Yes, all the time
- ☒ Yes, most of the time
- ☐ No, not very often
- ☐ No, not at all

This would mean: "I have felt happy most of the time" during the past week. Please complete the other questions in the same way.

In the past 7 days:

1. I have been able to laugh and see the funny side of things
   - ☐ As much as I always could
   - ☐ Not quite so much now
   - ☐ Definitely not so much now
   - ☐ Not at all

2. I have looked forward with enjoyment to things
   - ☐ As much as I ever did
   - ☐ Rather less than I used to
   - ☐ Definitely less than I used to
   - ☐ Hardly at all

*3. I have blamed myself unnecessarily when things went wrong
   - ☐ Yes, most of the time
   - ☐ Yes, some of the time
   - ☐ Not very often
   - ☐ No, never

4. I have been anxious or worried for no good reason
   - ☐ No, not at all
   - ☐ Hardly ever
   - ☐ Yes, sometimes
   - ☐ Yes, very often

*5 I have felt scared or panicky for no very good reason
   - ☐ Yes, quite a lot
   - ☐ Yes, sometimes
   - ☐ No, not much
   - ☐ No, not at all

*6. Things have been getting on top of me
   - ☐ Yes, most of the time I haven't been able to cope at all
   - ☐ Yes, sometimes I haven't been coping as well as usual
   - ☐ No, most of the time I have coped quite well
   - ☐ No, I have been coping as well as ever

*7 I have been so unhappy that I have had difficulty sleeping
   - ☐ Yes, most of the time
   - ☐ Yes, sometimes
   - ☐ Not very often
   - ☐ No, not at all

*8 I have felt sad or miserable
   - ☐ Yes, most of the time
   - ☐ Yes, quite often
   - ☐ Not very often
   - ☐ No, not at all

*9 I have been so unhappy that I have been crying
   - ☐ Yes, most of the time
   - ☐ Yes, quite often
   - ☐ Only occasionally
   - ☐ No, never

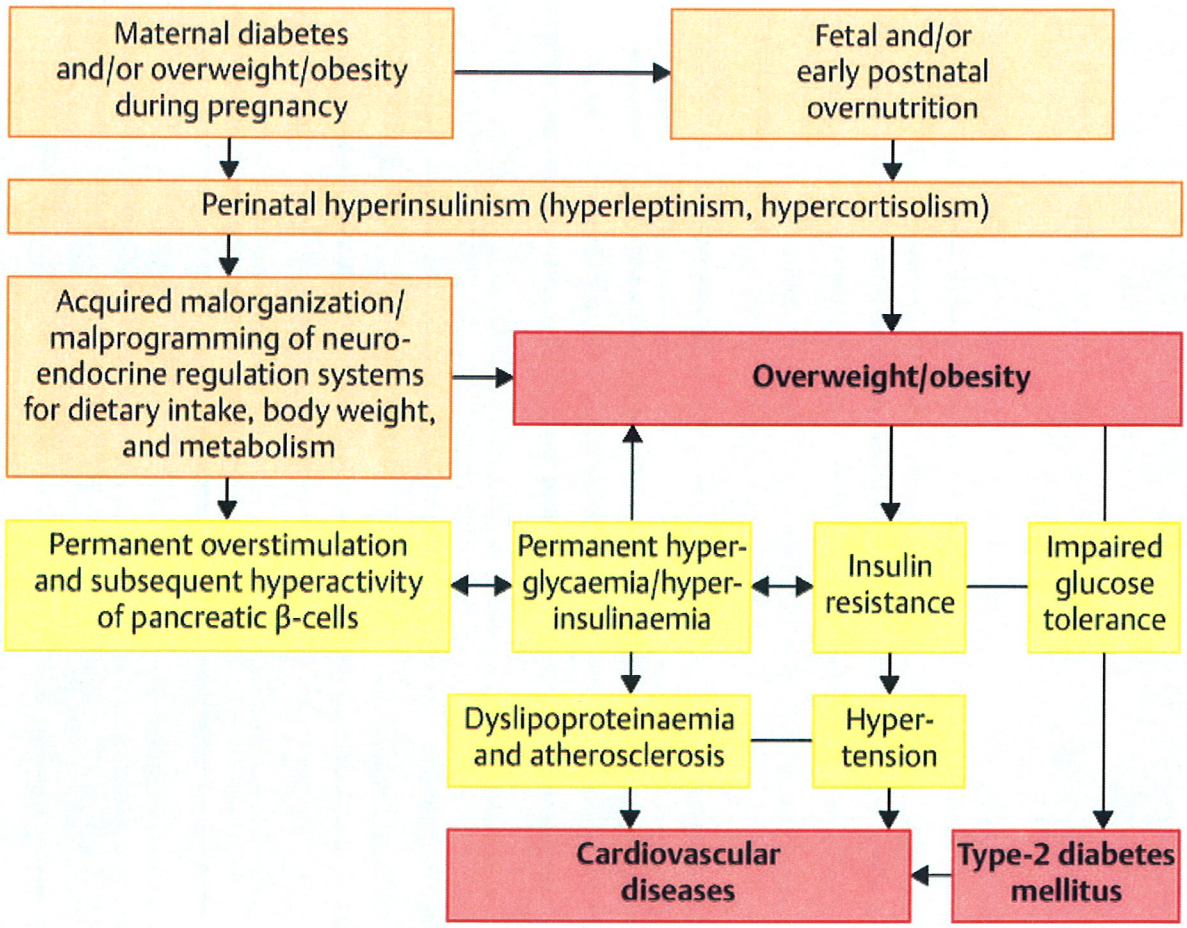
*10 The thought of harming myself has occurred to me
   - ☐ Yes, quite often
   - ☐ Sometimes
   - ☐ Hardly ever
   - ☐ Never

Administered/Reviewed by _____ Date _____

[1]Source: Cox, J.L., Holden, J.M., and Sagovsky, R. 1987. Detection of postnatal depression: Development of the 10-item Edinburgh Postnatal Depression Scale. *British Journal of Psychiatry* 150:782-786 .

[2]Source: K. L. Wisner, B. L. Parry, C. M. Piontek, Postpartum Depression N Engl J Med vol. 347, No 3, July 18, 2002, 194-199

Appendix C



Pathogenetic mechanisms, and consequences of perinatal malprogramming. Aetiology of pre- and neonatal overfeeding and hyperinsulinism for excessive weight gain, overweight/obesity, type-2 diabetes mellitus and subsequent cardiovascular diseases in later life.

[Stupin et al., 2014]

IDNumber	BMI	Height (inches)	Weight (lbs)	Edinburgh1	Edinburgh2	Edinburgh3
001'	16.97	5'. 4"	100.6.4	0	2	99
004	30.99	4'11	156	8	5	99
005'	18.65	5' 2"	102	5	5	0
006'	31.88	5' .1"	171.8	99	99	99
007'	29.69	5'	152	99	99	99
008'	29.12	5'.7"	186	99	99	99
009'	30.42	5' 8"	200	6	5	0
010'	33.02	5'.3"	188	4	4	2
012'	32.3	5.'6"	200	3	3	99
013'	30.68	5'.5"	190	4	0	99
015'	22.18	5'.9"	150.4	11	8	99
017'	32.65	5'.7"	208.8	6	3	99
018'	32.69	5'3"	184	17	2	99
019'	29.41	5'.3"	166	5	15	99
021'	27.4	5'4"	160	99	99	99
022'	51.05	5'3"	295	7	3	7
025	29.72	5'.7"	189.12	99	3	99
027'	27.11	5'.3"	153	99	1	1
028	28.12	5'8"	189	99	2	2
030'	24.14	5'2"	132	99	99	99
031'	21.81	5'.4"	129	0	99	99
035'	32.22	5'5"	199.8	6	2	99
036'	31.72	5'.3"	179	99	4	99
037'	34.53	5"5"	207.8	8	6	99
038'	37.4	5'.4"	218	99	0	99
040'	32.24	5'.8"	212	3	2	99
041'	29.06	5'.3"	164	99	99	99
042'	42.07	5'.4"	247	8	7	4
043'	34.2	5'3."	193	8	2	99
047'	23.58	5"6"	146	99	99	99
048'	22.99	5'.2"	125	12	6	99
049'	25.69	5'.3"	145	10	99	99
051'	21.05	5'2"	116	12	14	12
052'	29.94	5'.4"	157	0	1	0
053	38.94	5'.1"	206		6	
054	32.45	5'.5"	195	7	99	1
055	40.31	5'.2"	220	10	3	99
057'	40.37	5'.6"	250	0	99	99
60'	27.89	5'.4"	167	99	0	99
061'	29.37	5'.9"	199	3	99	99
062	39.68	5'.3"	224	6	99	19
063'	27.5	5"6"	171	8	99	4
066'	22.07	5'	113	8	99	3

070'	32.7	5'8"	215	99	6	99
071'	27.43	5'.2"	150	99	99	99
072'	26.75	5'	141.8	14	15	14
073'	30.35	5'.4"	176.14	2	6	0
075'	31.8	5'2.9"	179	4	8	99
076'	37.83	5'.3"	213	99	0	99
080'	39.11	5'.5"	235	3	9	99
081'	24.8	5'.6"	156	1	1	99
082'	30.26	5'.1"	161	10	99	3
084'	23.44	5'.1"	124	4	99	99
085'	26.37	5'.7"	171	99	3	99
086'	27.2	5'.4"	162	99	99	99
087'	33.95	5'.9"	230	1	3	99
088'	43.99			5	3	99
089'	35.53	5'.6"	220	22	24	99
090'	26.94	5'.4"	157	7	9	99
091'	21.79	5'.2"	123	17	14	23
093'	32.25	5'.4"	188	8	17	99
095'	28.71	5'.3"	157	8	14	9
096'	32.43	5'.3"	183	1	4	0
097'	54.86	5'.2"	300	16	26	99
101'	39.41	5'.9	267	3	1	99
102'	36.11	5'.5"	222	6	99	3
104'	25.09	4'.10"	120	15	0	99
105'	22.72	5'.3"	128	7	6	3
109'	30.26	5'.9	176.5	99	99	99
110'	24.22	5'.6"	150	12	99	99
111'	36.63	5'.3"	206	3	0	99
112'	22.86	5'.7."	146	8	99	99
116'	23.68	5'.4"	138	16	12	99
118'	35.22	5'.11"	252	8	99	99
120'	28.03	5'.2"	157	99	99	99
121'	36.18	5'4.5"	214	2	99	99
122'	21.51	5'.6"	135	7	99	99
123'	24.81	5'.3"	140	13	0	99
125'	32.62	5'.6"	202	7	3	0
126'	30.53	5'.7"	195	0	3	99
127'	26.63	5'.5"	160	10	99	99
128'	25.84	5'.5"	160	14	11	99
129'	30.2	5'.4"	176	3	9	99
130'	19.73	5'.4"	115	99	99	99
132'	21.68	5'.3"	123	99	4	99
133'	26.03	5'.1"	140	99	99	99
136'	23.42	4'.11"	116	8	99	4

139'	25.91	5'.4"	151	2	1	99
141'	44.3	5'.3"	250	8	6	99
143'	21.03	4'.9"	100	0	99	99
144'	54.1	5'.5"	325	7	15	6
145'	21.74	4'.10"	104	99	99	99
146'	27.58	5'.2"	152	8	7	99
147'	28.63	5'.5"	174	0	0	0
148'	39.15	5'.7"	250	13	99	99
149'	39.98	5'.3"	220	99	10	6
150'	32.94	5'.4"	192	5	5	99
151'	30.17	5'.2"	165	99	99	99
152'	28.5	5'.7"	182	3	0	10
153'	21.03	5'.9"	142	2	99	99
155'	38.5	5'.3"	220	99	99	0
156'	20.8	5'.1"	110	22	19	99
158'	33.14	5'.4"	196	3	99	99
161'	30.76	5'.3"	173	5	5	99
164'	44.82	4'.6"	186	99	28	99
165'	22.11	5'.5"	136	0	99	99
166'	32.3	5'.6"	200	7	1	6
168'	45.37	5'.1"	240	16	12	99
170'	30.17	5'.2"	165	7	7	99
171'	29.53	4'.2"	105	0	99	99
172'	29.35	5'.2"	160	12	8	99
173'	25.46	5'.6"	159	99	13	99
174'	41.69	5'.2"	231	3	10	3
175'	28.34	5'.9"	192	18	12	99
176'	41.52	5'.4"	242	6	6	99
177'	21.09	5'.3"	119	99	99	99
179'	27.64	5'.3"	156	99	99	99
181'	33.13	5'.3"	187	0	99	99
183'	28.69	5'.3"	166	99	99	99
184'	35.86	5'.4"	209	2	99	99
187'	43.41	5'.4"	253	10	2	0
189'	34.45	5'.7"	220	8	99	99
190'	39.89	5'.10"	278	7	99	9
191'	35.43	5'.9"	240	10	99	99
192'	22.51	5'.3"	144	0	1	99
195'	46.11	5'.1"	248	9	5	99
196'	26.42	5'.4"	154	1	2	99
197'	24.14	5'.2"	132	13	99	99
198'	41.51	5'.4"	249	6	99	99
199'	30.26	5'.7"	193	9	99	99
202'	42.43	5'.5"	255	99	1	2

203'	24.91	5'.2"	136	10	0	99
204'	26.29	5'.5"	158	99	7	99
209'	33.03	5'.8"	217	1	99	99
211'	21.79	5'.4"	130	17	13	99
213'	28.88	5'.3"	163	99	99	99
214'	27.53	5'.7"	181	99	99	99
215'	48.27	5'.2"	264	5	5	99
216'	27.7	5'.1"	146	18	7	99
220'	31.82	5'.2"	174	99	99	99
222'	34.98	5'.8"	230	0	0	99
224'	29.95	5'	138	99	99	99
225'	23.5	5'4"	137	99	99	99
226'	25.77	5'.9"	177	8	99	99
227'	45.35	5'.2"	248	1	0	0
228'	32.35	5'.6"	202	99	99	99
230'	28.82	5'.4"	168	1	0	99
232'	19.39	5'.4"	113	99	99	99
234'	23.44	5'	120	99	2	3
235'	24.69	5'.2"	135	8	99	99
236'	36.67	5'.1"	194	99	99	99
241'	35.59	5'.8"	234	0	2	1
242'	35.78	5'.5"	215	99	99	99
268'	31.75	5'5"	190	99	99	99
269'	24.88	5'.4"	145	99	1	6
274'	39.32	5'8"	264	99	8	99
275'	24.84	5'.3"	140	3	99	99

Date: January 26, 2015

RE: Revised IRB Exemption Request

Principal Investigator: Renee Mestad, MD

Study Name: 389664-1:Correlates and Consequences of Prenatal Depression: An exploratory Study

To Whom This May Concern,
Study staff have been updated to include a new study staff member, Matthew Keller, MD, who will assist with collection of retrospective data. Additionally I am including a revised data abstraction form to include the following data points requested per the study PI:

- Height
- Weight
- Pregnancy History
- ER Admissions during pregnancy

The aforementioned data points are available within the patient charts and are germane to the study objectives of finding correlation between maternal prenatal health and perinatal depression.

Thank you for your time and consideration in reviewing this revised application for exemption. If you have any questions regarding this request for study exemption, please contact the study coordinator, Meghan Hall, at hallm@upstate.edu or at (315) 464-4631.

Kind Regards,

Meghan Hall, MS, CCRP

Frequencies

Statistics

BMI

N	Valid	157
	Missing	0
Mean		30.8685
Median		29.9500
Std. Deviation		7.30493
Percentiles	25	25.5750
	50	29.9500
	75	35.1000

BMI

		Frequency	Percent	Valid Percent	Cumulative Percent
Valid	16.97	1	.6	.6	.6
	18.65	1	.6	.6	1.3
	19.39	1	.6	.6	1.9
	19.73	1	.6	.6	2.5
	20.80	1	.6	.6	3.2
	21.03	2	1.3	1.3	4.5
	21.05	1	.6	.6	5.1
	21.09	1	.6	.6	5.7
	21.51	1	.6	.6	6.4
	21.68	1	.6	.6	7.0
	21.74	1	.6	.6	7.6
	21.79	2	1.3	1.3	8.9
	21.81	1	.6	.6	9.6
	22.07	1	.6	.6	10.2
	22.11	1	.6	.6	10.8
	22.18	1	.6	.6	11.5
	22.51	1	.6	.6	12.1
	22.72	1	.6	.6	12.7
	22.86	1	.6	.6	13.4
	22.99	1	.6	.6	14.0
	23.42	1	.6	.6	14.6
	23.44	2	1.3	1.3	15.9
	23.50	1	.6	.6	16.6
	23.58	1	.6	.6	17.2
	23.68	1	.6	.6	17.8
	24.14	2	1.3	1.3	19.1

BMI

	Frequency	Percent	Valid Percent	Cumulative Percent
24.22	1	.6	.6	19.7
24.69	1	.6	.6	20.4
24.80	1	.6	.6	21.0
24.81	1	.6	.6	21.7
24.84	1	.6	.6	22.3
24.88	1	.6	.6	22.9
24.91	1	.6	.6	23.6
25.09	1	.6	.6	24.2
25.46	1	.6	.6	24.8
25.69	1	.6	.6	25.5
25.77	1	.6	.6	26.1
25.84	1	.6	.6	26.8
25.91	1	.6	.6	27.4
26.03	1	.6	.6	28.0
26.29	1	.6	.6	28.7
26.37	1	.6	.6	29.3
26.42	1	.6	.6	29.9
26.63	1	.6	.6	30.6
26.75	1	.6	.6	31.2
26.94	1	.6	.6	31.8
27.11	1	.6	.6	32.5
27.20	1	.6	.6	33.1
27.40	1	.6	.6	33.8
27.43	1	.6	.6	34.4
27.50	1	.6	.6	35.0
27.53	1	.6	.6	35.7
27.58	1	.6	.6	36.3
27.64	1	.6	.6	36.9
27.70	1	.6	.6	37.6
27.89	1	.6	.6	38.2
28.03	1	.6	.6	38.9
28.12	1	.6	.6	39.5
28.34	1	.6	.6	40.1
28.50	1	.6	.6	40.8
28.63	1	.6	.6	41.4
28.69	1	.6	.6	42.0
28.71	1	.6	.6	42.7
28.82	1	.6	.6	43.3

BMI

	Frequency	Percent	Valid Percent	Cumulative Percent
28.88	1	.6	.6	43.9
29.06	1	.6	.6	44.6
29.12	1	.6	.6	45.2
29.35	1	.6	.6	45.9
29.37	1	.6	.6	46.5
29.41	1	.6	.6	47.1
29.53	1	.6	.6	47.8
29.69	1	.6	.6	48.4
29.72	1	.6	.6	49.0
29.94	1	.6	.6	49.7
29.95	1	.6	.6	50.3
30.17	2	1.3	1.3	51.6
30.20	1	.6	.6	52.2
30.26	3	1.9	1.9	54.1
30.35	1	.6	.6	54.8
30.42	1	.6	.6	55.4
30.53	1	.6	.6	56.1
30.68	1	.6	.6	56.7
30.76	1	.6	.6	57.3
30.99	1	.6	.6	58.0
31.72	1	.6	.6	58.6
31.75	1	.6	.6	59.2
31.80	1	.6	.6	59.9
31.82	1	.6	.6	60.5
31.88	1	.6	.6	61.1
32.22	1	.6	.6	61.8
32.24	1	.6	.6	62.4
32.25	1	.6	.6	63.1
32.30	2	1.3	1.3	64.3
32.35	1	.6	.6	65.0
32.43	1	.6	.6	65.6
32.45	1	.6	.6	66.2
32.62	1	.6	.6	66.9
32.65	1	.6	.6	67.5
32.69	1	.6	.6	68.2
32.70	1	.6	.6	68.8
32.94	1	.6	.6	69.4
33.02	1	.6	.6	70.1

BMI

	Frequency	Percent	Valid Percent	Cumulative Percent
33.03	1	.6	.6	70.7
33.13	1	.6	.6	71.3
33.14	1	.6	.6	72.0
33.95	1	.6	.6	72.6
34.20	1	.6	.6	73.2
34.45	1	.6	.6	73.9
34.53	1	.6	.6	74.5
34.98	1	.6	.6	75.2
35.22	1	.6	.6	75.8
35.43	1	.6	.6	76.4
35.53	1	.6	.6	77.1
35.59	1	.6	.6	77.7
35.78	1	.6	.6	78.3
35.86	1	.6	.6	79.0
36.11	1	.6	.6	79.6
36.18	1	.6	.6	80.3
36.63	1	.6	.6	80.9
36.67	1	.6	.6	81.5
37.40	1	.6	.6	82.2
37.83	1	.6	.6	82.8
38.50	1	.6	.6	83.4
38.94	1	.6	.6	84.1
39.11	1	.6	.6	84.7
39.15	1	.6	.6	85.4
39.32	1	.6	.6	86.0
39.41	1	.6	.6	86.6
39.68	1	.6	.6	87.3
39.89	1	.6	.6	87.9
39.98	1	.6	.6	88.5
40.31	1	.6	.6	89.2
40.37	1	.6	.6	89.8
41.51	1	.6	.6	90.4
41.52	1	.6	.6	91.1
41.69	1	.6	.6	91.7
42.07	1	.6	.6	92.4
42.43	1	.6	.6	93.0
43.41	1	.6	.6	93.6
43.99	1	.6	.6	94.3

BMI

	Frequency	Percent	Valid Percent	Cumulative Percent
44.30	1	.6	.6	94.9
44.82	1	.6	.6	95.5
45.35	1	.6	.6	96.2
45.37	1	.6	.6	96.8
46.11	1	.6	.6	97.5
48.27	1	.6	.6	98.1
51.05	1	.6	.6	98.7
54.10	1	.6	.6	99.4
54.86	1	.6	.6	100.0
Total	157	100.0	100.0	

Frequencies

BMI category

		Frequency	Percent	Valid Percent	Cumulative Percent
Valid	Underweight	1	.6	.6	.6
	Normal weight	30	19.1	19.1	19.7
	Overweight	48	30.6	30.6	50.3
	Obese	60	38.2	38.2	88.5
	Morbidly obese	18	11.5	11.5	100.0
	Total	157	100.0	100.0	

Any depression

		Frequency	Percent	Valid Percent	Cumulative Percent
Valid	Not derpressed	118	75.2	75.6	75.6
	Any depression	38	24.2	24.4	100.0
	Total	156	99.4	100.0	
Missing	System	1	.6		
Total		157	100.0		

Frequencies

Statistics

		Edinburgh1	Edinburgh2	Edinburgh3
N	Valid	156	157	156
	Missing	1	0	1
Mean		34.60	44.46	76.58
Median		8.00	12.00	99.00
Std. Deviation		42.652	46.209	40.416
Percentiles	25	4.00	3.00	99.00
	50	8.00	12.00	99.00
	75	99.00	99.00	99.00

Edinburgh1

		Frequency	Percent	Valid Percent	Cumulative Percent
Valid	0	13	8.3	8.3	8.3
	1	7	4.5	4.5	12.8
	2	5	3.2	3.2	16.0
	3	11	7.0	7.1	23.1
	4	4	2.5	2.6	25.6
	5	6	3.8	3.8	29.5
	6	7	4.5	4.5	34.0
	7	10	6.4	6.4	40.4
	8	16	10.2	10.3	50.6
	9	2	1.3	1.3	51.9
	10	7	4.5	4.5	56.4
	11	1	.6	.6	57.1
	12	4	2.5	2.6	59.6
	13	3	1.9	1.9	61.5
	14	2	1.3	1.3	62.8
	15	1	.6	.6	63.5
	16	3	1.9	1.9	65.4
	17	3	1.9	1.9	67.3
	18	2	1.3	1.3	68.6
	22	2	1.3	1.3	69.9
	99	47	29.9	30.1	100.0
	Total	156	99.4	100.0	
Missing	System	1	.6		
Total		157	100.0		

Edinburgh2

		Frequency	Percent	Valid Percent	Cumulative Percent
Valid	0	13	8.3	8.3	8.3
	1	9	5.7	5.7	14.0
	2	10	6.4	6.4	20.4
	3	10	6.4	6.4	26.8
	4	4	2.5	2.5	29.3
	5	7	4.5	4.5	33.8
	6	8	5.1	5.1	38.9
	7	5	3.2	3.2	42.0
	8	4	2.5	2.5	44.6
	9	3	1.9	1.9	46.5
	10	2	1.3	1.3	47.8
	11	1	.6	.6	48.4
	12	3	1.9	1.9	50.3
	13	2	1.3	1.3	51.6
	14	3	1.9	1.9	53.5
	15	3	1.9	1.9	55.4
	17	1	.6	.6	56.1
	19	1	.6	.6	56.7
	24	1	.6	.6	57.3
	26	1	.6	.6	58.0
	28	1	.6	.6	58.6
	99	65	41.4	41.4	100.0
	Total	157	100.0	100.0	

Edinburgh3

		Frequency	Percent	Valid Percent	Cumulative Percent
Valid	0	10	6.4	6.4	6.4
	1	3	1.9	1.9	8.3
	2	3	1.9	1.9	10.3
	3	6	3.8	3.8	14.1
	4	3	1.9	1.9	16.0
	6	4	2.5	2.6	18.6
	7	1	.6	.6	19.2
	9	2	1.3	1.3	20.5
	10	1	.6	.6	21.2
	12	1	.6	.6	21.8
	14	1	.6	.6	22.4
	19	1	.6	.6	23.1
	23	1	.6	.6	23.7
	99	119	75.8	76.3	100.0
	Total	156	99.4	100.0	
Missing	System	1	.6		
Total		157	100.0		

Frequencies

Statistics

		Edinburgh1	Edinburgh2	Edinburgh3
N	Valid	28	28	28
	Missing	0	0	0
Mean		13.93	32.18	83.18
Median		13.00	12.50	99.00
Std. Deviation		3.548	39.813	34.736
Percentiles	25	10.25	6.25	99.00
	50	13.00	12.50	99.00
	75	16.75	80.75	99.00

Frequency Table

Edinburgh1

		Frequency	Percent	Valid Percent	Cumulative Percent
Valid	10	7	25.0	25.0	25.0
	11	1	3.6	3.6	28.6
	12	4	14.3	14.3	42.9
	13	3	10.7	10.7	53.6
	14	2	7.1	7.1	60.7
	15	1	3.6	3.6	64.3
	16	3	10.7	10.7	75.0
	17	3	10.7	10.7	85.7
	18	2	7.1	7.1	92.9
	22	2	7.1	7.1	100.0
	Total	28	100.0	100.0	

Edinburgh2

		Frequency	Percent	Valid Percent	Cumulative Percent
Valid	0	3	10.7	10.7	10.7
	2	2	7.1	7.1	17.9
	3	1	3.6	3.6	21.4
	6	1	3.6	3.6	25.0
	7	1	3.6	3.6	28.6
	8	2	7.1	7.1	35.7
	11	1	3.6	3.6	39.3
	12	3	10.7	10.7	50.0
	13	1	3.6	3.6	53.6
	14	2	7.1	7.1	60.7
	15	1	3.6	3.6	64.3
	19	1	3.6	3.6	67.9
	24	1	3.6	3.6	71.4
	26	1	3.6	3.6	75.0
	99	7	25.0	25.0	100.0
	Total	28	100.0	100.0	

Edinburgh3

		Frequency	Percent	Valid Percent	Cumulative Percent
Valid	0	1	3.6	3.6	3.6
	3	1	3.6	3.6	7.1
	12	1	3.6	3.6	10.7
	14	1	3.6	3.6	14.3
	23	1	3.6	3.6	17.9
	99	23	82.1	82.1	100.0
	Total	28	100.0	100.0	

Frequencies

Statistics

		Edinburgh1	Edinburgh2	Edinburgh3
N	Valid	19	19	19
	Missing	0	0	0
Mean		26.95	15.47	66.37
Median		16.00	14.00	99.00
Std. Deviation		32.482	5.221	44.066
Percentiles	25	8.00	12.00	12.00
	50	16.00	14.00	99.00
	75	22.00	17.00	99.00

Frequency Table

Edinburgh1

		Frequency	Percent	Valid Percent	Cumulative Percent
Valid	3	1	5.3	5.3	5.3
	5	1	5.3	5.3	10.5
	7	1	5.3	5.3	15.8
	8	2	10.5	10.5	26.3
	12	1	5.3	5.3	31.6
	14	2	10.5	10.5	42.1
	16	3	15.8	15.8	57.9
	17	2	10.5	10.5	68.4
	18	1	5.3	5.3	73.7
	22	2	10.5	10.5	84.2
	99	3	15.8	15.8	100.0
	Total	19	100.0	100.0	

Edinburgh2

		Frequency	Percent	Valid Percent	Cumulative Percent
Valid	10	2	10.5	10.5	10.5
	11	1	5.3	5.3	15.8
	12	3	15.8	15.8	31.6
	13	2	10.5	10.5	42.1
	14	3	15.8	15.8	57.9
	15	3	15.8	15.8	73.7
	17	1	5.3	5.3	78.9
	19	1	5.3	5.3	84.2
	24	1	5.3	5.3	89.5
	26	1	5.3	5.3	94.7
	28	1	5.3	5.3	100.0
	Total	19	100.0	100.0	

Edinburgh3

		Frequency	Percent	Valid Percent	Cumulative Percent
Valid	3	1	5.3	5.3	5.3
	6	2	10.5	10.5	15.8
	9	1	5.3	5.3	21.1
	12	1	5.3	5.3	26.3
	14	1	5.3	5.3	31.6
	23	1	5.3	5.3	36.8
	99	12	63.2	63.2	100.0
	Total	19	100.0	100.0	

Frequencies

Statistics

		Edinburgh1	Edinburgh2	Edinburgh3
N	Valid	5	5	5
	Missing	0	0	0
Mean		10.40	28.40	15.60
Median		12.00	14.00	14.00
Std. Deviation		5.771	39.954	5.320
Percentiles	25	4.50	7.00	11.00
	50	12.00	14.00	14.00
	75	15.50	57.00	21.00

Frequency Table

Edinburgh1

		Frequency	Percent	Valid Percent	Cumulative Percent
Valid	3	1	20.0	20.0	20.0
	6	1	20.0	20.0	40.0
	12	1	20.0	20.0	60.0
	14	1	20.0	20.0	80.0
	17	1	20.0	20.0	100.0
	Total	5	100.0	100.0	

Edinburgh2

		Frequency	Percent	Valid Percent	Cumulative Percent
Valid	0	1	20.0	20.0	20.0
	14	2	40.0	40.0	60.0
	15	1	20.0	20.0	80.0
	99	1	20.0	20.0	100.0
	Total	5	100.0	100.0	

Edinburgh3

		Frequency	Percent	Valid Percent	Cumulative Percent
Valid	10	1	20.0	20.0	20.0
	12	1	20.0	20.0	40.0
	14	1	20.0	20.0	60.0
	19	1	20.0	20.0	80.0
	23	1	20.0	20.0	100.0
	Total	5	100.0	100.0	

Frequencies

Statistics

		Edinburgh1	Edinburgh2	Edinburgh3
N	Valid	156	157	156
	Missing	1	0	1
Mean		34.60	44.46	76.58
Median		8.00	12.00	99.00
Std. Deviation		42.652	46.209	40.416
Percentiles	25	4.00	3.00	99.00
	50	8.00	12.00	99.00
	75	99.00	99.00	99.00

Frequency Table

Edinburgh1

		Frequency	Percent	Valid Percent	Cumulative Percent
Valid	0	13	8.3	8.3	8.3
	1	7	4.5	4.5	12.8
	2	5	3.2	3.2	16.0
	3	11	7.0	7.1	23.1
	4	4	2.5	2.6	25.6
	5	6	3.8	3.8	29.5
	6	7	4.5	4.5	34.0
	7	10	6.4	6.4	40.4
	8	16	10.2	10.3	50.6
	9	2	1.3	1.3	51.9
	10	7	4.5	4.5	56.4
	11	1	.6	.6	57.1
	12	4	2.5	2.6	59.6
	13	3	1.9	1.9	61.5
	14	2	1.3	1.3	62.8
	15	1	.6	.6	63.5
	16	3	1.9	1.9	65.4
	17	3	1.9	1.9	67.3
	18	2	1.3	1.3	68.6
	22	2	1.3	1.3	69.9
	99	47	29.9	30.1	100.0
	Total	156	99.4	100.0	
Missing	System	1	.6		
Total		157	100.0		

Edinburgh2

		Frequency	Percent	Valid Percent	Cumulative Percent
Valid	0	13	8.3	8.3	8.3
	1	9	5.7	5.7	14.0
	2	10	6.4	6.4	20.4
	3	10	6.4	6.4	26.8
	4	4	2.5	2.5	29.3
	5	7	4.5	4.5	33.8
	6	8	5.1	5.1	38.9
	7	5	3.2	3.2	42.0
	8	4	2.5	2.5	44.6
	9	3	1.9	1.9	46.5
	10	2	1.3	1.3	47.8
	11	1	.6	.6	48.4
	12	3	1.9	1.9	50.3
	13	2	1.3	1.3	51.6
	14	3	1.9	1.9	53.5
	15	3	1.9	1.9	55.4
	17	1	.6	.6	56.1
	19	1	.6	.6	56.7
	24	1	.6	.6	57.3
	26	1	.6	.6	58.0
	28	1	.6	.6	58.6
	99	65	41.4	41.4	100.0
	Total	157	100.0	100.0	

Edinburgh3

		Frequency	Percent	Valid Percent	Cumulative Percent
Valid	0	10	6.4	6.4	6.4
	1	3	1.9	1.9	8.3
	2	3	1.9	1.9	10.3
	3	6	3.8	3.8	14.1
	4	3	1.9	1.9	16.0
	6	4	2.5	2.6	18.6
	7	1	.6	.6	19.2
	9	2	1.3	1.3	20.5
	10	1	.6	.6	21.2
	12	1	.6	.6	21.8
	14	1	.6	.6	22.4
	19	1	.6	.6	23.1
	23	1	.6	.6	23.7
	99	119	75.8	76.3	100.0
	Total	156	99.4	100.0	
Missing	System	1	.6		
Total		157	100.0		

```
CROSSTABS
  /TABLES=bmicat BY anydepress
  /FORMAT=AVALUE TABLES
  /STATISTICS=CHISQ
  /CELLS=COUNT ROW COLUMN
  /COUNT ROUND CELL.
```

Crosstabs

Case Processing Summary

	Cases					
	Valid		Missing		Total	
	N	Percent	N	Percent	N	Percent
BMI category * Any depression	156	99.4%	1	0.6%	157	100.0%

BMI category * Any depression Crosstabulation

			Any depression		
			Not derpressed	Any depression	Total
BMI category	Underweight	Count	1	0	1
		% within BMI category	100.0%	0.0%	100.0%
		% within Any depression	0.8%	0.0%	0.6%
	Normal weight	Count	21	9	30
		% within BMI category	70.0%	30.0%	100.0%
		% within Any depression	17.8%	23.7%	19.2%
	Overweight	Count	34	14	48
		% within BMI category	70.8%	29.2%	100.0%
		% within Any depression	28.8%	36.8%	30.8%
	Obese	Count	51	8	59
		% within BMI category	86.4%	13.6%	100.0%
		% within Any depression	43.2%	21.1%	37.8%
	Morbidly obese	Count	11	7	18
		% within BMI category	61.1%	38.9%	100.0%
		% within Any depression	9.3%	18.4%	11.5%
Total		Count	118	38	156
		% within BMI category	75.6%	24.4%	100.0%
		% within Any depression	100.0%	100.0%	100.0%

Chi-Square Tests

	Value	df	Asymp. Sig. (2-sided)
Pearson Chi-Square	7.239[a]	4	.124
Likelihood Ratio	7.727	4	.102
Linear-by-Linear Association	.213	1	.645
N of Valid Cases	156		

a. 3 cells (30.0%) have expected count less than 5. The minimum expected count is .24.

Oneway

Means

Case Processing Summary

	Cases					
	Included		Excluded		Total	
	N	Percent	N	Percent	N	Percent
BMI * Any depression	156	99.4%	1	0.6%	157	100.0%

Report

BMI

Any depression	Mean	N	Std. Deviation	Median
Not derpressed	30.6614	118	6.66588	30.2300
Any depression	31.2989	38	9.07692	28.4200
Total	30.8167	156	7.29954	29.9450

ANOVA Table

			Sum of Squares	df	Mean Square
BMI * Any depression	Between Groups	(Combined)	11.682	1	11.682
	Within Groups		8247.216	154	53.553
	Total		8258.898	155	

ANOVA Table

			F	Sig.
BMI * Any depression	Between Groups	(Combined)	.218	.641
	Within Groups			
	Total			

Correlations

Correlations

		Edinburgh1	Edinburgh2	Edinburgh3	BMI
Edinburgh1	Pearson Correlation	1	.254**	.138	-.123
	Sig. (2-tailed)		.001	.085	.125
	N	156	156	156	156
Edinburgh2	Pearson Correlation	.254**	1	.204*	-.200*
	Sig. (2-tailed)	.001		.011	.012
	N	156	157	156	157
Edinburgh3	Pearson Correlation	.138	.204*	1	-.142
	Sig. (2-tailed)	.085	.011		.078
	N	156	156	156	156
BMI	Pearson Correlation	-.123	-.200*	-.142	1
	Sig. (2-tailed)	.125	.012	.078	
	N	156	157	156	157

**. Correlation is significant at the 0.01 level (2-tailed).

*. Correlation is significant at the 0.05 level (2-tailed).

Correlations

Correlations

		Any depression	BMI category
Any depression	Pearson Correlation	1	-.037
	Sig. (2-tailed)		.646
	N	156	156
BMI category	Pearson Correlation	-.037	1
	Sig. (2-tailed)	.646	
	N	156	157

Frequencies

No Edinburgh 1

		Frequency	Percent	Valid Percent	Cumulative Percent
Valid	Edinburgh 1 completed	109	69.4	69.4	69.4
	No Edinburgh 1	48	30.6	30.6	100.0
	Total	157	100.0	100.0	

Crosstabs

Case Processing Summary

	Cases					
	Valid		Missing		Total	
	N	Percent	N	Percent	N	Percent
BMI category * No Edinburgh 1	157	100.0%	0	0.0%	157	100.0%

BMI category * No Edinburgh 1 Crosstabulation

			No Edinburgh 1		
			Edinburgh 1 completed	No Edinburgh 1	Total
BMI category	Underweight	Count	1	0	1
		% within BMI category	100.0%	0.0%	100.0%
		% within No Edinburgh 1	0.9%	0.0%	0.6%
	Normal weight	Count	21	9	30
		% within BMI category	70.0%	30.0%	100.0%
		% within No Edinburgh 1	19.3%	18.8%	19.1%
	Overweight	Count	27	21	48
		% within BMI category	56.3%	43.8%	100.0%
		% within No Edinburgh 1	24.8%	43.8%	30.6%
	Obese	Count	44	16	60
		% within BMI category	73.3%	26.7%	100.0%
		% within No Edinburgh 1	40.4%	33.3%	38.2%
	Morbidly obese	Count	16	2	18
		% within BMI category	88.9%	11.1%	100.0%
		% within No Edinburgh 1	14.7%	4.2%	11.5%
Total		Count	109	48	157
		% within BMI category	69.4%	30.6%	100.0%
		% within No Edinburgh 1	100.0%	100.0%	100.0%

Chi-Square Tests

	Value	df	Asymp. Sig. (2-sided)
Pearson Chi-Square	8.015[a]	4	.091
Likelihood Ratio	8.722	4	.068
Linear-by-Linear Association	2.456	1	.117
N of Valid Cases	157		

a. 2 cells (20.0%) have expected count less than 5. The minimum expected count is .31.

Frequencies

No Edinburgh 2

		Frequency	Percent	Valid Percent	Cumulative Percent
Valid	Edinburgh 2 completed	92	58.6	58.6	58.6
	Edinburgh 2 not cvmpleted	65	41.4	41.4	100.0
	Total	157	100.0	100.0	

Crosstabs

Case Processing Summary

	Cases					
	Valid		Missing		Total	
	N	Percent	N	Percent	N	Percent
BMI category * No Edinburgh 2	157	100.0%	0	0.0%	157	100.0%

BMI category * No Edinburgh 2 Crosstabulation

			No Edinburgh 2		
			Edinburgh 2 completed	Edinburgh 2 not cvmpleted	Total
BMI category	Underweight	Count	1	0	1
		% within BMI category	100.0%	0.0%	100.0%
		% within No Edinburgh 2	1.1%	0.0%	0.6%
	Normal weight	Count	12	18	30
		% within BMI category	40.0%	60.0%	100.0%
		% within No Edinburgh 2	13.0%	27.7%	19.1%
	Overweight	Count	27	21	48
		% within BMI category	56.3%	43.8%	100.0%
		% within No Edinburgh 2	29.3%	32.3%	30.6%
	Obese	Count	36	24	60
		% within BMI category	60.0%	40.0%	100.0%
		% within No Edinburgh 2	39.1%	36.9%	38.2%
	Morbidly obese	Count	16	2	18
		% within BMI category	88.9%	11.1%	100.0%
		% within No Edinburgh 2	17.4%	3.1%	11.5%
Total		Count	92	65	157
		% within BMI category	58.6%	41.4%	100.0%
		% within No Edinburgh 2	100.0%	100.0%	100.0%

Chi-Square Tests

	Value	df	Asymp. Sig. (2-sided)
Pearson Chi-Square	11.949[a]	4	.018
Likelihood Ratio	13.492	4	.009
Linear-by-Linear Association	7.967	1	.005
N of Valid Cases	157		

a. 2 cells (20.0%) have expected count less than 5. The minimum expected count is .41.

Frequencies

No Edinburgh 3

		Frequency	Percent	Valid Percent	Cumulative Percent
Valid	Edinburgh 3 completed	37	23.6	23.6	23.6
	Edinburgh 3 not completed	120	76.4	76.4	100.0
	Total	157	100.0	100.0	

Crosstabs

Case Processing Summary

	Cases					
	Valid		Missing		Total	
	N	Percent	N	Percent	N	Percent
BMI category * No Edinburgh 3	157	100.0%	0	0.0%	157	100.0%

BMI category * No Edinburgh 3 Crosstabulation

			No Edinburgh 3		
			Edinburgh 3 completed	Edinburgh 3 not completed	Total
BMI category	Underweight	Count	0	1	1
		% within BMI category	0.0%	100.0%	100.0%
		% within No Edinburgh 3	0.0%	0.8%	0.6%
	Normal weight	Count	7	23	30
		% within BMI category	23.3%	76.7%	100.0%
		% within No Edinburgh 3	18.9%	19.2%	19.1%
	Overweight	Count	9	39	48
		% within BMI category	18.8%	81.3%	100.0%
		% within No Edinburgh 3	24.3%	32.5%	30.6%
	Obese	Count	14	46	60
		% within BMI category	23.3%	76.7%	100.0%
		% within No Edinburgh 3	37.8%	38.3%	38.2%
	Morbidly obese	Count	7	11	18
		% within BMI category	38.9%	61.1%	100.0%
		% within No Edinburgh 3	18.9%	9.2%	11.5%
Total		Count	37	120	157
		% within BMI category	23.6%	76.4%	100.0%
		% within No Edinburgh 3	100.0%	100.0%	100.0%

Chi-Square Tests

	Value	df	Asymp. Sig. (2-sided)
Pearson Chi-Square	3.275[a]	4	.513
Likelihood Ratio	3.282	4	.512
Linear-by-Linear Association	1.380	1	.240
N of Valid Cases	157		

a. 3 cells (30.0%) have expected count less than 5. The minimum expected count is .24.

```
RECODE bmicat (SYSMIS=1) (5=1) (1 thru 4=0) INTO morbid.
VARIABLE LABELS  morbid 'Morbid obesity'.
EXECUTE.
CROSSTABS
  /TABLES=morbid BY anydepress
  /FORMAT=AVALUE TABLES
  /STATISTICS=CHISQ
  /CELLS=COUNT ROW COLUMN
  /COUNT ROUND CELL.
```

Crosstabs

Case Processing Summary

	Cases					
	Valid		Missing		Total	
	N	Percent	N	Percent	N	Percent
Morbid obesity * Any depression	156	99.4%	1	0.6%	157	100.0%

Morbid obesity * Any depression Crosstabulation

			Any depression	
			Not derpressed	Any depression
Morbid obesity	Not morbidly obese	Count	107	31
		% within Morbid obesity	77.5%	22.5%
		% within Any depression	90.7%	81.6%
	Morbidly obese	Count	11	7
		% within Morbid obesity	61.1%	38.9%
		% within Any depression	9.3%	18.4%
Total		Count	118	38
		% within Morbid obesity	75.6%	24.4%
		% within Any depression	100.0%	100.0%

Morbid obesity * Any depression Crosstabulation

			Total
Morbid obesity	Not morbidly obese	Count	138
		% within Morbid obesity	100.0%
		% within Any depression	88.5%
	Morbidly obese	Count	18
		% within Morbid obesity	100.0%
		% within Any depression	11.5%
Total		Count	156
		% within Morbid obesity	100.0%
		% within Any depression	100.0%

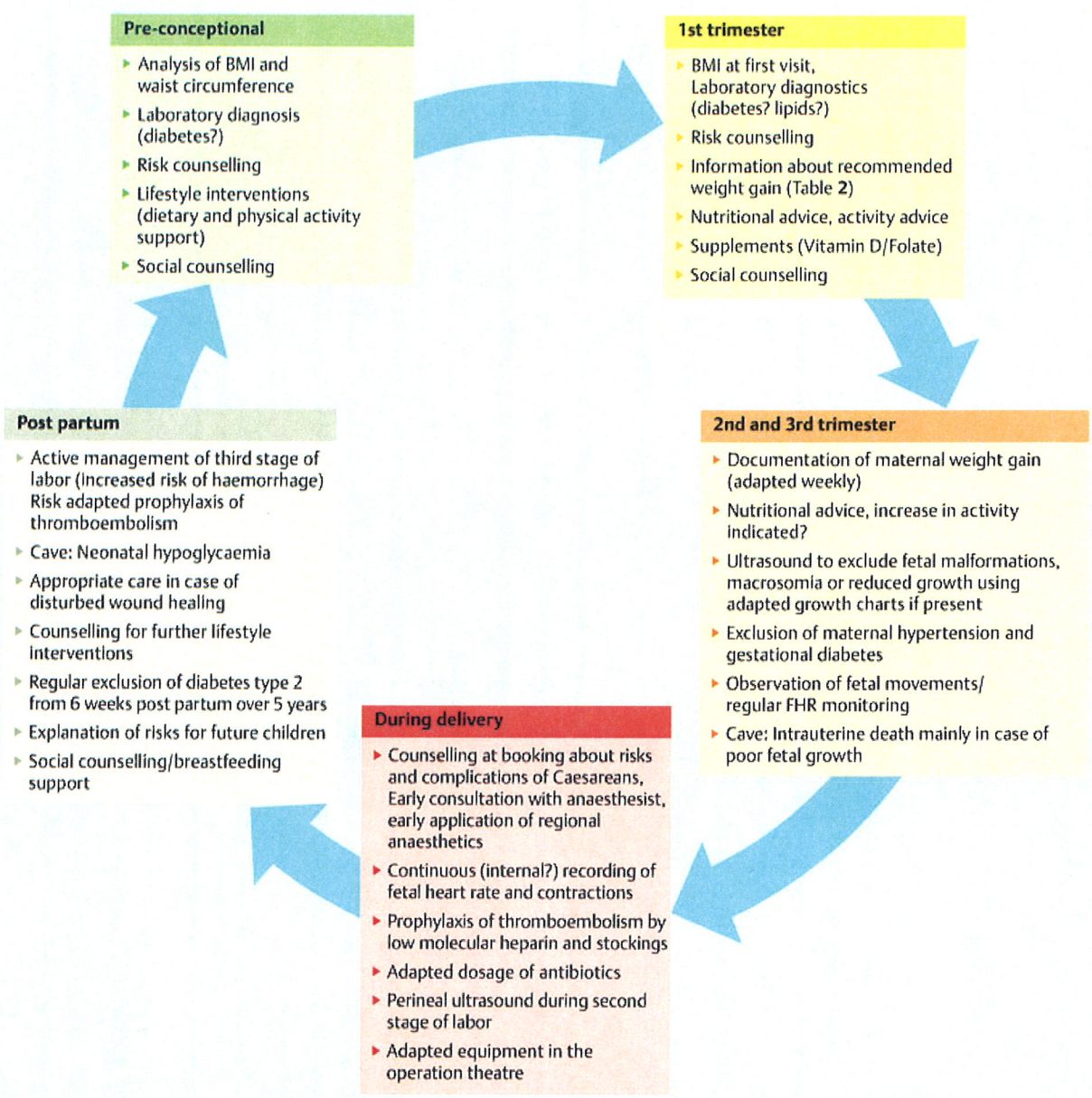
Chi-Square Tests

	Value	df	Asymp. Sig. (2-sided)	Exact Sig. (2-sided)	Exact Sig. (1-sided)
Pearson Chi-Square	2.331[a]	1	.127		
Continuity Correction[b]	1.525	1	.217		
Likelihood Ratio	2.131	1	.144		
Fisher's Exact Test				.147	.111
Linear-by-Linear Association	2.317	1	.128		
N of Valid Cases	156				

a. 1 cells (25.0%) have expected count less than 5. The minimum expected count is 4.38.

b. Computed only for a 2x2 table

Appendix G



Pragmatic flow chart of treatment of overweight and obese women pre-, peri- and postnatally.

[Arabin et al., 2014]

Appendix H

Weight classification/WHO, with modified criteria of the Canadian guidelines and recommended weight gain according to the Institute of Medicine (IOM), commented by Rasmussen.

Criteria	**BMI (kg/m^2)**	**Recommended weight gain**		
		Course of pregnancy		**2nd and 3rd trimester**
		Singleton pregnancy	**Twin pregnancy**	**Singleton pregnancy**
Underweight	< 18,5	12.5–18 kg	no information	0.51 (0.44–0.58) kg/gestational week
Normal weight	18.5–24.9	11.5–16 kg	17–25 kg	0.42 (0.35–0.50) kg/gestational week
Overweight	25–29.9	7–11.5 kg	14–23 kg	0.28 (0.23–0.33) kg/gestational week
Obesity class I	30–34.9	5–9 kg	11–19 kg	0.22 (0.17–0.27) kg/gestational week
Obesity class II	35–39.9	5–9 kg	11–19 kg	0.22 (0.17–0.27) kg/gestational week
Obesity class III	> 40	5–9 kg	11–19 kg	0.22 (0.17–0.27) kg/gestational week

[Arabin et al., 2014]

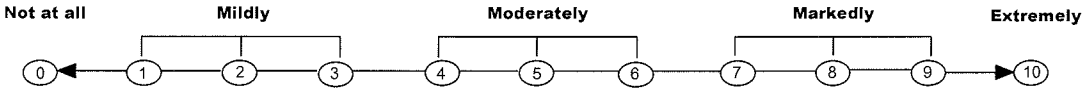
SHEEHAN DISABILITY SCALE

A BRIEF, PATIENT RATED, MEASURE OF DISABILITY AND IMPAIRMENT

Please mark ONE circle for each scale.

WORK* / SCHOOL

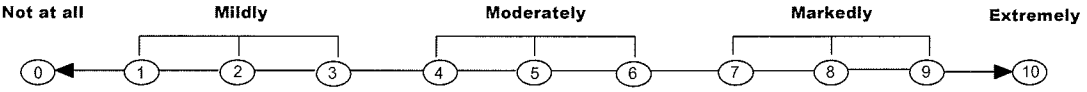
The symptoms have disrupted your work / school work:



☐ I have not worked /studied at all during the past week for reasons unrelated to the disorder.
* Work includes paid, unpaid volunteer work or training

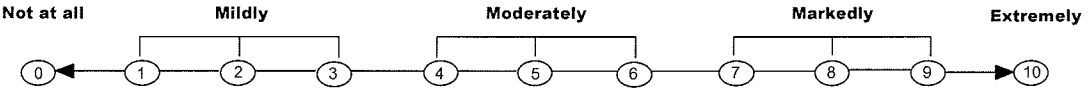
SOCIAL LIFE

The symptoms have disrupted your social life / leisure activities:



FAMILY LIFE / HOME RESPONSIBILITIES

The symptoms have disrupted your family life / home responsibilities:



DAYS LOST

On how many days in the last week did your symptoms cause you to miss school or work or leave you unable to carry out your normal daily responsibilities? _____

DAYS UNDERPRODUCTIVE

On how many days in the last week did you feel so impaired by your symptoms, that even though you went to school or work, your productivity was reduced? _____

Appendix J

Physiological and pathophysiological effects of selected adipokines.

TNF-alpha	Inflammation, apoptosis, impact on insulin resistance, stimulation of endothelial dysfunction and atherogenesis
IL-6	Inflammation, immune regulation (modulation of the insulin receptor), insulin resistance, atherogenesis
Adiponectin	Stimulation of insulin secretion, increase in insulin sensitivity, stimulation of glucose uptake in the muscle, inflammation reduction, plasma lipid reduction, atheroprotective effect
Leptin	Saturation, increase in energy utilization, weight control, modulation of insulin sensitivity, reduction of insulin secretion, angiogenesis

[Stupin et al., 2014]

SEED GRANT: DATA ABSTRACTION FORM

Study Record ID: ___ ___ ___

Maternal Demographics

Age	
Race	
Ethnicity	

Birth Date:_____

Foreign Born: Yes ☐ No ☐

Marital Status: Yes ☐ No ☐

Employment Status: Yes ☐ No ☐

Insurance: Private ☐ Public ☐ Other_____

Public Assistance: Yes ☐ No ☐

If yes please specify (TANF, WIC, SNAP, Housing Assistance etc...)_____

Paternal/Partner Demographics

Age	
Race	
Ethnicity	

Foreign Born: : Yes ☐ No ☐

Marital Status: Yes ☐ No ☐

Employment Status: Yes ☐ No ☐

Insurance: Private ☐ Public ☐ Other_____

Public Assistance: Yes ☐ No ☐

If yes please specify (TANF, WIC, SNAP, Housing Assistance etc...)_____

Pertinent Medical HX:

Body Systems	Condition(s)	Medication(s)	Present During Pregnancy Y/N
HEENT			
Respiratory			
Cardiovascular			
GI/Hepatic			
Urinary/Reproductive			
Endocrine/Metabolic			
Neurological			
Muscoskeletal			
Psychiatric/Illness			
Immune and Inflammatory			
Infectious Disease			
Substance Use			
Chlamydia /Gonorrhea/GBS			

Pregnancy

Height: _____ **Weight:**_____

BMI (if available): _____

Early Prenatal Care (1st trimester): Yes ☐ No ☐
# of prenatal visits_____

Prior Perinatal Depression HX: Yes ☐ No ☐
If yes, Edinburgh Score:_____

Prior Perinatal MH Referral: Yes ☐ No ☐

Prenatal Depression DX: Yes ☐ No ☐

If Yes, was referral to additional mental services provided by clinic staff: Yes ☐ No ☐

Receipt of Referral: Yes ☐ No ☐ Pt Refused ☐

If No, please specify reason for denied referral:_____

Receiving SSRI Treatment for Depression during Pregnancy: Yes ☐ No ☐

If Yes, did patient maintain treatment plan through duration of pregnancy:
Yes ☐ No ☐

Edinburgh Depression Scores :
1st _____ 2nd(28W)_____ 3rd(PP)_____

ED Admissions #___ **Domestic Violence:** Yes ☐ No ☐

Referrals:

Social Work	Y	N	Refused
School Based Program	Y	N	Refused
Substance Abuse	Y	N	Refused
Counseling	Y	N	Refused
Genetic Counseling	Y	N	Refused
Dietician	Y	N	Refused
WIC	Y	N	Refused
Other			

Comments:

Study Record # _____

Birth

Weeks Gestation at Birth:___ _____

Birth: Vaginal ☐ VBAC ☐ Cesarean ☐

Birth Weight :_____g LBW ☐ (2500g) VLBW ☐ (1500g)

Previous Number of live births #_____

Prior neonatal death: Yes ☐ No ☐

Prior fetal death: Yes ☐ No ☐

Circle Any Prior TOP/SAB #___ Trimester_____

Support Partner at Birth : Yes ☐ No ☐

If yes, was this individual the biological/ co-parent
Yes ☐ No ☐

Intendedness of Pregnancy : Yes ☐ No ☐

Infant

APGAR Scores: 1minute_____ 5minute_____

10 min_____ Additional Scores_____

Feeding Style: Breast ☐ Bottle ☐ Both ☐

NICU Referral: Yes ☐ No ☐

Healthy Newborn: Yes ☐ No ☐

Congenital/Birth Defect DX: Yes ☐ No ☐

D/C Status: Home w/baby ☐ Still in Nursery ☐
Transferred ☐ Neonatal Death ☐ Adopted Out ☐

Notes:

SEED Grant Maternal Depression Project

Data Dictionary

Variable	**Coding**	
Age	Continuous	
Race	White/Caucasian	1
	African American	2
	Hispanic	3
	Asian	4
	Native American	5
	Other	6
Maternal Employment	Unemployed	0
	Unemployed	1
	Unknown	2
Maternal Marital Status	Single	0
	Married	1
	Unknown	2
Insurance	Private	0
	Public	1
	Unknown	2
Foreign Born	Native (No)	0
	Foreign Born (yes)	1
	Unknown	2
Paternal Employment Status	Unemployed	0
	Employed	1
	Unknown	2
Paternal Marital Status	Single	0
	Married	1
	Unknown	2
Early Care (≤1st tri)	No	0
	Yes	1
	Unknown	2
H/O Abuse	No	0
	Yes	1
	Unknown	2
H/O Mental Health	No	0
	Yes	1
	Unknown	2
STI or Infection During Pregnancy	No	0
	Yes	1
	Unknown	2
#Prenatal Visits	Continuous	#
Prior Perinatal Depression Diagnosis	No	0
	Yes	1
	Unknown	2
SSRI Treatment During Pregnancy	No	0

SEED Grant Maternal Depression Project

Data Dictionary

	Yes	1
	Unknown	2
Edinburgh1 (continuous)	99	Not done
Edinburgh2 (continuous)	99	Not done
Edinburgh3 (continuous)	99	Not done
Social Work Referral	No	0
	Yes	1
	Unknown	2
	Refused	3
Public Health Nurse Referral	No	0
	Yes	1
	Unknown	2
	Refused	3
Dietician Referral	No	0
	Yes	1
	Unknown	2
	Refused	3
WIC Referral	No	0
	Yes	1
	Unknown	2
Dental Referral	No	0
	Yes	1
	Unknown	2
	Refused	3
#Weeks Gestation	Continuous	
Birth Method	Vaginal	1
	VBAC	2
	Cesarean	3
	Unknown	4
Birth Weight (grams)	Continuous	
#Previous Live Births	Continuous	
#Neonatal Deaths	Continuous	
#Fetal Deaths	Continuous	
# TOP	Continuous	
#SAB	Continuous	
Intendedness of Pregnancy	No	0
	Yes	1
	Continuous	2
#ER Admissions During Pregnancy	Continuous	
1 minute APGAR Score (0-10)	Continuous	
5 minute APGAR Score (0-10)	Continuous	
Feeding Style After Birth	Breast	1
	Bottle	2

SEED Grant Maternal Depression Project

Data Dictionary

	Both	3
NICU Referral	0	No
	1	Yes
	2	Unknown
Discharge Status	Home w/baby	1
	Infant still in NICU	2
	Infant Transferred	3
	Adopted Out	4
	Unknown	5
	Neonatal Death	6