

Effects of Prenatal Stress and Poverty on Fetal Growth

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Dedication

This work is dedicated to all the women in Tennessee who have suffered stress disproportionately due to their standing in society and to their babies who deserve the love and nurture required to develop to their full potential.

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Abstract

Background. Prenatal stress has negative effects on the developing fetus through the activation of the hypothalamic-pituitary-adrenal (HPA) axis. Programming of the stress response system during gestation has lifelong effects that put the infant at risk for multiple stress-related pathologies. Populations most vulnerable to prenatal stress are African-Americans and individuals of low socioeconomic status.

Methods. The Pregnancy Risk Assessment Monitoring System (PRAMS) research project, a collaboration between the Centers for Disease Control and Prevention and individual state health departments, was utilized for this study. Tennessee data from 2009 were compiled from individual birth certificates and PRAMS questionnaire responses to examine three constructs: fetal development, stressful life events, and poverty in order to examine the influence of maternal stressors and poverty on fetal development.

Results. Latent class analysis revealed two classes of mothers with quantitative and qualitative differences in stressful life events, but class membership was not a significant predictor of problematic birth outcomes. The number of stressors was only a significant predictor of having an infant small for gestational age when moderated by Medicaid status. Medicaid status proved to be a significant predictor of all four measures of fetal growth. The relationship between race and problematic birth overall was moderated by age, with young African-American mothers less likely than European-Americans and older African-American mothers to have problematic births.

Conclusion. Stressors, as measured in the field of social work through life events and daily hassles, could potentially be inadequate measures. Further examination of prenatal stress measures is needed.

Keywords: stress, prenatal stress, poverty, fetal growth, birth outcomes

Table of Contents

CHAPTER 1: LITERATURE REVIEW	1
Background	1
Stress: Social Work's Understanding	1
What is Stress?	3
Measuring Stress	4
Fight or Flight Response	5
The Hormonal Stress Response	6
Hypothalamic-Pituitary-Adrenal (HPA) Axis	6
Prenatal Stress	8
Stress Hormones during Pregnancy	8
Transmission of Maternal Stress to Fetus	9
Fetal Programming.....	10
Prenatal Stress and Brain Neurotransmitter Systems.....	12
Negative Outcomes Associated with Prenatal Stress.....	14
Effects on the Brain	14
Neurodevelopmental Effects.....	17
Prenatal Stress and Physiological Disorders in Adulthood.....	18
Differences in Stress Reactivity.....	19
Whom does Prenatal Stress Affect?.....	20
Socioeconomic Status	20
Marital Status	21
Maternal Education.....	22
Maternal Race	23
Maternal Age	23
Future Implications & Social Work's Role	24
CHAPTER 2: METHODOLOGY	26
Methodology	26
Design	26
Data Sources	27
Variables Used in the Present Study	28
CHAPTER 3: RESULTS	32
Results.....	32
Sample.....	32
Maternal Stressor Typology.....	36
Birth Outcomes and Demographic Variables and Medical Risk	39
Birth Outcomes and Stressors.....	41
CHAPTER 4: DISCUSSION.....	43
Summary of Results.....	43
Sample and Design	43
Stress	50
Poverty	53
Discussion.....	54

Implications.....	56
For Practice	56
For Policy.....	57
For Research	58
References	60
Appendix.....	88
Appendix A: PRAMS Questionnaire.....	107
Appendix B: PRAMS Sampling Frame.....	124
Appendix C: PRAMS Non-Response Methodology	125
Appendix D: Example of U.S. Birth Certificate	126
Appendix E: IRB Exemption Form A	128
Vita.....	129

List of Tables

Table 1. Demographic Characteristics of Sample Population	88
Table 2. Percentages Experiencing Individual Stressors	89
Table 3. Birth Outcomes for Sample Population.....	90
Table 4. Latent Class Analysis Models.....	91
Table 5. Predictors of Overall Problematic Births.....	92
Table 6. Predictors of Preterm Birth.....	93
Table 7. Predictors of Large for Gestational Age.....	94
Table 8. Predictors of Small for Gestational Age.....	95
Table 9. Bivariate Predictors of Birth Outcomes.....	96

List of Figures

Figure 1. Number of Stressors Experienced.....	97
Figure 2. Gestational Age in Weeks.....	98
Figure 3. Unadjusted Birthweights in Grams.....	99
Figure 4. Adjusted Birthweight Z-Scores.....	100
Figure 5. Adjusted Birthweight Percentiles.....	101
Figure 6. 5-Minute APGAR Scores.....	102
Figure 7. Proportion of Stressors Experienced by Class.....	103
Figure 8. Interaction between Age and Problematic Birth by Race.....	104
Figure 9. Interaction between Number of Stressors and SGA by Medicaid Status.....	105

Chapter 1: Literature Review

Background

The issue of prenatal stress is a particularly problematic one because of its negative effects on the developing fetus. Through a series of biological mechanisms, the fetus responds to the mother's stress by adapting physiologically and putting him/her at risk for multiple stress-related pathologies. Pregnancy itself as a major life event can cause anxiety and stress for mothers during instances of unplanned pregnancy, testing/diagnosis of medical complications, and in thinking of and planning for childbirth (Geller, 2004). When the stress of pregnancy is compounded with food insecurity, substandard housing, violence, and other environmental factors associated with poverty the developing fetus becomes increasingly susceptible to stress hormones, thus putting him/her at risk for long-term negative effects. The populations most at risk for experiencing prenatal stress are the same vulnerable populations the field of social work commonly advocates for. Our code of ethics and mission in the field is to "enhance human well-being and help meet the basic needs of all people, with particular attention to the needs and empowerment of people who are vulnerable, oppressed, and living in poverty (NASW, 2008)." Therefore, the social work field has an obligation to recognize the consequences of prenatal stress and advocate for those mothers and infants at risk for experiencing chronic stress during pregnancy.

Stress: Social Work's Understanding

The field of social work has only indirectly explored the relationship between prenatal stress and its associated negative birth outcomes. Social workers have always worked for the disadvantaged and have long understood that environmental stressors are particularly bad for those living in poverty. The field has consistently recognized the social injustice of health

inequality and acknowledged that poverty is the primary factor leading to the well-documented disparities (Hernandez, Montana, & Clarke, 2010). The concept of an allostatic load, or the cumulative effects of chronic stress, associated with living in poverty has been established (Johner, 2007) and its effects on health have been recognized (Gorin, 2000).

The social work field has historically been particularly aware of these effects on women and children. The Children's Bureau, established in 1912, through the work of leaders in the settlement house movement such as Florence Kelley, Julia Lanthrop, Grace and Edith Abbott, and Jane Addams (Combs-Orme, 1988), was charged with the task of investigating and reporting on all matters related to the welfare of children and child life (Bradbury & Eliot, 1956). Between 1912 and 1921, the Children's Bureau published 46 documents on child and maternal health in the United States focusing on both macro-level interventions, such as state and federal infant health policies, and micro-level interventions, such as visiting nurse programs that advocated for prenatal care (Pierce, 2004). The Bureau's research, conducted by early social workers, was particularly focused on infant mortality (Combs-Orme, 1988). As a result, the Sheppard-Towner Maternity and Infancy Act of 1921 was passed, conducting almost 145,000 health conferences where children and mothers were given medical care, establishing approximately 3,000 centers for prenatal care, and conducting close to 20,000 infant and maternal care classes (U.S. Children's Bureau, 1931). Following the Act's implementation, the U.S. infant mortality rate fell from 76 per 1,000 live births in 1921 to 65 per 1,000 in 1927 (Combs-Orme, 1988). Since then, the field of social work has identified the disproportionate rate of infant mortality among racial minorities (Berger, 2001; Combs-Orme, 1987) and acknowledged the importance of prenatal care (Loveland Cook, Selig, Wedge, & Gohn-Baube, 1999; Combs-Orme, 1990). Furthermore,

social workers have been actively involved in public health settings that offer prenatal care (Kerson & McCoyd, 2010).

While the field of social work has not identified prenatal stress as a major professional concern, it has been involved in clinical services that deal with prenatal care and its associated stressors as well as programs that serve children dealing with the negative outcomes related to prenatal stress. The field has acknowledged that stress hormones influence individual biology and disease (Gehlert, Mininger, Sohmer, & Berg, 2008), including infant health and development (Combs-Orme & Cain, 2006). However, social work has yet to articulate how interventions might prevent or treat such problems.

What is Stress?

The survival of living organisms depends on homeostasis, the balance of complex systems to support physical and psychological functioning (Weinstock, 2001). Stress, then, is most commonly conceptualized as a perceived threat to an organism's homeostasis (the maintenance of a stable environment) and as a situation that causes increases in autonomic nervous system activity and hormone secretion (McEwen & Sapolsky, 1995). Social work has typically characterized stressors as psychosocial events that cause a physiological stress response (Gorin, 2000). It is the physiological stress response rather than the stressor itself that has been shaped by natural selection and which functions to increase the ability of organisms to cope with situations that require action or defense (Neese, Bhatnagar, & Young, 2007). Thus, the stress response is an adaptive process that promotes an individual's survival.

Measuring Stress

The term “stress” is full of ambiguities in that it is often used to describe either a distressing event or the response to that event (McEwen & Wingfield, 2003). This debate over the “stimulus” and/or perception of the stimulus or “response” perspective continues and no universally accepted characterization of the term exists (Dohrenwend, 2006). Given the differing views as to its nature, a variety of measures exist, thus leading to a problem in accurately measuring stress (McQuaid et al., 1992). The most common approach to assessing stress has been through self-report checklists of traumatic life events and daily hassles (Monroe, 2008). However, psychological stress research in human pregnancies does not account for systematic events applied to randomly selected individuals but rather reflects women’s appraisal of their daily lives through the lens of their mood states and personality inclinations (DiPietro, 2012). For this reason the study of prenatal stress suffers from a methodological challenge in attempting to isolate the effects of a stressor from other confounding influences (DiPietro, Costigan, & Sipsma, 2008). Therefore, measuring stress suffers from a great amount of error (Dohrenwend, 2006) and has proven to be unreliable (Monroe, 2008).

A systematic review of psychometric instruments to assess psychosocial stress during pregnancy examined 115 publications published between 1999 and 2009. This review identified 43 various instruments being used to measure 7 assorted constructs (*e.g.*, anxiety, depression, daily hassles, aspects of psychological symptomatology, life events, specific socio-environmental stressors, and stress related to pregnancy and parenting). Findings across the studies were inconsistent and only a few assessed all potential aspects of stress (*i.e.*, the stressor itself, the individual perception of stress, and individual reactions to stress) (Nast, Bolten,

Meinlschmidt, & Hellhammer, 2013). Applying multiple methods and obtaining varying results impedes comparability of prenatal stress research and hinders interpretability.

Because stress is a physiological response that originates in the brain, paper and pencil methods are only able to measure the occurrence of stressful stimuli or the perception of that event but not the stress response itself. Therefore, it is important to understand the biological mechanisms at work.

Fight or Flight Response

When an organism is confronted with a threat to its homeostasis, such as the sudden appearance of a tiger or a car in front of him/her slamming on its brakes, the limbic system of the brain reacts by initiating the autonomic nervous system (ANS), priming it for a fight or flight response (Latendresse, 2009). This begins the alarm phase in which the sympathetic branch of the ANS increases arousal, blood pressure, heart rate, respiratory rate, and physical activity through the release of norepinephrine and epinephrine (or *adrenaline*, the American term for *epinephrine*) (McEwen & Wingfield, 2003) while stressors reduce parasympathetic activity by inhibiting muscular growth, storing energy, and shunting blood to digestion and bodily repair (Neese, Bahtnagar, & Young, 2007). In essence, the sympathetic branch increases everything one needs to get away while the parasympathetic branch shuts down everything that is not needed so that all energy can be placed on survival. During this time, the anterior pituitary also secretes a neurotransmitter (β -endorphin), a natural analgesic, into the blood stream that numbs or dulls the body's reaction to pain (Chrousos, 1998). Thus, β -endorphin prepares the body to fight the tiger or survive the impact of a car without going into shock.

The Hormonal Stress Response

Stress triggers the activation of the hypothalamic-pituitary-adrenal (HPA) axis, which is the fundamental neuroendocrine stress response.

Hypothalamic-Pituitary-Adrenal (HPA) Axis

The HPA axis is the primary neuroendocrine system that controls the body's reactions to stress. The axis operates on a feedback loop involving the hypothalamus, the anterior pituitary, and the adrenal glands. Cells in the hypothalamus produce corticotropin-releasing hormone (CRH) in response to a stressor. CRH then binds to receptors in the anterior pituitary, which produces adrenocorticotrophic hormone (ACTH). It is here, after the fight or flight response has been initiated, that β -endorphin is released to help continue the fight for survival and blunt the body's reaction to pain. ACTH is then transported to the adrenal glands where adrenal hormones, such as cortisol, are secreted. The activation of the sympathetic nervous system then stimulates the release of adrenaline (Chrousos, 1998).

Feedback regulation and allostasis. The HPA axis operates under a negative feedback loop so that after the adrenal glands secrete cortisol it binds with receptors in the hypothalamus and anterior pituitary to inhibit secretion of CRH and ACTH, respectively (Weinstock, 1997). This negative feedback system brings the body back into hormonal homeostasis and is an example of allostasis, which is the effort the body exerts to maintain normal functioning. For example, a high body temperature initiates sweating to cool the body down, high blood sugar levels initiate extra production of insulin to lower blood sugar levels, and low blood pressure initiates an increased heart rate to improve blood flow. Because the body attempts to combat the threat and return to normal functioning, this is why after we slam on the car brakes and avoid a collision we are quickly able to return to listening to our radio program and continue driving.

Allostasis is thus a protective process in the short run, but when drawn out for longer periods of time becomes a harmful allostatic load that has deleterious effects on the body (McEwen & Winfield, 2003).

Allostatic load and glucocorticoid receptors. Allostatic load refers to the cumulative effects of “wear and tear” on the body over a period of exposure to the processes of allostasis (Latendresse, 2009). Continuous time spent in this state can lead to serious pathophysiology (McEwen, 2000). For example, activation of the stress system stimulates arousal and suppresses sleep with over-activation sometimes resulting in insomnia. Stress also inhibits the gastrointestinal system, which can easily lead to constipation (Chrousos, 2009). Additionally, glucocorticosteroids, the body’s stress hormones, promote the conversion of protein and lipids to usable carbohydrates so that the body’s energy reserves will be replenished after a period of activity such as running away from that tiger (Krysiak et al., 2001). This is useful during times of strenuous cardiovascular activity but not beneficial during sedentary periods. Inactivity and lack of energy expenditure combined with a decreased metabolism create a situation where chronically elevated glucocorticosteroids can impede the action of insulin to promote glucose uptake, leading to high insulin levels. Together, high insulin levels and elevated glucocorticosteroids promote the deposition of body fat leading to problems of obesity, diabetes, and coronary heart disease (McEwen & Wingfield, 2003). Furthermore, a chronic allostatic load leads to a dysregulation of the HPA axis through a desensitization of glucocorticoid receptors. Glucocorticoid receptors are widely distributed throughout the brain, allowing for glucocorticoids to bind to them and initiate the negative feedback loop by inhibiting the synthesis and release of CRH (Meany, 2010). Repeated stress in which the organism is unable to return to homeostasis decreases the number of glucocorticoid receptors and results in a dysregulation of

the stress response and chronically elevated circulating stress hormones (Jacobsen & Sapolsky, 1991).

Prenatal Stress

During pregnancy, the mother's HPA axis functions normally while the fetus is developing its own. However, the effects of stressors during pregnancy can be compounded through the release of placental CRH.

Stress Hormones during Pregnancy

From eight to ten weeks' gestation, CRH is also produced by the placenta and secreted both to the mother and fetus. Cortisol stimulates placental CRH (pCRH) rather than inhibiting it like it does in the adult hypothalamus and anterior pituitary (Latendresse, 2009). Later in pregnancy pCRH, entering through the umbilical vein, also stimulates the fetal HPA axis to produce hormones, resulting in a large increase in cortisol (Majzoub & Karalis, 1999). This increase guarantees that all organs will be fully developed upon delivery. However, if pCRH enters the fetal compartment too early it can result in premature labor and delivery (Mulder et al., 2001). Under normal circumstances, a CRH-binding protein (CRH-BP) inactivates pCRH, except for during the last 2-4 weeks of pregnancy. During this time, there is a free flow of pCRH and the normal negative feedback loop of the HPA axis becomes a positive one so that levels of CRH, ACTH, and cortisol rise rapidly (Majzoub & Karalis, 1999). The high levels of cortisol prompt the body to release a chemical compound, prostaglandin, which triggers uterine contractions to begin the labor process (Challis, Matthews, van Meir, & Ramirez, 1995). However, under abnormal circumstances these alterations to the system may be initiated prematurely, leading to premature delivery and low birthweight. For example, Lederman et al. (2004) found that in the wake of the terrorist attacks of September 11th, women living in New

York City during their first trimester of pregnancy at the time of the World Trade Center's collapse delivered infants with significantly shorter gestation and a smaller head circumference.

Transmission of Maternal Stress to Fetus

It has been postulated that maternal stress is transmitted to the fetus via three possible mechanisms, which may operate simultaneously and amplify each other's effects (Mulder et al., 2002). These mechanisms include: reduced uteroplacental blood flow, transplacental transport of maternal stress hormones, and secretion of placental CRH to the fetus (Mulder et al., 2002).

Reduced uteroplacental blood flow. Corticosteroids are stress response hormones which have abundant receptors in the placenta and exert strong effects on the tone of peripheral blood vessels. The activation of the sympathetic nervous system can reduce blood flow from the placenta to the uterus and fetus, leading to the restriction of fetal growth (Ohkawa et al., 1991; Nusken et al., 2011). This growth retardation is the result of a reduced level of oxygen and water transport through the placenta as well as decreased fetal uptake of glucose (Wallace et al., 2002). Teixeira, Fisk, and Glover (1999) found increased resistance of the uterine artery in women with high anxiety scores at 32 weeks' gestation. Kramer, Seguin, Lydon, & Goulet (2000) found several mediating factors of socio-economic disparities in intrauterine growth restriction (IUGR) including cigarette smoking, low gestational weight gain, and short stature, as well as maternal work and physical activity.

Transplacental transport of maternal stress hormones. In the placenta, 50-90% of maternal cortisol is transformed into cortisone, an inert and undamaging substance in the placenta, via the enzyme 11 β -hydroxysteroid dehydrogenase type 2 (11 β -HSD-2). This process serves to protect the fetus from excessive cortisol exposure. However, disproportionate amounts of cortisol can still reach the fetus if maternal cortisol levels are very high, 11 β -HSD-2 is

reduced or impaired, the placenta is immature during early pregnancy, or if placental function is poor (Seckl, 1997). Also, a high level of maternal prenatal stress can reduce the expression and activity of 11 β -HSD-2, leaving the fetus less well protected (Charil, Laplante, Villancourt, & King, 2010).

During a normal pregnancy, a reduction in 11 β -HSD-2 activity between 38-40 weeks' gestation has been associated with a rise in cortisol and subsequently a decrease in fetal growth (Murphy & Clifton, 2003). This decrease in activity is the mechanism by which cortisol concentrations rise at term to regulate fetal maturation and activate pathways associated with labor (Murphy et al., 2002). However, if a reduction in 11 β -HSD-2 activity occurs too early in the pregnancy, premature birth and a very low birth weight can occur (Seckl, 2004).

Fetal Programming

Fetal programming may explain the association between prenatal environmental events, altered fetal growth and development, and later pathophysiology (Seckl, 2004). It emphasizes that the developing HPA axis of the fetus in utero is programmed to respond to the environment it will soon enter, thereby producing effects that persist throughout life (Glover, O'Connor, & O'Donnell, 2010). In other words, the mother's outside environment is communicated to the developing fetus so that it can properly prepare and adapt to living outside the womb. The evolutionary origins of this process were adaptive so that mothers exposed to stressful environments would give birth to offspring with especially responsive stress systems that could give them an advantage in harsh environments (Neese, Bhatnagar, & Young, 2007). However, these developmental adaptations can permanently change structure, physiology, and metabolism, so that if the child is born into a peaceful environment where an overactive stress response is not

needed this mismatch predisposes the child to cardiovascular, metabolic, and endocrine disease in adult life (Godfrey & Barker, 2001).

A study of men and women in middle and late life born in Hertfordshire, UK between 1911 and 1930 found that those born with low birth weights had increased death rates from coronary heart disease in adult life. Among those 15,726 individuals death rates from heart disease fell progressively with increasing birth weight in both men and women (Osmond et al., 1993). Another study found that it was the infants born with a low birth weight due to intra-uterine growth retardation rather than prematurity who were at increased risk for coronary heart disease (Barker et al., 1993). Similar findings have been shown for the relationships between growth retardation and hypertension and diabetes (Godfrey & Barker, 2001).

People that experience impaired growth in *utero* may also continue to be exposed to an adverse environment in childhood and adult life, and these later stressors may produce the effects attributed to fetal programming (Godfrey & Barker, 2001). However the effects of fetal programming appear real because several studies have replicated the findings that there is an association between birth weight and coronary heart disease even when allowing for confounding variables in lifestyle, such as smoking, employment, diet, alcohol consumption, and exercise (Leon et al., 1997). The effects of fetal programming are pronounced when a fetus is born into an environment that he or she was not programmed for. For example, a fetus that has gestated during a famine is programmed to be small and need less food to survive. However, if that fetus is born after the famine has ended and lives when food is plentiful the infant will grow into adulthood consuming significantly more calories than he or she was programmed to need. Thus, fetal programming represents a set of mechanisms by which individuals that experience early life stress can suffer adverse effects of heart disease and diabetes.

Prenatal Stress and Brain Neurotransmitter Systems

Prenatal stress has been shown to alter the development of neurotransmitter systems, leading to morphological and neurochemical changes in the brain that increase the risk for pathophysiologies (Kofman, 2002).

The serotonergic system. There is a close relationship between the regulation of the HPA axis and the serotonin (5-HT) system (Mitchell, Rowe, Boska & Meaney, 1990). 5-HT is believed to play an important role in early brain development through the facilitation of synapse formation and maintenance in the central nervous system (Huizink, Mulder, & Buitelaar, 2004).

Hiyashi et al. (1998) exposed pregnant rats to mild prenatal stress through the use of crowding and saline injections during days 15-21 of gestation. On postnatal day 35 the 5-HT levels of the rats had decreased by 17%, 5-HIAA (a metabolite of 5-HT) levels had increased by 18%, and the metabolic rate had increased by 49%. These same changes have been associated with a reduced number of 5-HT_{1A} receptor binding sites in the hippocampus (Peters, 1990).

This disruption of the serotonergic system, which has been shown to be associated with mood disorders, anxiety, aggression, and impulsivity (Gorman, 2002), could be associated with changes in behavior of prenatally stressed offspring (Huizink, Mulder, & Buitelaar, 2004).

The dopaminergic system. Dopamine is involved in controlling movement and aiding the flow of information to the frontal cortex, which regulates thought and emotion. It is also linked to reward systems in the limbic system. Alterations in the dopaminergic system have been documented following prenatal stress (Huizink, Mulder, & Buitelaar, 2004). Henry et al. (1995) found that prenatal restraint stress (restraining pregnant rats in order to produce a stress response) induced increases in the dopamine sensitivity of the nucleus accumbens through changes in dopamine receptor density. The density of the D₂ receptor, whose activation increases

locomotion and facilitates memory consolidation (Richtand, Woods, Berger, & Strakowski, 2001), increased in the nucleus accumbens (Henry et al., 1995) whereas the density of the D3 receptor, whose activation inhibits locomotion as well as memory consolidation (Richtand, Woods, Berger, & Strakowski, 2001), decreased in the core and the shell of the nucleus accumbens (Henry et al., 1995).

The cholinergic system. The cholinergic system is involved in learning and memory (Messer, 2002) and also may be involved in mood and sleep disorders (Mitchell, Dalrymple-Alford, & Christie, 2002). Stress has been shown to increase the release of acetylcholine in the hippocampus (Mark, Rada, & Shors, 1996). Day et al. (1998) found that administration of CRH to prenatally stressed rats produced a greater release of acetylcholine in the hippocampus than did administration of an inactive substance. This same cholinergic hypersensitivity has been found in individuals with depression (Janowsky, Overstreet, & Nurnberger, 1994) and could contribute to mood disorders resulting from prenatal stress.

The noradrenergic system. A number of studies have shown that noradrenaline (NE) modulates aspects of memory, including acquisition of new information, the attentional component of memory storage, and working memory (Vermetten & Bremner, 2002). Reduced levels of NE were found in the cerebral cortex and locus coeruleus (the principal site for synthesis of norepinephrine in the brain) in prenatally stressed adult rats when measured immediately following a shock stress rather than under basal conditions (Takahashi, Turner, & Kalin, 1992). However, prenatal stress has been reported to elevate the basal concentration of NE in the hypothalamus (Peters, 1982). These findings suggest that prenatal stress leads to a dysregulation of the noradrenergic system.

Negative Outcomes Associated with Prenatal Stress

Effects on the Brain

Fetal brain development is a long process that takes place after the 10th gestational week when the foundations for all organs have been established (Charil, Laplante, & Vaillancourt, 2010). The fetal brain then undergoes rapid growth that is characterized by a high turnover of neuronal connections making it especially vulnerable to hormones that reach it in excessive amounts as a result of maternal stress (Weinstock, 2008). This excess can impede the formation of neural connections, reduce synaptic plasticity and neurotransmitter activity, which can lead to changes in cognitive function and behavior (Weinstock, 2008).

Animal studies. The majority of experiments on the effects of prenatal stress on brain development have been carried out in rats and rhesus monkeys (Weinstock, 2001). Stressors used in animal models, such as periods of physical restraint, unpredictable noise, repeated tail shocks, crowding, suspension, immobilization, and saline injections, are able to be applied in a controlled manner in terms of duration, frequency, and intensity (DiPietro, 2012). Animal studies thus provide valuable information on certain aspects of effects of prenatal stress on later development that cannot be addressed by studying humans (Huizink, Mulder, & Buitelaar, 2004), particularly because the developing rat brain closely resembles that of a human, especially in the embryonic stages (Bayer et al., 1993).

However it is important to note that the results, particularly in rodents, cannot be fully generalized to humans for several reasons. In contrast to most other mammals, the human and nonhuman primate fetus is relatively protected from increased maternal cortisol levels due to placental 11 β -HSD activity (Kofman, 2002). Furthermore, the human situation is more complicated with regard to stress responses because social support and coping style may have

moderating effects on the stress response and may reflect aspects of cognitive control over a stressful situation (Huizink, 2000).

Hippocampus. The hippocampus, a part of the limbic system, is important in learning and memory (Fujioka et al., 2006). Neurogenesis, the birth of neurons, continues throughout life and contributes to learning (Snyder, Kee, & Wojtowicz, 2001). Coe et al. (2003) found that when pregnant rhesus monkeys were acutely stressed for 25% of their 24-week gestation it resulted in a reduced hippocampal volume and an inhibition of neurogenesis in the dentate gyrus (a part of the hippocampal formation) of their offspring. Zhu et al. (2004) found that prenatal stress in rats caused oxidative stress (an imbalance in cell maintenance that can be damaging to the organism) leading to neuronal damage and loss of hippocampal volume in developing offspring. Furthermore, glucocorticoid receptors in the hippocampus are profuse because during development glucocorticoids are necessary for neuronal maturation (Fujioka et al., 2006). However, Uno et al. (1990) found that an excessive level of glucocorticoids in the hippocampus prenatally can produce toxic alterations in its neurons.

Research has also been done on the effects of poverty on the hippocampus. Hanson, Chandra, Wolfe, and Pollak (2011) found that hippocampal gray matter (neuronal cell bodies) is associated with household income, as children from lower SES backgrounds had less hippocampal gray matter and children from more affluent backgrounds had greater concentrations of gray matter. Farah et al. (2006) found that environmental stimulation and parental nurturance (associated with income level) were related to memory function, which is consistent with altered hippocampal function. Additionally, higher levels of chronic stress appear to be associated with smaller hippocampal volumes in adults (Gianaros et al., 2007).

Amygdala. The amygdala is a part of the limbic system and plays a central role in the processing of memory and emotions, in particular fear (Seymour & Dolan, 2008). Krazspulski et al. (2006) found that prenatal stress-exposed rats had offspring with 20-25% larger amygdalar nuclei volumes than the controls and 25-30% fewer neurons and glial cells. Higher levels of CRH and its receptors were found in the amygdala of prenatally stressed rats and larger amounts were released on stimulation (Ward et al., 2000). Prenatal stress was also found to cause expansion of the lateral nucleus of the amygdala, an area in which learned fear is encoded (Salm et al., 2004). These developmental differences may predispose exposed animals to fear-related behaviors, which may manifest in anxiety-like symptoms later in life (Lazinksi, Shea, & Steiner, 2008).

Prefrontal cortex. The prefrontal cortex, responsible for integration of executive function, is highly interconnected with other brain regions (Arnsten et al., 2010). Reduced neural density within it would therefore decrease the prefrontal cortex's ability to regulate executive functions such as planning, working memory, attention, problem solving, verbal reasoning, inhibition, and monitoring of actions (Elliot, 2003).

Murmu et al. (2006) examined the development of neurons in the limbic anterior cingulate (located around the corpus callosum, it is a central station for linking behavioral outcomes to motivation) and the orbitofronal cortex (a region in the frontal lobes involved in the cognitive processing of decision making). These cortical areas have been implicated in attentional processes, working memory, and in the regulation of emotional behavior (Dalley, Cardinal, & Robbins, 2004). In this study, pregnant rats were stressed daily from 15-20 days' gestation (of a 21-day gestation) and brains were then removed after 23 days of age for examination of dendrites. Murmu et al. (2006) found that offspring of mothers exposed to stress

during pregnancy (PS) displayed significantly lower spine density in the dorsal anterior cingulate (plays a role in rational cognitive functions) and orbitofrontal cortex (involved in decision-making) compared to control animals. This suggests that the brain regions of prenatally stressed offspring are less connected than those of controls and therefore may lack more integrated cognitive processes.

Neurodevelopmental Effects

Intellectual and language functioning. As stated previously, animal studies allow for random assignment while human studies do not. However, occasionally studies are able to take advantage of naturally occurring population-based disasters (DiPietro, 2012). In January of 1998 the Quebec Ice Storm created just such an event. The storm resulted in electrical power failures for three million individuals for anywhere from 6 hours to 5 weeks. During this time large numbers of pregnant women in various stages of pregnancy were randomly exposed to varying degrees of storm-related hardship (King & Laplante, 2005).

Laplante et al. (2004) studied the stressful effects of the ice storm on pregnant mothers and the subsequent development of their children. They administered the Bayley Mental Development Index (MDI), which is predictive of reading and spelling abilities at 8 years of age and general intellectual functioning in later development, to 58 toddlers who had experienced the ice storm *in utero*. They found that poorer Bayley MDI scores and lower productive and receptive language abilities were predicted by more severe prenatal stress, particularly if the stress occurred early in the pregnancy. Birth weight accounted for part of the variance on the Bayley scores, but only when the exposure occurred in the third trimester, suggesting that vulnerability to prenatal stress decreased as pregnancy progressed.

Behavioral problems. Gutteling et al. (2005) examined the relationship between infant temperament (behavioral style resulting from neurobiological factors) and maternal prenatal stress by measuring salivary cortisol levels throughout pregnancy and assessing the infant's temperament at 27 months using the Infant Characteristics Questionnaire (ICQ). The ICQ assesses difficult behavior (negative mood, withdrawal, high intensity, and low regularity of physical rhythms), adaptability (adaptation to novel situations), and attention regulation (task persistence and attention span). Gutteling et al. (2005) found that higher prenatal cortisol levels were predictors for both increased difficult behavior and decreased attention regulation in infants.

Prenatal Stress and Physiological Disorders in Adulthood

The correlation between prenatal stress and disease in adulthood is a direct result of fetal programming and the interaction between the prenatal and postnatal environment (Louey & Thornburg, 2005).

Cardiovascular disease. Cortisol increases blood pressure by increasing the sensitivity of the vasculature to epinephrine and norepinephrine (Seckl, 2004). Dodic et al. (1998) found that adult hypertension was produced in sheep exposed to excess glucocorticoids in utero. Furthermore, preterm birth and low birth weight, two outcomes of prenatal stress, have been associated with cardiovascular disease in adulthood (Latendresse, 2009). Eriksson et al. (2001) found that low birth weight boys who had rapid growth (increased body weight and body mass index) following their first year of life had an increased risk of coronary heart disease. Forsen et al. (2004) found that girls who were born short, followed by a rapid increase in body weight and body mass index in childhood were also at greater risk for heart disease. These effects of prenatal

stress are a direct result of the mis-match in the uterine environment and the outside world into which the child is born.

Diabetes. Cortisol counteracts insulin so that excessive glucocorticoids cause high blood sugar (Seckl, 2004). High glucose levels, also known as hyperglycemia, if unchecked can lead to diabetes (Joseph & Kramer, 1996). The activation of the HPA axis appears to contribute to the accumulation of fat tissue and vice versa with obesity itself constituting a chronic stressful state that may cause HPA dysfunction (Kyrou, Chrousos, & Tsigos, 2006). Obesity is well known to lead to an increased risk for conditions such as diabetes (Ford, Williamson, & Liu, 1997).

The prevalence of diabetes is quickly reaching epidemic proportions in the United States (and in the UK, though the numbers are different), with a national increase of 61% since 1990 (Mokad et al., 2001). In a random digital telephone survey of 195,005 adults across the country, African-Americans were found to have the highest rate of diagnosed diabetes (11.2%) among all race groups, and adults with less than a high school education had the highest rate (13.0%) among the educational levels (Mokad et al., 2001). This discrepancy could perhaps largely be explained by increased exposure to poverty-related stress.

Differences in Stress Reactivity

Individual differences in stress reactivity are in part mediated by varying levels of parental care (Caldji, Diorio, & Meaney, 2000). Parental rearing that results in enhanced reactivity to stress appears to increase the risk for illness in later life due to the fact that increased levels of glucocorticoids promote the development of multiple forms of chronic illness (Seckl & Meaney, 1994). The good news is that maternal care, as well as an environmentally enriched environment, can mitigate the negative effects of stress so that even the negative effects of poverty can be remediated (Meaney, 2001).

Caldji, Diorio, and Meaney (2000) studied the variations in maternal licking and grooming behavior of rat dams to explore the relationship between parental care and stress reactivity. They found that the offspring of high licking and grooming rats had a more modest HPA response to stress (decreased CRF levels in the hypothalamus and enhanced glucocorticoid negative feedback sensitivity). Interestingly, the magnitude of the stress response (measured by cortisol levels) was significantly correlated with the frequency of maternal licking and grooming during the first ten days of life. This timeframe coincides with a sensitive period of development of the HPA axis in humans (Phillips, 2001), suggesting that greater levels of maternal caresses, hugs, and kisses (equivalent to licking and grooming in rats) is beneficial to the development of a healthy stress response in newborns.

Caldji, Diorio, and Meaney (2001) also found that offspring of low-licking and grooming rats differed in behavioral responses to novelty. These offspring showed greater startle responses, decreased open field exploration, and longer latencies to eat food provided in a novel environment. This could be a link to difficult infant temperament in humans and potentially related to parental neglect.

Whom does Prenatal Stress Affect?

Socioeconomic Status

Socioeconomic status (SES), a measure of one's overall status and position in society, strongly influences an individual's experiences from childhood through adulthood. It is a complex construct based on household income, material resources, education and occupation, as well as related neighborhood and family characteristics, such as exposure to violence and toxins, prenatal care and provision of a cognitively stimulating environment (Hackman, Farrah, Meaney, 2010). Individuals of low childhood SES face various social and economic barriers to success

and wellbeing, likely accounting for the intergenerational transmission of poverty (Conger & Donnellan, 2007).

It has been shown that individuals from lower SES report greater exposure to stressful life events and a greater impact of these events on their lives than individuals from higher SES (Dohrenwend, 1973). The association between SES and stress may stem from environmental and social-psychological factors (Lupien, King, Meaney, & McEwen, 2001). Individuals lower on the SES ladder have fewer choices in residential environments and more frequently these environments are associated with increased mortality rates and crime (Haan, Kaplan, & Syme, 1989). Furthermore, it has been shown that higher SES decreases the likelihood of exposure to negative events such as social aggression and risk behaviors (Dohrenwend & Dohrenwend, 1970). Individuals of lower SES are exposed to a higher rate of change or instability in their lives, and this instability has been found to produce a higher level of individual distress (Broadhead, Kaplan, & James, 1983). These factors explain why a pregnant women living under these conditions might have an overactive stress response system and thereby influence the fetus' developmental path. Individual measures of SES such as marital status, maternal education, race, and age all interact to create toxic levels of stress.

Marital Status

The absence of positive social relationships has proven to be a risk factor for a wide range of negative health outcomes and is presumed to occur through a dysregulation of the HPA axis (Robles & Kiecolt-Glaser, 2003). "Social buffering", the ability of a partner to reduce the stress response, is a well-studied benefit of marriage (Hennessy, Kaiser, & Sachser, 2009). Oxytocin (a neurohypophysial hormone produced by the hypothalamus) is released during physical contact and promotes affiliative behavior and the formation of social bonds (Smith &

Wang, 2012). Attachment behaviors and social contact that occur through the release of oxytocin, thus, increase an individual's sense of security during times of stress and subsequently reduce HPA activity (Ravitz, Maunder, Hunter, Sthankiya, & Lancee, 2010). Furthermore, times of stress lead to an increased desire for intimacy. Studies using monkeys have shown that social isolation prior to the establishment of a new social pair increases proximity-seeking behavior, a behavior associated with higher cortisol levels (Smith, Birnie, & French, 2011). Therefore, social buffering, often in the context of marriage, has positive effects on many physiological responses (*e.g.*, cardiovascular reactivity, the immune system, the sympathetic nervous system, as well as behavioral reactions during times of stress) that promote health and overall wellbeing (Hennessy, Kaiser, & Sachser, 2009). Additionally, marriage may be a protective factor mitigating the risk of living in poverty, as the rate of married couples in poverty is only 6.9%, less than half the national average of 15.1% (U.S. Census Bureau, 2010).

Maternal Education

Education clusters together with other measures of SES and it is difficult to parse out its exact influence. There is a well-documented education gradient for both health status and health behaviors that can easily be linked to SES (Cutler & Lleras-Muney, 2006). The link between adverse birth outcomes and SES is typically identified using maternal level of education, which is listed on the birth certificate (Luo, Wilkins, & Kramer, 2006). A systematic review of socioeconomic disparities in adverse birth outcomes (Blumenshire, Egerter, Barclay, Cubbin, & Braveman, 2010) found that of the 106 studies reviewed 93 reported a significant association between at least one socioeconomic measure and one birth outcome. Low birthweight proved to be the adverse birth outcome with the strongest relationship with maternal education level. However, these effects varied a great deal across racial and ethnic subgroups. A significant

association between maternal education and low birthweight was found for both non-Hispanic white (Jaffee & Perloff, 2003) and African-American women (Nicolaidis, Ko, Saha, & Koepsell, 2004), but not for Hispanic women (Pearl, Braveman, & Abrams, 2001).

Maternal Race

There are severe racial discrepancies in poverty rates in the United States. In 2010, a national average of 15.1% lived in poverty. African-Americans greatly exceeded this average at 27.4% while only 9.9% of white Americans lived in poverty. These rates were highest for families headed by a single woman (31.6%), particularly if the woman was African-American or Hispanic (U.S. Census Bureau, 2010). Furthermore, stress-related diseases are disproportionately prevalent within the African-American population. Diabetes is 60% more common in African-Americans than white Americans and more than 45% of African-American women 20 years and older have high blood pressure (Commission on Social Determinants of Health, 2008). These glaring racial disparities in poverty and disease consequently translate into adverse birth outcomes (Hogue & Bremner, 2005). African-Americans suffer from the highest rate of infant mortality (Matthews & MacDorman, 2012) and are 2.2 times more likely to have low birth weight infants than their white counterparts (Dominguez, 2011).

Maternal Age

A 1992 groundbreaking study conducted by Arline Geronimus through the Harvard School of Public Health found that African-American women had better birth outcomes at a younger age than their white counterparts regardless of socioeconomic status. She posited that these disparities in adverse pregnancy outcomes may exist as a result of a lifetime of exposure to chronic stress (Hogue & Bremner, 2005). Her “weathering” hypothesis stated that African-Americans experience early health deterioration as a consequence of the cumulative impact of

repeated experience with social or economic adversity and political marginalization and that the stress inherent in living in a race-conscious society stigmatizes and disadvantages African-Americans causing disproportionate physiological deterioration (Geronimus, Hicken, Keen, & Bound, 2006). The effects of discrimination compounded by the stresses of living in poverty add up to become a toxic level of stress that weathers the reproductive system, leading to poor birth outcomes among older African-American women (Geronimus, 1992). Maternal age is thus critical to birth outcomes, particularly for African-Americans.

Future Implications & Social Work's Role

As we've seen, prenatal stress contributes to adverse birth outcomes (low birth weight, preterm birth, and small head circumference), which have been associated with changes in brain structure and function, cognitive and behavioral problems, as well as disease. Vulnerable populations, such as minorities and those living in poverty, are most susceptible to prenatal stress and its negative outcomes. However, despite fetal programming effects on later functioning, maternal caregiving and/or an enriched environment has been shown to decrease stress reactivity in offspring (Meaney, 2001). Therefore, social work has an opportunity to promote positive child development through the mother both during pregnancy and after birth. What is not known and needs further investigation is how to decrease circulating glucocorticoids amidst poverty, racism, and chronic life stress.

This study aims to examine the effects of prenatal stress and poverty on fetal growth. Questions to be addressed are: (1) Are there discrete types (subpopulations) of mothers in terms of reported stressful life events and, if so, how many different types of mothers are there, how prevalent are different types of mothers, and how are these mothers different in terms of stressful life events? (2) Is there a positive relationship between problematic births and maternal poverty,

medical risk related to fetal development, and race (African-American); a negative relationship between problematic births and maternal education, age, and marital status (married); and is the relationship between problematic births and maternal age different for European- and African-American mothers? (3) When controlling for demographic variables and medical risk related to fetal development, is there a stronger positive relationship between problematic births and mothers' stressful life events for mothers who are poor (i.e., are the effects of stressful life events moderated by poverty)? And finally, (4) when controlling for demographic variables and medical risk related to fetal development, is there a positive relationship between problematic births and mothers' stressful life events (i.e., is there a main effect of stressful life events)?

Chapter 2: Methodology

Methodology

Data for my dissertation were drawn from the Pregnancy Risk Assessment Monitoring System (PRAMS) research project. PRAMS is a joint research project between the state departments of health and the Centers for Disease Control and Prevention (CDC). PRAMS enhances information from birth certificates and is used to identify groups of women and infants at high risk for health problems, to monitor changes in health status, and to measure progress towards goals in improving the health of mothers and infants.

The CDC obtained the initial approval for the overall project through the CDC Institutional Review Board when it first began in 1987 and it continues to be reviewed each year. Individual states gain approval from federally-assured local IRBs and also go under review each year. The PRAMS questionnaire obtains sensitive and individually identifiable data but only PRAMS staff, contractors working on the PRAMS state project, and the PRAMS' computer system administrators have access to this information and they are trained in the privacy policy and required to sign a confidentiality pledge. I obtained the dataset without any identifying information and received IRB exemption status under 45 CFR 46 exempt category 4 from the University of Tennessee Office of Research and Engagement (Appendix E).

Design

The PRAMS sample is chosen from among all women who have had recent live births within the 40 participating states and New York City. Each state draws a stratified systematic sample of 100 to 250 new mothers every month from a frame of eligible birth certificates. Women from some groups are sampled at higher rates to ensure adequate data are available in smaller but higher-risk populations (i.e. most states oversample low weight births, and many

states stratify by mother's race or ethnicity as well). Annual sample sizes range from 1000 to 3400 per state.

PRAMS provides statewide estimates of specific perinatal health events among women delivering live infants. Each participating state uses a standardized data collection methodology developed by the CDC. However, stratification varies by state. Each state selects up to two stratification variables (e.g., birthweight, maternal race/ethnicity, maternal education, maternal age, geographic area, or Medicaid status).

This study used PRAMS data for the state of Tennessee from 2009. In Tennessee, exclusion criteria include cases of out-of-state births to residents, in-state births to nonresidents, delayed or early processing of birth certificates, missing information that does not allow for follow-up, multiple gestation infants (in this case, one member is randomly selected during data collection from twins or triplet sets for inclusion, and multiple gestations involving four or more siblings are excluded), adopted infants, and surrogate births. Tennessee uses birthweight as the stratification variable and therefore oversamples mothers who give birth to infants of low birth weight (weighing less than 2,500 grams or 5.5 pounds).

Data Sources

PRAMS data comes from two sources: birth certificates and questionnaires completed by mothers.

PRAMS questionnaire. PRAMS employs a mixed-mode data collection methodology; up to three self-administered surveys are mailed to mothers in the sample, and non-responders are followed up with telephone interviews. The first survey is typically mailed 2 to 3 months after delivery to collect information about postpartum maternal and infant experiences.

Self-reported survey data are linked to selected birth certificate data and weighted for

sample design (i.e., stratification), non-response, and non-coverage to create the PRAMS analysis data sets. The non-coverage weight accounts for omissions from the sampling frame, for example, if a birth certificate was filed late, after the sample was selected.

The PRAMS questionnaire is revised periodically, and each revision is referred to as a "phase." The 2009 data, the most recent data, which was used in this study, was collected with the sixth phase of the questionnaire that was in the field from 2009 through the end of 2012 (Appendix A).

Birth certificate. Data from the birth certificate are gathered from the individual states' vital statistics departments. Questionnaire responses are then linked to the birth certificate. Birth certificates provide fundamental information about delivery, health of the child at birth, and parental demographic information (Appendix D).

Variables Used in the Present Study

This study used variables from the PRAMS questionnaire and the birth certificate. These variables were used to operationalize three constructs: fetal development, stressful life events, and poverty.

Fetal growth variables. Fetal growth variables were derived from birth certificates. These variables included (1) preterm birth; (2) small for gestational age (SGA), defined as at or below the 10th percentile; (3) large for gestational age (LGA), defined as at or above the 90th percentile; and (4) problematic birth.

Preterm birth. Pregnancy dating has routinely been based off of a calculation, known as "Naegele's rule", which uses the date of the last menstrual period to estimate date of delivery (Dias, 2011). This calculation assumes the length of gestation to be 40 weeks. Gestational age in this study was measured in weeks, with 40 being the standard. I created a new variable to assess

for preterm birth, with below 37 weeks being the cut-off point (Goldenberg, Culhane, Iams, & Romero, 2008) and coded as 0 = no and 1 = yes.

SGA. Small for gestational age defined as at or below the 10th percentile was used as a measure of birthweight. This measure accounts for newborns who are smaller in size for their gestational age, which could indicate fetal growth restriction. The percentiles in this measure and the one for LGA come from population-based weight data obtained from newborns at the same gestational age and are the standards for defining SGA and LGA (Behrman & Butler, 2006). The variable was coded based on the percentile as 0 = no and 1 = yes.

LGA. Large for gestational age defined as at or above the 90th percentile was used as another measure of birthweight. This measure accounts for newborns who are larger in size for their gestational age, which could indicate diabetes in the mother. The variable was coded based on the percentile as 0 = no and 1 = yes.

Problematic birth. This variable was created to measure problematic birth outcomes. Any case that had one of the birth outcomes listed above was classified as having a problematic birth. The variable was coded as 0 = no and 1 = yes.

APGAR scores, birthweight in grams, and gestational age in weeks were used to describe the sample but were not used as measures of fetal growth for several reasons. APGAR scores were developed to assess a newborn's physical condition immediately after birth to determine need for extra medical care. While it is an assessment of a newborn's overall wellbeing at birth, it is not a predictive measure of future wellbeing and the score can easily be affected by a difficult birth. Furthermore, the APGAR score was established to measure the status of a term infant and therefore is not applicable to infants born prematurely (Papile, 2001; Casey, McIntire, & Leveno, 2001). Birthweight alone is not an accurate measure of fetal development in that it

does not account for confounding factors such as size and age of the mother, smoking during pregnancy, and prenatal care and nutrition (Wilcox, 2001). Gestational age is a relatively unreliable measure in that dating conception can be difficult. Both ultrasounds and calculations based on the last menstrual period are used to date a pregnancy and neither does so perfectly. In addition, in order for gestational age to be meaningful all births within the sample would have had to occur spontaneously (Behrman & Butler, 2007). Labor induction, which can confound measures of gestational age, occur for multiple reasons both elective and due to emergency (*e.g.*, overdue pregnancy, large for gestational age fetal weight, maternal health conditions, and preexisting baby health conditions). Only spontaneous labor is an accurate measure of gestational age (Heffner, Elkins, & Frett, 2003).

Stressors. Stressors were operationalized in two different ways. First, cumulative stress was examined by creating a variable summing the total number of stressors (0-17) experienced in the 12 months prior to delivery. And second, patterns of stressors were examined by identifying discrete subgroups of mothers with different patterns of stress through latent class analysis.

There are 17 items in the PRAMS questionnaire that address likely stressful events. Respondents indicate whether any of these events occurred during 12 months before birth. The items include: (1) arguing a lot, (2) inability to pay bills, (3) death of a loved one, (4) drug use by mother or others, (5) divorce, (6) ill family member, (7) physical fighting, (8) homelessness, (9) husband/partner in jail, (10) husband/partner lost job, (11) mother lost job, (12) change in address, (13) husband/partner does not want pregnancy, (14) car crash injury, (15) physical abuse from husband/partner in the 12 months before pregnancy, (16) physical abuse from husband/partner during pregnancy, and (17) pre-pregnancy history or treatment for anxiety/depression. Each of these variables was coded as 0 and 1 (0 = no, 1 = yes).

Poverty. Measurement of poverty in the United States is based on the Orshansky Poverty Thresholds, issued by the Census Bureau and the Poverty Guidelines, issued by the Department of Health and Human Services. While used for different purposes, they are both determined based on the cost of living for families of different sizes relative to income. These measurements determine eligibility for government assistance (Fisher, 2003).

However, the Tennessee PRAMS questionnaire asks for the participant's income based on levels (*e.g.* \$20,000-\$24,999). This does not allow for comparison between income level and number of dependents. Therefore, while it is not ideal, poverty in this study was operationalized based on method of payment for the delivery. Medicaid, recorded from the birth certificate, was used as an indication of poverty. The variable was recoded as 0 and 1, where 1 = Medicaid and 0 = all other forms of payment (including private insurance, self-pay, Indian Health Service, CHAMPUS/TriCare, and other government assistance). Eligibility for TennCare in 2009 was 185% of the federal poverty level (\$22,050 for a family of 4), making the yearly income for a family of four \$40,792. Within this study's sample, 46.6% made less than \$20,000 a year and only 25.1% made over \$50,000 a year, implying that Medicaid status is a reasonable measure of poverty.

Control variables and demographic characteristics. Control variables included marital status [married (1) or other (0)], maternal education measured in year intervals (0-8, 9-11, 12, 13-15, and >16), race (African-American or European-America), medical risk, which included diabetes, hypertension, and previous preterm birth [coded as yes (1) or no (0)], maternal age measured in year intervals (<17, 18-19, 20-24, 25-29, 30-34, 35-39, and 40+), and a cross-product variable measuring the interaction between age and race. Also, income, urban/rural status, and child's sex data were used to further describe important sample characteristics.

Chapter 3: Results

Results

This chapter will explain the results of my analyses of the 2009 Tennessee PRAMS data. It includes a summary of the sample characteristics, the outcomes of my exploration of a maternal stressor typology, the relationship between birth outcomes and demographic characteristics as well as medical risk, and the relationship between problematic birth and stressful life events when controlling for demographic characteristics and medical risk.

Sample

This section will describe the sample size and demographic characteristics. It will also report information about unit (i.e., failure of a mother to respond to the survey as a whole) and item nonresponse (i.e., failure of a mother to answer one or more survey items that the mother is eligible to answer).

Stata (Version 13) was used to compute sample statistics. More specifically, given the complex survey design, the “svy” function was used, with a stratification variable (STRATUMC), a finite population correction variable (TOTCNT), and a sampling weight variable (WTANAL).

Sample size. The total unweighted sample was 1,161, including 591 (50.9%) low-weight births. The weighted sample, taking account of the oversampling of low weight births, was 80,157 and represented the total number of Tennessee births eligible for study participation in 2009.

A total of 739 of 1,161 eligible mothers (63.7%) completed the PRAMS questionnaire. This subsample, weighted for non-response, was 80,157. Of the 422 mothers who did not complete the PRAMS questionnaire, 177 (15.2%) were contacted but did not participate and 245

(21.1%) were not contacted (Appendix 1 – PRAMS sampling frame).

In the present study three cases were excluded from the subsample of 739 mothers who completed the PRAMS questionnaire because gestational age was reported to be less than 22 weeks (i.e., 17, 20, and 21 weeks). These cases were excluded because it is questionable whether births before 22 weeks are viable, so it is possible that a recording error was made.

The sample consisted primarily of European- and African-American mothers, with very small absolute numbers of mothers of other races/ethnicities. Putting all the others in an “other” category would have made that category very heterogeneous, and because there are clear known differences in birth outcomes between European- and African-American mothers, mothers who were not European- or African-American were excluded from the present study. This deleted 84 cases, bringing the unweighted sample to 652.

The 652 births were used in the analyses reported in the present study to represent information concerning 69,743 eligible births.

Demographic characteristics. The majority of the sample was between the ages of 20 and 34 (70.6%) with a mean age of 26.3 ($SD = 6.48$) (Table 1). Mothers age 19 years or younger accounted for 17.0% of the sample, and 12.4% were 35 years or older.

The majority of mothers identified themselves as European-American (79.7%), and 20.3% identified themselves as African-American. Most mothers lived in urban settings (72.6%), and over half were married (54.7%).

Over three quarters of the sample (81.2%) had high school educations or more, with 26.8% having 1-3 years of post-secondary education and 21.4% having 4 or more years of post-secondary education.

No data were missing for race, age, education, or marital status. Data for rural/urban living status were missing for 4.9%.

Poverty. Almost half of the sample had incomes of less than \$20,000 a year (46.6%), while 28.3% had incomes between \$20,000 and \$50,000, and 25.1% made more than \$50,000 a year. Over half of the population received Medicaid (58.8%) and enrolled in Women Infant and Children nutrition services (WIC) (55.4%). Of the mothers on Medicaid, 86% had incomes less than \$25,000 a year while 56% of those with other types of insurance made more than \$50,000 a year. On income 6.5% of the data were missing, and on type of insurance 3.8% were missing.

Stressors. Mothers reported a mean number of stressors of 2.62 out of the 17 possible stressors ($SD = 2.32$, 95% CI [2.39, 2.85]). Number of stressors experienced was positively skewed and kurtotic ($skew = 1.02$, $kurtosis = 3.72$) (Figure 1). Moving was the most often reported stressor (43.1%), and car crash injury was the lowest (2.4%) (Table 2). Other commonly reported stressors were having ill family members (30.7%), arguing a lot (26.6%), and being unable to pay bills (26.3%). The largest percentage of missing data (7.9%) came from the question about domestic abuse during pregnancy. This is misleading, though, because mothers under the age of 18 were not asked questions about abuse, therefore this percentage is inflated. After accounting for the 33 individuals younger than 18 years of age, the percentage of missing data dropped to 2.1% and 2.4% on those two questions. The least amount of missing data (0.36%) occurred on the question about a move in the last 12 months.

Fetal growth. Infant gestational age was calculated using weeks, based on mothers' reports of their last menstrual periods. Gestational weeks ranged from 22 to 44, with a mean of 38.39 ($SD = 2.00$, 95% CI [38.26, 38.52]). The distribution was negatively skewed and kurtotic

(*skew* = -2.91, *kurtosis* = 17.27) (Figure 2). Only 10.8% of births occurred before 37 weeks' gestation (i.e., preterm birth) (Table 3).

Unadjusted birthweights ranged from 320 to 4,451 grams with a mean of 3,236.44 (*SD* = 532.39, 95% CI [3199.31, 3273.58]). The distribution of unadjusted birthweight was negatively skewed and kurtotic (*skew* = -1.31, *kurtosis* = 6.72) (Figure 3).

Most births (91.6%) were of normal birthweight of 2,500 grams or more. Births classified as being small for gestational age (SGA) based on the 10th percentile totaled 8.7%.

Adjusting for gestational age and infant sex (Oken, Kleinman, Rich-Edwards, & Gillman, 2003), the mean Z-value of birthweight for the sample was -0.17 (*SD* = .85, 95% CI [-0.25, -0.09]) indicating that the sample's birthweight was lower than the national average by .17 standard deviations, a relatively small difference (Figure 4). Mean birthweight percentile for the sample was 45.00 (*SD* = 25.88, 95% CI [42.49, 47.52]), also lower than the national average (Figure 5).

APGAR scores ranged from 0 – 10 with a mean of 8.77 (*SD* = .76, 95% CI [8.71, 8.83]). The majority of the sample (97.8%) had normal APGAR scores, with only 0.41% having critically low scores (below 4). The distribution was also negatively skewed and kurtotic (*skew* = -4.67, *kurtosis* = 36.88) (Figure 6).

Medical risk. Of the five diagnoses of medical risk listed on the birth certificate, specific data were only available and examined for three, none of which had any missing data. Mothers diagnosed with diabetes made up 3.36% of the sample, and those with hypertension made up 6.95%. Previous preterm births accounted for 2.88% of the sample.

Maternal Stressor Typology

Research question. Are there discrete types (subpopulations) of mothers in terms of reported stressful life events and, if so, how many different types of mothers are there, how prevalent are different types of mothers, and how are these mothers different in terms of stressful life events?

Data analysis. I used latent class analysis (LCA) to: (1) explore whether there were discrete types of mothers in terms of reported stressful life events; (2) determine the size of the subgroups; and (3) assign mothers to subgroups. LCA is a statistical method used to identify discrete subtypes (subpopulations) of similar cases, in this case mothers with particular patterns of reported stressors, within an overall population (Collins & Lanza, 2010; Muthén, 2008). These subtypes are referred to as “latent classes,” and the classes form the categories of a discrete latent variable. The variable is latent in that it is not directly observable but is inferred from observed variables (*indicators*), in this case reported stressors among 17 categories. The variable is discrete in that it is not presumed to reflect a continuum.

I did not have *a priori* hypotheses about the number of latent classes so I estimated and compared models with one through five classes using Mplus 7.11 (Muthén & Muthén, 1998-2012) and the robust maximum likelihood ratio estimator; the indicator variables were dichotomous. Covariances were fixed among latent class indicators to zero, and I allowed thresholds to vary across classes. Using full information maximum likelihood under the assumption that data were missing at random, all available data were used to estimate the model. I used “complex” survey analysis with the stratification variable (STRATUMC), the finite population correction variable (TOTCNT), and the sampling weight variable (WTANAL).

There is general agreement that theory, interpretability, parsimony, and class size should be considered in selecting the number of latent classes. In addition, statistical indices can be used in model selection. However, there is no definitive test of the “true” number of classes, so I considered four of the most promising statistical criteria (Nylund, Asparoutiov, & Muthén, 2007): (1) the Bayesian information criterion (BIC: Schwarz, 1978); (2) the sample-size adjusted Bayesian information criterion (SSABIC; Sclove, 1987); (3) the Vuong-Lo-Mendell-Rubin likelihood ratio test (Lo, Mendell, & Rubin, 2011); and (4) the Lo-Mendell-Rubin adjusted likelihood ratio test (LMR) (Lo et al., 2001) (The parametric bootstrapped likelihood ratio test [McLachlan & Peel, 2000] is preferable to either of these tests, but it is not available for the analysis of complex samples.). In addition, after identifying the most plausible model I considered the degree of error associated with the assignment of mothers to classes as indicated by entropy, an aggregate measure of classification uncertainty and, more importantly, the mean probability of class membership for each class.

Results. I selected a two-class model for several reasons. First, although the BIC and SSABIC continued to decrease as the number of classes increased, in going from the one- to the two-class model there was a large decrease, followed by a much smaller decrease for the three-, four-, and five- class solution. This pattern suggested that the addition of a third, fourth, and fifth class did not meaningfully improve model fit, and that the two-class model provided the best balance between model fit and parsimony. Second, there was minimal error associated with the assignment of mothers to these two latent classes, suggesting that the model adequately defined the data; the mean probability of class membership was .93 and .88 for Class-1 and Class-2 respectively, and entropy was .69 (Table 4). Third, class sizes were adequate in the two-class model. Fourth, multiple sets of starting values indicated a single maximum likelihood solution

for the two-class model, suggesting that it was identified. Fifth, characteristics of the two classes were interpretable and substantively meaningful. Finally, although results of the Vuong-Lo-Mendell-Rubin likelihood ratio test and the Lo-Mendell-Rubin adjusted likelihood ratio test were not statistically significant (i.e., $p = .058$ and $.059$, respectively), in combination with other indicators the two-class model fit relatively well.

One class of mothers accounted for 60% of the sample, and I will refer to these as *Unstressed* mothers. The second class accounted for 40% of the sample, and I will refer to these as *Stressed* mothers. However, it is important to note that these labels refer to the relative standing of these two groups, not to an absolute standard.

There was a quantitative difference between classes in that the proportion of *Stressed* mothers who experienced stressors was higher overall (.20 mean difference) and also higher for each stressful life event. In addition, there were qualitative disparities in that these differences were higher for some types of stressful life events than for others (.01 to .46) (Figure 7).

Notable differences existed in the percentage of *Stressed* and *Unstressed* mothers on seven of the 17 stressful life events (Figure 7). In the 12 months prior to giving birth *Stressed* mothers were more likely to have: (1) someone very close to them die ($OR = 3.70$, $p = .042$); (2) a family member who was very sick and had to go into the hospital ($OR = 2.75$, $p = .012$); or (3) moved to a new address ($OR = 2.65$, $p < .001$). In addition, *Stressed* mothers were the only mothers who reported: (4) separation or divorce; (5) experiencing a physical fight; (6) homelessness; or (7) being pushed, hit, slapped, kicked, choked or physically hurt by a husband or partner in some other way in the 12 months before pregnancy. Given that no *Unstressed* mothers had these latter experiences it was not possible to compute odds ratios or conduct tests of statistical significance.

Birth Outcomes and Demographic Variables and Medical Risk

Research questions. Is there a positive relationship between problematic births and maternal poverty, medical risk related to fetal development, and race (African-American); a negative relationship between problematic births and maternal education, age, and marital status (married); and is the relationship between problematic births and maternal age different for European- and African-American mothers?

Data analysis. Using Mplus (Version 7.11) to examine the relationship between birth outcomes on the one hand, and demographic variables, medical risk, and stressors on the other, and because all four indicators of birth outcomes were dichotomous, I used logistic regression in each of these analyses. Finally, I applied the “complex” survey analysis with the stratification variable (STRATUMC), the finite population correction variable (TOTCNT), and the sampling weight variable (WTANAL).

I tested non-directional hypotheses because, although previous research suggested certain directional relationships, results contrary to these expectations would be important to identify.

Number of stressors and maternal age were centered to facilitate interpretation of results, and to minimize multicollinearity resulting from use of these variables to create cross-product variables to test interactions (i.e., age multiplied by African-American, number of stressors multiplied by Medicaid, stress typology multiplied by Medicaid).

I used multiple imputation to handle the small percentage of missing data. Medical risk and all demographic variables were purposely employed to impute missing data in all the analyses reported below. In addition, the specific dependent variable used in each particular analysis was used in imputation. Finally, for each analysis 100 data sets were imputed.

Results. Medicaid status and medical risk had the most consistent and prominent relationships with problematic births. Controlling for medical risk and other demographic characteristics, Medicaid recipients were more likely than mothers with other types of insurance to have some type of problematic birth (Table 5), or to give birth prematurely (Table 6), or to have children who were large for gestational age (Table 7), but they were not more likely to have children who were small for gestational age (Table 8). These relationships similarly existed through bivariate analysis as Medicaid status was a positive predictor of both problematic birth and preterm delivery, but did not predict large for gestational age.

Both the bivariate results and results controlling for demographic characteristics showed that mothers with medical risks (i.e., diabetes, hypertension, previous preterm birth, previous poor pregnancy outcome, or previous cesarean delivery) were more likely than those without medical risks to have some type of problematic birth, or to give birth preterm, but not more likely to give birth to children who were small or large for gestational age.

African-American mothers were less likely than European-Americans to give birth to children who were large for gestational age, controlling for medical risk and other demographic characteristics, but there were no other statistically significant main effects for race. However, the relationship between race and problematic birth overall was moderated by age. This is illustrated in Figure 8, which shows the probability of problematic births for European- and African-American mothers across the age range in the sample (i.e., approximately 14 through 44 years old, or 12 years below and 18 years above the mean age). In particular, young African-American mothers (below the mean age) were less likely than European-Americans to have problematic births overall. At a younger age African-Americans had better birth outcomes than European-Americans, but that advantage disappeared with age. At 12 years below the mean age

(14.3 years) the largest difference in probabilities of problematic birth between the two races was .06. Beyond that, the differences were even smaller. Finally, there were two statistically significant bivariate relationships between race and birth outcomes: African-American mothers were less likely than European-Americans to give birth to children who were large for gestational age, and to give birth preterm.

Finally, results demonstrated no statistically significant main effects of age and education on any birth outcomes when controlling for medical risk and other demographic characteristics, but these two variables did have negative bivariate relationships with small for gestational age. Notably, mothers who were more educated and mothers who were younger were less likely to give birth to children who were small for gestational age.

Birth Outcomes and Stressors

Research questions. When controlling for demographic variables and medical risk related to fetal development, is there a stronger positive relationship between problematic births and mothers' stressful life events for mothers who are poor (i.e., are the effects of stressful life events moderated by poverty)?

When controlling for demographic variables and medical risk related to fetal development, is there a positive relationship between problematic births and mothers' stressful life events (i.e., is there a main effect of stressful life events)?

Results. With one exception, stressors had no statistically significant main or interaction effects on problematic births when controlling for demographic variables and medical risk. However, Medicaid status did moderate the relationship between the number of stressors and small-for-gestational-age births. This is illustrated in Figure 9, which shows the probabilities of small-for-gestational births for mothers who did and did not receive Medicaid across the range of

stressors in the sample (i.e., 0 through 12, or approximately 3 below through 9 above the mean). The figure demonstrates very little or a slightly negative relationship between number of stressors and the probability of small-for-gestational-age births for mothers who did not receive Medicaid, but a positive relationship for Medicaid recipients. Furthermore, this divergence was most pronounced for mothers who had more than the average number of stressors (2.62, approximately the same number of stressors that differentiated *Stressed* and *Unstressed* mothers, as discussed above).

Finally, statistically significant bivariate relationships existed between number of stressors and problematic births overall and small-for-gestational-age births. That is, mothers who had more stressors were more likely to have problematic births overall and small-for-gestational-age births in particular.

Chapter 4: Discussion

Summary of Results

The objective of this study was to determine the effects of prenatal stress on fetal growth for women living in poverty, and in particular to determine whether the effects were different for African-American and European-American women.

Results demonstrated that women who participated in the 2009 Tennessee PRAMS study fit into one of two classes, *stressed* or *unstressed*. The *stressed* mothers experienced a higher overall proportion of stressors and were the only mothers to experience certain stressors (i.e. separation or divorce, a physical fight, homelessness, or partner abuse 12 months before pregnancy). However, being in the *stressed* class did not increase a mother's chance of having a problematic birth. The only way in which stress had an effect on fetal growth was moderated by Medicaid. The probability of having an infant small for gestational age increased by the number of stressors experienced for women receiving Medicaid.

Both Medicaid status and medical risk were significant predictors of problematic birth. In particular, having a medical risk or receiving Medicaid increased the probability of giving birth prematurely and having an infant large for gestational age, but not having an infant small for gestational age. Results also indicated that young African-American women were less likely to have a problematic birth and that at a younger age African-Americans actually had slightly better birth outcomes than European-American women.

Sample and Design

While PRAMS is a unique and valuable national surveillance project, it is not without limitations. The biggest methodological limitation of PRAMS is in its survey design. PRAMS data are comprised of birth certificate information and mailed questionnaire responses. The

questionnaire serves as the principal source of maternal behavioral information for the time before, during, and after the mother's most recent pregnancy. Therefore, it is critical to the overall project but is subject to error in its nature as a mailed survey.

Mailed surveys suffer from non-coverage and non-response bias. PRAMS only surveys women who have had a live birth and does not include women who have experienced a miscarriage, fetal death, or stillbirth. Non-coverage of these women could lead to a misrepresentation of the sample population due to the fact that these women might represent those who had poorer health during pregnancy and/or less access to prenatal care. Similarly, women who did not have good birth outcomes may be more hesitant to respond to the questionnaire, thus attenuating the results.

While the goal of PRAMS is to achieve a 100% response rate, 65% is considered adequate. This accepted rate was lowered from 70% in 2007 due to declining national response rates (National Research Council, 2013). In 2009, Tennessee had a 67% weighted response rate (60% within the low birthweight stratum and 68% within the normal birthweight stratum).

Non-response to mailed surveys is a well-studied occurrence that exists for several reasons and may be particularly relevant to the data used in this dissertation. This error can be broken down into two categories: noncontact and refusal.

Noncontact, failure to reach the intended subject, is a limitation that can occur due to insufficient postage, incorrect mailing address, bulk mail delay or non-delivery by post office, or interception and disposal of mail by a family member or significant other (Daly, Jones, Gereau, & Levy, 2011). In this study, *stressed* mothers reported moving as the greatest stressor, implying that among this group a change of address occurred frequently. This could weaken results in that

mothers experiencing greater amounts of stress were unable to be located to complete the questionnaire.

Refusal is another constraint that occurs for reasons such as: no postage-paid return envelope provided, unclear survey instructions, survey too long or complicated, mistrust of confidentiality assurances, insufficient incentive/payment, unappealing survey topic, lack of interest, or competition with other mailings (2011). Non-response due to refusal exists on two levels: unit non-response, the complete absence of a questionnaire, and item non-response, absence of answers to specific questions (Yan & Curtin, 2010). In this study, teenagers were given a different questionnaire without questions regarding abuse before and during pregnancy. This, along with a lower response rate to abuse questions in general, increases the degree of item non-response and may limit the power of analyses related abuse during pregnancy.

However, because mailed surveys suffer from lower response rates, PRAMS employs a mixed mode method of surveillance and utilizes Dillman's Tailored Design Method to increase response rates. PRAMS has ready access to mothers' mailing addresses, therefore mail is used as the primary method of data collection. Telephone follow-up calls are made after three mailed non-responses. Aggregate data from 19 PRAMS states in 2000 showed that telephone follow-ups increased response rates by an average of 15% (Shulman, Gilbert, & Lansky, 2006). Don Dillman, a distinguished survey methodology researcher, served as a consultant during the PRAMS methodology development. His Tailored Design Method (TDM) (2000) is currently utilized to achieve desired response rates. Features of the TDM employed by PRAMS to increase response rates include: making multiple and varied contacts, providing a token incentive, developing a "respondent-friendly" questionnaire, providing envelopes with first class stamps,

and personalizing all correspondence. Again, this method was designed with PRAMS in mind, specifically.

Multiple contact attempts via mail include a pre-letter, three separate mailings including the survey, and a “tickler” (thank you/reminder note between the first and second survey mailing). Telephone calls to non-responders are attempted 15 times, staggered over different times of the day and different days of the week (Shulman, Gilbert, & Lansky, 2006). Mail respondents are given a \$10 gift card and telephone respondents a \$20 gift card.

Four versions of the questionnaire are available (English adult, Spanish adult, English teen, and Spanish teen) to make it more “respondent-friendly” and a bilingual telephone interviewer is also available for telephone respondents. Mailed questionnaires are affixed with first class stamps as opposed to bulk mail postage being printed on the envelope, which has been shown to increase response rates by 2-4% (Dillman, 2000).

Furthermore, PRAMS developed a unique non-response weight to account for non-respondents. Non-response adjustment factors were developed to attempt to compensate for the tendency of women having certain characteristics to respond at lower rates than women without those characteristics. For example, women of lower education and/or who are unmarried have proven to have higher rates of non-response (Shulman, Gilbert, & Lansky, 2006). These adjustment factors are used to calculate the non-response weight under the assumption that non-responders would have provided similar answers to responders within their stratum and adjustment category (CDC, PRAMS model protocol, 2009). Additionally, within this study I used multiple imputation, a statistical inference of the missing value (Rubin, 1987), during data analysis in order to account for small percentage of item non-response.

Another limitation of the PRAMS questionnaire is that it is self-reported. Self-report measures suffer from social desirability and recall bias (Dietz et al., 2014). The questionnaire is mailed to the selected sample 3-6 months after the delivery of their infants, during which time the mother could easily have forgotten events and behaviors that took place during her pregnancy. Recall also is related to birth outcomes, with moms with worse birth outcomes remembering more negative aspects of pregnancy. This could lead to an overestimation of negative events for mothers who had poor birth outcomes and weaken life stressors as a measure of stress. Certain items on the questionnaire are also personal in nature, which could lead to an overestimation of positive behaviors (*i.e.*, prenatal care and exercise) and underestimation of negative behaviors (*i.e.*, smoking, drinking, and abuse).

Fetal Growth Variables

Fetal growth is a difficult construct to measure due to the various factors involved in human pregnancy and the complexity of development. The first issue to arise is in accurately assessing gestational age.

The most common method of dating pregnancy is by calculating the date of a woman's last menstrual period (LMP). Known as Naegele's rule, the expected delivery date is estimated by adding one year, subtracting three months, and adding seven days to the date of the first day of a woman's LMP (Dias, 2011). This method, which PRAMS uses to calculate gestational age, suffers from a great deal of error. Naegele assumes every woman's menstrual cycle to be 28 days and for ovulation to occur mid-cycle. However, recollection of menstrual dates is difficult and time of ovulation varies from person to person (Rowland et al., 2002). Inaccuracies in recording the date of LMP have been noted particularly among women with low socioeconomic status (Savitz et al., 2002). Women with poor nutrition, higher rates of stress, and who abuse

substances have more irregular periods (Fleming, Velazquez, Eckert, Lucas, & Watkins, 2012), making gestational age hardest to measure among the population this study intended to examine and perhaps weakening results related to preterm birth and small for gestational age.

A more accurate measure of pregnancy dating is through ultrasound examination of fetal crown-rump-length. Within the first 12 weeks of gestation there is little variation in fetal growth regardless of maternal physical characteristics, environment, and behaviors (Lazairu, Davis, & McNutt, 2013). Therefore a fetal crown to rump length during this time can closely date conception (Dias, 2011). Yet, women who are socioeconomically disadvantaged are more likely to receive late or no prenatal care, again causing inaccuracies in dating pregnancy (National Academy of Sciences Committee on Understanding Premature Birth and Assuring Healthy Outcomes, 2007). This fact further points to a potential weakness in accurately measuring gestational age among women who are highly stressed and impoverished within the PRAMS sample.

Premature birth is defined as any birth occurring before 37 weeks' gestation. However, prematurity can be further categorized as extremely preterm (less than 28 weeks gestation), very preterm (28-32 weeks gestation), and moderate to late preterm (32-37 weeks gestation) (Goldenberg, Culhane, Iams, & Romero, 2008). The majority of preterm infants are born between 32 and 37 weeks' gestation and many have normal birthweights (McCormick et al., 2006), with infants born before 32 weeks most at risk for morbidity and mortality (Engle, Tomashek, & Wallman, 2007). Thus, there is great heterogeneity among preterm births. This study classified preterm birth as any birth occurring before 37 weeks gestation, lumping extremely preterm and late preterm infants together. Preterm birth, as defined by time (all births before 37 weeks gestation), is an unusual outcome measure in that its clinical manifestations

vary widely (Kramer et al., 2012). More accurate measures of fetal and infant maturity exist but are more challenging to assess. Therefore, within this study infants who were measured as being born late preterm may have actually been born on time but were inaccurately measured for gestational age. These late preterm births may erroneously increase the rate of preterm births confounding results.

The last organ to fully develop *in utero* is the lungs, leaving them vulnerable and underdeveloped when infants are born prematurely. Surfactant, a fatty substance that coats the lining of the aveoli (air sacs that aid in the oxygenation of blood), keeps the lungs from collapsing and supports exhalation. Fetuses begin to produce surfactant between 24 and 28 weeks' gestation, increasing production incrementally as gestation lengthens (Torday & Rehan, 2007). Surfactant is dispersed into amniotic fluid in increasing concentrations and can be measured to assess for lung maturation. This, however, can only be done through amniocentesis, a somewhat risky procedure performed prenatally. Measuring surfactant, while risky, would prove gestational age and accurately portray preterm birth. It is also important to note that extremely preterm infants would have less surfactant and thus need more immediate attention following birth, adding to reported complications.

The distribution of white matter, consisting of myelinated axons in the brain and critical to learning, has been correlated with gestational age (the lower the gestational age, the lower the white matter integrity) (Kapellou et al., 2006). Functional MRIs, though costly, could be used to assess for white matter and evaluate infant maturity (Ment & Vohr, 2008). This procedure would unlikely to be covered by Medicaid and thus would be only available to populations in higher SES brackets, increasing the disparity in birth outcomes between high and low SES populations.

Birthweight is a composite of fetal growth and length of gestation and is as complex and multifactorial a condition as preterm delivery. Traditional classifications of small for gestational age (SGA) and large for gestational age (LGA) are defined as infants below the 10th and above the 90th percentiles at each gestational age (Wilcox, 2001). However, there are large differences between and within populations, which remain unaccounted for when diagnosing SGA and LGA (Butler & Behrman, 2007). Furthermore, associations of fetal growth with later disease span the entire birthweight spectrum, and are not limited to infants above or below a certain cut-point (Gillman, 2002). Fetal growth variables (*i.e.*, SGA and LGA) in this study did not take into account factors such as sex of the infant, race, ethnicity, and family medical history that would provide a more reliable interpretation of expected birthweight for gestational age, thereby potentially weakening results.

Stress

The greatest limitation to understanding stress and particularly measuring it is in the lack of agreement as to its source. Stress is a physiological response but is often measured as an occurrence of stressful stimuli and perception of those events through the appraisal of daily life hassles and checklists (Monroe, 2008). PRAMS uses this method of measuring daily life stressors in its questionnaire, but fails to account for timing of the stressor and the mode through which it presents itself in the mothers' lives. This could be especially important to my findings because the timing of stressors has a great impact on fetal development, where greater stress during certain periods of pregnancy could increase chances of suffering from adverse birth outcomes.

It is well known that the developing fetus is physiologically responsive to maternal stress, influencing development *in utero* (Christian, 2012). However, the threat that stress poses during

specific periods of pregnancy has just recently become a topic of research. A large population-based study of 2.6 million Swedish pregnancies between the years of 1973 and 2004 found that for the 32,286 pregnant women who experienced stress through the death of the fetus' father or a first-degree relative of the mother, gestational months 5 and 6 proved to be the most vulnerable to adverse birth outcomes (shortened gestation, premature birth, low birthweight and SGA) (Class, Lichtenstein, Långström, & D'Onofrio, 2011). This study contradicts several previous findings suggesting that the first trimester is the most sensitive to maternal stress (deWeerth & Buitelaar, 2005). Because the source of maternal glucocorticoid delivery to the fetus is through the placenta, it is thought to be the placental enzyme 11β -HSD2 that controls the difference in sensitive timing effects. Measuring this enzyme is of utmost importance for future research into prenatal stress and its relation to distressing life events.

During the first trimester of pregnancy when the placenta is still immature and 11β -HSD2 is suppressed, there is a free flow of cortisol (Mulder et al., 2002). This primary surge in cortisol weakens the mother's immune system, ensuring that her body does not attack and reject the newly growing fetus (Makrigiannakis et al., 2001). During the third trimester, 11β -HSD2 is inactivated to guarantee final maturation of all organs and initiate labor (Majzoub & Karalis, 1999). Therefore, it makes sense that the second trimester level of cortisol would be most influential on fetal development and adverse birth outcomes. The PRAMS questionnaire does not ask the mother to specify during which month or trimester of pregnancy she experienced each stressful life event. Knowing the time frame in which a stressor was experienced would provide a greater depth to life events as a measure of cause of stress.

Stress is prevalent in society and affects many, yet differs in type. Chronic stress emerges from factors such as enduring economic strain and discriminatory experiences while acute stress

transpires from severe life events such as war, natural disaster, and divorce (Schneiderman, Ironson, & Siegel, 2005). While we can infer the differentiation in type based on the PRAMS questions, there is no way to determine if a stressor occurred one time or was a consistent presence in the mother's life. Therefore, this study implies all stressors have equal significance throughout pregnancy.

While acute stress can lead to chronic stress, the effects of an acute stressor are temporary and do not have enduring effects. A car crash injury, for example, would cause a flood of cortisol and actually help the body respond to that shock. If this said car crash occurred during pregnancy, the enzyme 11 β -HSD2 would convert the cortisol to cortisone, effectively protecting the fetus from the acute stressor. However, if the body is in a constant state of stress, for example experiencing homelessness, the severe influx of cortisol would weaken the placental enzyme's ability to convert it to cortisone, thus inundating the fetus with cortisol and leaving it susceptible to its damaging effects. Without a question about duration or frequency on the PRAMS questionnaire, nor for that matter on the perceived degree of disruption the stressor poses, we are left to assume the severity of exposure to circulating glucocorticoids. Chronic stress, or allostatic overload, and the correlation with adverse birth outcomes have been highly associated with ethnic minorities (Strutz et al., 2014). Accurately measuring the degree of stress a mother experiences would lend itself to better prevention and intervention methods. In this study, significance of fetal growth variables are diminished due to a lack of measurement of timing. If this study were to have that measurement, the relationships that I found would most likely be strengthened.

Poverty

This study used Medicaid as a proxy measure of poverty. The PRAMS questionnaire asks about income level in increments (*i.e.*, \$10,000 to \$14,999), which do not exactly match federal poverty guidelines, prohibiting a calculation of percentage of the federal poverty level. Using Medicaid as a measure of poverty is a limitation in that pregnancy extends coverage through “medically-needy” status, thus broadening the scope of poverty. On the other hand, in Tennessee, incurring medical bills through pregnancy allows an individual to “spend down” (subtract those bills from their income) to a lower income bracket, consequently placing them at a higher poverty level and qualifying them for Medicaid coverage, known as TennCare in Tennessee (185% FPL in 2009) (DHHS, 2009). Therefore, using TennCare as a measure of poverty is a limitation in that I was unable to assess the extent of poverty a woman was living in. However, it is also reasonable measure for several reasons.

First, income that is self-reported on surveys has consistently been proven to be subject to bias and error through under- or over-estimation and omission (Moore, Stinson, & Welniak, 2000). The source of payment for delivery was obtained from birth certificate records, making Medicaid a more reliable variable. Second, of those who were receiving Medicaid during their pregnancy, 86% had incomes less than \$25,000 a year. The 2009 Federal Poverty Guidelines for a family of four was \$22,050 (DHHS, 2009). While I was unable to get the household to income ratio, it seems logical that Medicaid is a more than reasonable measure of poverty in this study. Lastly, this mode of measuring poverty may include those living just above the poverty line, who often suffer just as greatly and yet do not always qualify for assistance (Rossi & Curtis, 2013).

Discussion

Two findings were of considerable importance. First, the latent class for stressors proved to have little significance as it was only statistically significant for delivering an infant small for gestational age when moderated by Medicaid status. While stressors appeared inconsequential in this study, perhaps due to using imperfect measures of stress, Medicaid status proved to be a positive predictor of all four adverse birth outcomes, confirming what research has repeatedly shown, that living in poverty has deleterious effects on health.

Second, African-American women had better birth outcomes at a younger age, corroborating Geronimus' concept of weathering on reproductive health. This finding, just as Geronimus shows, suggests that the African-American women in this study suffered from a disproportionate amount of stress compared to European-American women. This disproportionate level of stress is theorized to be a result of an overactive HPA axis that is unable to return the body to homeostasis. This allostatic load has deleterious effects on the body, including worse birth outcomes. However, at a younger age, particularly for African-Americans, the chronic wear and tear of an allostatic load has yet to take the same toll on the body, in particular the reproductive system, whereby giving birth at a younger age is more beneficial to the health of the infant. This, however, is only applicable to African-Americans who suffer the added stress of racism and discrimination present in our society. In this study, while this relationship did exist, it was weak, and may have been attenuated due to limitations already outlined.

Poverty is obviously of critical importance to understanding the root of poor fetal growth. It is more than a lack of money or material wealth; it is a situation of few resources and is inextricably linked with stress. Poverty has a well-documented connection with health status

through environmental and social-psychological factors, with lifelong exposure increasing rates of disease (Lupien, King, Meaney, & McEwen, 2001). A study correlating lifelong residence in low-income neighborhoods to low birthweight found that African-American women had a low birthweight incidence of 17% compared to the 10.1% incidence of white women with the same lifelong residence. Interestingly, African-Americans who had a lifelong residence in high-income neighborhoods still had a higher incidence of low birthweight (11.7%) than white women of low-income neighborhoods (Collins, Wambach, David, & Rankin, 2009). This attests to both the idea that fetal programming is an origin of intergenerational poverty and that weathering based on race is a root of poor birth outcomes.

However, both fetal programming and the concept of weathering are based on the function of the HPA axis during gestation, and this study was unable to conclude that prenatal stress plays an overtly influential role in birth outcomes. Why might this be the case? First, multiple factors that comprise socioeconomic status (*i.e.*, education, marital status, race) were controlled for, implying that stress is irrelevant outside the context of lived experience. Controlling for these factors also fails to separate things that may be inextricably linked.

Second, this study lacked a biological marker of stress to confirm activation of the HPA axis. Without a measure of cortisol we are left to assume dysregulation through self-reports of perceived life stress and to assume that these specific events provoke the same level of distress and stress across all women. These points combined strongly suggest the need for a better understanding of stress during pregnancy and the creation of innovative measures to accurately assess the effects of stressful life events on the developing fetus.

Implications

For Practice

Social workers in perinatal settings are tasked with the challenge of improving birth outcomes, responding to infants in need, supporting the parent-child relationship, and advocating for change within the health care setting (Lind & Bachman, 2012). Programs such as Healthy Start aim to reduce infant mortality, particularly among racial minorities, and efforts have been promising (Biermann, Dunlop, Brady, Dubin, & Brann, 2006).

A number of interventions hold promise both for reducing pregnant women's stress and for mitigating the effects of stress on their offspring. For example, research on the effectiveness of positive imagery and relaxation techniques on cortisol levels during pregnancy has shown some promise (Jallo, Ruiz, Elswick, & French, 2014; Chuang et al., 2012; Jallo, Bourguignon, Taylor, Ruiz & Goehler, 2008). Social support, particularly partner support, has proven to be especially salient in reducing stress prenatally (Stapleton et al., 2012). Social workers are thus critical to intervention at the personal level. Maternal warmth and care after birth can mitigate the negative effects stress plays on the fetus (Meaney, 2001), and thus parent education and support of positive parenting is imperative (Olds, 2002). Furthermore, the act of breastfeeding is associated with an attenuation of the HPA axis as well as the release of oxytocin, promoting warm maternal behavior (Heinrichs, Neumann, & Elhert, 2002). Breastfeeding could be a protective behavior to reduce stress and promote loving feelings towards their newborn. Social workers, thus, have a critical role in postpartum care as well.

However, providing women access to resources necessary to meet their daily needs remains the most imperative role of social workers in prenatal care settings (Alexander & Koltechuck, 2001). Poverty in this study proved to be the strongest predictor of negative birth

outcomes, therefore social workers have an obligation to impoverished women, particularly those who are pregnant.

For Policy

The 2014 Shriver Report from the Center for American Progress reported that one in three women in the United States live in poverty and that two-thirds of minimum wage workers are women with no paid sick days or leave. Furthermore, the average woman is paid 77 cents for every dollar a man makes with the figure much lower for African-American and Latina women at only 64 cents and 55 cents respectively (Shriver, 2014). Therefore, advocating for equal rights for women in the workplace is vital to decreasing the disproportionate rate of economic hardship experienced by women and thus incrementally reducing poverty.

Furthermore, based on estimates by the Institute of Medicine's Committee on Understanding Preterm Birth, the annual societal economic burden associated with premature delivery in the United States in 2005 alone was \$26.2 billion (Behrman & Butler, 2007). Globally, the United States rate of preterm birth ranks 131st out of 184 countries, many of them much more impoverished (Blencowe et al., 2013). Clearly we are failing at providing resources necessary to promote healthy birth outcomes. Advocating for change in policy related to the health of pregnant women is vital. Pushing for expansions in assistance policies during pregnancy and after birth, including income assistance, Medicaid policy, WIC policy, and maternity leave would be an adequate start.

The damaging effects of poverty and stress need to be better understood by policy makers in order to effectively benefit vulnerable populations. Currently, Temporary Assistance for Needy Families (TANF) enforces a 5 year limit to receiving aid and work must be in place within 2 years of that limit. This requirement can be detrimental to both mothers and children in

that working for low pay puts additional stress on the family, reduces quality time spent with children, and diverts income to work-related expenses like transportation and childcare (Lichter & Jayakody, 2002). Moreover, TANF allows states to impose family caps which deny additional cash benefits to women who give birth while receiving TANF. This policy, according to Romero, Kwan, and Chavkin (2013) violates several articles within varying international human and reproductive rights documents and is inherently discriminatory towards poor women and children. A greater number of dependents calls for an increase in assistance. Denying this simply perpetuates poverty, impacts children disproportionately, and creates greater need long-term. Assistance and intervention is most beneficial to the individual and society when implemented during pregnancy.

For Research

Poverty is a consistent area of research within social work and is a well-documented source of negative life events. This study demonstrated that poverty was the course through which stressful life events occurred. Evaluation of the way individuals living in poverty experience stress is needed. Furthermore, exploration of concepts of vulnerability and resistance is necessary to understanding populations most at risk during pregnancy. Identifying protective factors that contribute to resistance of stress in poverty is critical to furthering prenatal stress research.

Current research on prenatal stress suffers from methodological error in that there is no universal agreement on measurement, making cross-study comparison impractical and irrelevant (Monroe, 2008). Evaluation of the numerous paper and pencil methods of measuring stress and their correlation with biological markers of stress is imperative to the future of prenatal stress research, particularly within the social sciences. Social work research incorporates very little

from biological research and yet biological research is beginning to incorporate the social science measures. In order to remain a vital field, social work must adhere to evidence-based measures and look at collaborating across fields to accurately assess needs of the most vulnerable, in this case pregnant women.

Conclusions

Dysregulation of the stress response system during pregnancy programs a fetus for a lifetime risk of poor health and disease. Women living in poverty are particularly susceptible to heightened levels of stress and African-Americans suffer disproportionately. This study sought to prove these points but stumbled upon a greater methodological issue. Stress, as we currently measure it within the social sciences, is an inadequate representation and fails to account for its biological underpinnings. Collaboration between the biological and social sciences is needed now more than ever. Moreover, social work needs to integrate both much better into practice.

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Appendix

Table 1
Demographic Characteristics of Sample Population

Demographic	Weighted Percentage	Unweighted <i>N</i>	Weighted <i>N</i>	% Missing
Age group (yrs)				
<17	6.0	652	69,743	0.0
18-19	11.0			
20-24	26.7			
25-29	25.7			
30-34	18.2			
35-39	9.8			
40+	2.6			
Race				
White	79.7	652	69,743	0.0
Black	20.3			
Education (yrs)				
0-8	1.6	652	69,743	0.0
9-11	17.2			
12	33.0			
13-15	26.8			
>16	21.4			
Income				
< \$10,000	28.9	607	65,200	6.52
\$10,000-\$14,999	10.9			
\$15,000-\$19,999	6.8			
\$20,000-\$24,999	8.5			
\$25,000-\$34,999	10.8			
\$35,000-\$49,999	9.0			
>\$50,000	25.1			
Insurance				
Medicaid	58.8	629	67,112	3.8
Other	41.2			
Marital Status				
Married	54.7	652	69,743	0.0
Other	45.3			
Urban/Rural				
Urban	72.6	622	66,321	4.90
Rural	27.4			
Child's Sex				
Male	50.8	645	68,959	1.20
Female	49.2			

Table 2
Percentages Experiencing Individual Stressors

Stressors	Weighted % Yes	Unweighted <i>N</i>	Population <i>N</i>	% Missing Data	95% CI
Argue lots	26.6	649	69,312	0.62	22, 31
Couldn't pay bills	26.3	648	69,293	0.65	22, 31
Others died	20.5	647	69,274	0.68	17, 25
Others drugs	18.8	647	69,274	0.68	15, 23
Divorce	8.3	649	69,475	0.39	6, 12
Family member ill	30.7	649	69,467	0.40	26, 36
Physical fight	3.1	648	69,293	0.65	2, 5
Homeless	4.1	647	69,149	0.85	2, 7
Husband/partner in jail	5.7	648	69,293	0.65	4, 8
Husband/partner lost job	23.6	647	69,266	0.69	19, 28
Moved	43.1	650	69,490	0.36	38, 48
Husband/partner didn't want pregnancy	9.3	649	69,312	0.62	7, 13
Mom lost job	13.2	645	68,807	0.34	10, 17
Car crash injury	2.4	634	67,911	2.63	1, 5
Abuse by husband/partner (12 months before pregnancy)	2.9	612	64,481	7.54	2, 5
Abuse by husband/partner (during pregnancy)	3.2	611	64,251	7.87	2, 6
Pre-pregnancy ck/tx for depression/anxiety	17.0	645	68,454	1.85	13, 21

Note. Teenagers were not asked questions about abuse, therefore the percent of missing data is inflated. After accounting for the 33 individuals < 18 years of age, the population *N* for “abuse by husband/ partner (12 months before pregnancy)” was 64,228 and the % missing data was 2.1. For “abuse by husband/partner (during pregnancy)” the population *N* was 63,998 and the % of missing data was 2.4. Both 95% CI remained the same.

Table 3
Birth Outcomes for Sample Population

Birth Outcome	Weighted Percentage	Unweighted <i>N</i>	Weighted <i>N</i>	% Missing	95% CI
LGA					
Yes	6.2	613	68,638	1.60	4, 9
SGA (based on 10th %tile)					
Yes	8.7	613	68,638	1.60	6, 11
Preterm Birth (<37wks)					
Yes	10.8	645	68,959	1.20	9, 13
Problem Birth (LGA, SGA, or Preterm Birth)					
Yes	22.1	606	67,855	2.7	19, 26

Note. LGA = large for gestational age; SGA = small for gestational age.

Table 4

Latent Class Analysis Models

Number of Latent Classes	Number of Parameters Estimated	BIC	SSABIC	VLMRLRT	<i>p</i> -value	LMRALRT	<i>p</i> -value	Entropy
1	17	8299.47	8245.49	N/A	N/A	N/A	N/A	N/A
2	35	7900.35	7789.23	515.76	.058	511.37	.059	.686
3	53	7905.83	7737.55	111.16	.603	110.22	.606	.695
4	71	7948.23	7722.81	74.24	.652	73.604	.655	.751
5	89	7994.97	7712.39	69.91	.836	69.311	.836	.783

Note. BIC = Bayesian information criterion; SSABIC = sample-size adjusted Bayesian information criterion; VLMRLRT = Vuong-Lo-Mendell-Rubin likelihood ratio test; LMRALRT = Lo-Mendell-Rubin adjusted likelihood ratio test.

Table 5

Predictors of Overall Problematic Births

Predictors	<i>B</i>	<i>SE B</i>	<i>e^B</i>	<i>p</i>
Demographics & Medical ^a				
Medicaid	.425	.183	1.53	.021
Married	-.082	.186	0.92	.657
Education	-.003	.087	1.00	.970
African-American	-.148	.163	0.86	.366
Medical risk	.608	.168	1.84	.000
Age	.008	.014	1.01	.568
African-American X Age	.015	.025	1.02	.006
Stressors				
#Stressors ^b	.053	.031	1.05	.092
Stress Typology ^b	.082	.149	1.09	.584
Stressors X Medicaid				
#Stressors X Medicaid ^c	.089	.068	1.09	.191
Stress Typology X Medicaid ^d	.315	.317	1.37	.320

Note. e^B = odds ratio (OR) (confidence intervals not available); *p* is two-tailed.

^aResults obtained with only these predictors entered.

^bResults obtained with this predictor and demographic/medical predictors entered.

^cResults obtained with this predictor, #stressors, and demographic/ medical predictors entered.

^dResults obtained with this predictor, stress typology, and demographic/ medical predictors entered.

Table 6
Predictors of Preterm Birth

Predictors	<i>B</i>	<i>SE B</i>	e^B	<i>p</i>
Demographics & Medical ^a				
Medicaid	.607	.179	1.835	.001
Married	.056	.210	1.058	.790
Education	.041	.099	1.042	.681
African-American	.259	.183	1.296	.157
Medical risk	1.044	.174	2.841	.000
Age	.022	.016	1.022	.168
African-American X Age	-.011	.027	.989	.682
Stressors				
#Stressors ^b	.012	.033	1.012	.709
Stress Typology ^b	.151	.166	1.163	.364
Stressors X Medicaid				
#Stressors X Medicaid ^c	-.043	.068	.958	.524
Stress Typology X Medicaid ^d	.076	.371	1.079	.838

Note. e^B = odds ratio (OR) (confidence intervals not available); *p* is two-tailed.

^aResults obtained with only these predictors entered.

^bResults obtained with this predictor and demographic/medical predictors entered.

^cResults obtained with this predictor, #stressors, and demographic/ medical predictors entered.

^dResults obtained with this predictor, stress typology, and demographic/ medical predictors entered.

Table 7
Predictors of Large for Gestational Age

Predictors	<i>B</i>	<i>SE B</i>	<i>e^B</i>	<i>p</i>
Demographics & Medical ^a				
Medicaid	.795	.340	2.214	.019
Married	.097	.311	1.102	.754
Education	.245	.147	1.278	.096
African-American	-.903	.361	.405	.012
Medical risk	.399	.308	1.49	.194
Age	.009	.022	1.009	.682
African-American X Age	-.027	.103	.973	.794
Stressors				
#Stressors ^b	.021	.062	1.021	.735
Stress Typology ^b	.044	.285	1.045	.876
Stressors X Medicaid				
#Stressors X Medicaid ^c	.026	.198	1.026	.896
Stress Typology X Medicaid ^d	.161	.616	1.175	.794

Note. e^B = odds ratio (OR) (confidence intervals not available); *p* is two-tailed.

^aResults obtained with only these predictors entered.

^bResults obtained with this predictor and demographic/medical predictors entered.

^cResults obtained with this predictor, #stressors, and demographic/ medical predictors entered.

^dResults obtained with this predictor, stress typology, and demographic/ medical predictors entered.

Table 8
Predictors of Small for Gestational Age

Predictors	<i>B</i>	<i>SE B</i>	<i>e^B</i>	<i>p</i>
Demographics & Medical ^a				
Medicaid	-.140	.229	.869	.541
Married	-.173	.212	.841	.416
Education	-.135	.102	.874	.187
African-American	-.099	.183	.906	.589
Medical risk	.08	.155	1.083	.603
Age	-.021	.02	.979	.294
African-American X Age	.041	.026	1.042	.118
Stressors				
#Stressors ^b	.054	.034	1.055	.115
Stress Typology ^b	-.064	.166	.938	.699
Stressors X Medicaid				
#Stressors X Medicaid ^c	.156	.064	1.169	.014
Stress Typology X Medicaid ^d	.568	.308	1.765	.065

Note. e^B = odds ratio (OR) (confidence intervals not available); *p* is two-tailed.

^aResults obtained with only these predictors entered.

^bResults obtained with this predictor and demographic/medical predictors entered.

^cResults obtained with this predictor, #stressors, and demographic/ medical predictors entered.

^dResults obtained with this predictor, stress typology, and demographic/ medical predictors entered.

Table 9
Bivariate Predictors of Birth Outcomes

Predictors	Overall Problematic				Preterm				Small Gestational Age				Large Gestational Age			
	<i>B</i>	<i>SE B</i>	e^B	<i>p</i>	<i>B</i>	<i>SE B</i>	e^B	<i>p</i>	<i>B</i>	<i>SE B</i>	e^B	<i>p</i>	<i>B</i>	<i>SE B</i>	e^B	<i>p</i>
Medicaid	.375	.139	1.455	.007	.423	.138	1.527	.002	.181	.177	1.198	.306	.257	.222	1.293	.246
Married	-.177	.133	0.838	.185	-.133	.141	0.875	.344	-.259	.161	0.772	.108	.160	.217	1.174	.461
Education	-.083	.064	0.920	.195	-.035	.070	0.966	.618	-.184	.080	0.832	.021	.076	.104	1.079	.466
African-American	-.012	.147	0.988	.935	.311	.157	1.365	.047	-.084	.162	0.919	.605	-.729	.299	0.482	.015
Medical risk	.620	.166	1.859	.000	1.035	.171	2.815	.000	.014	.145	1.014	.922	.356	.256	1.428	.165
Age	.003	.010	1.003	.797	.017	.011	1.017	.109	-.027	.013	0.973	.040	.009	.016	1.009	.570
#Stressors	.072	.029	1.075	.013	.029	.029	1.029	.322	.076	.032	1.079	.017	.028	.047	1.028	.552
Stress Typology	.197	.135	1.218	.145	.207	.143	1.230	.146	.071	.160	1.074	.656	.101	.212	1.106	.636

Note. e^B = odds ratio (OR) (confidence intervals not available); *p* is two-tailed.

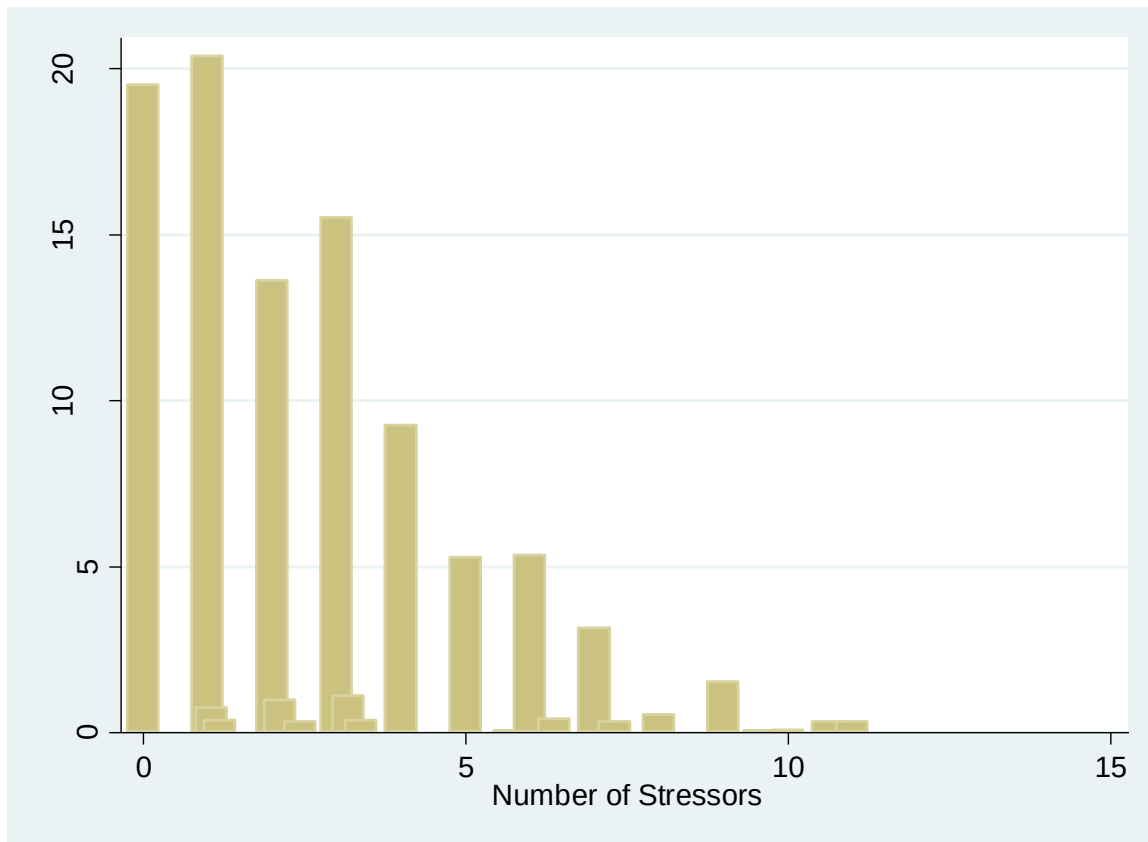


Figure 1
Number of Stressors Experienced

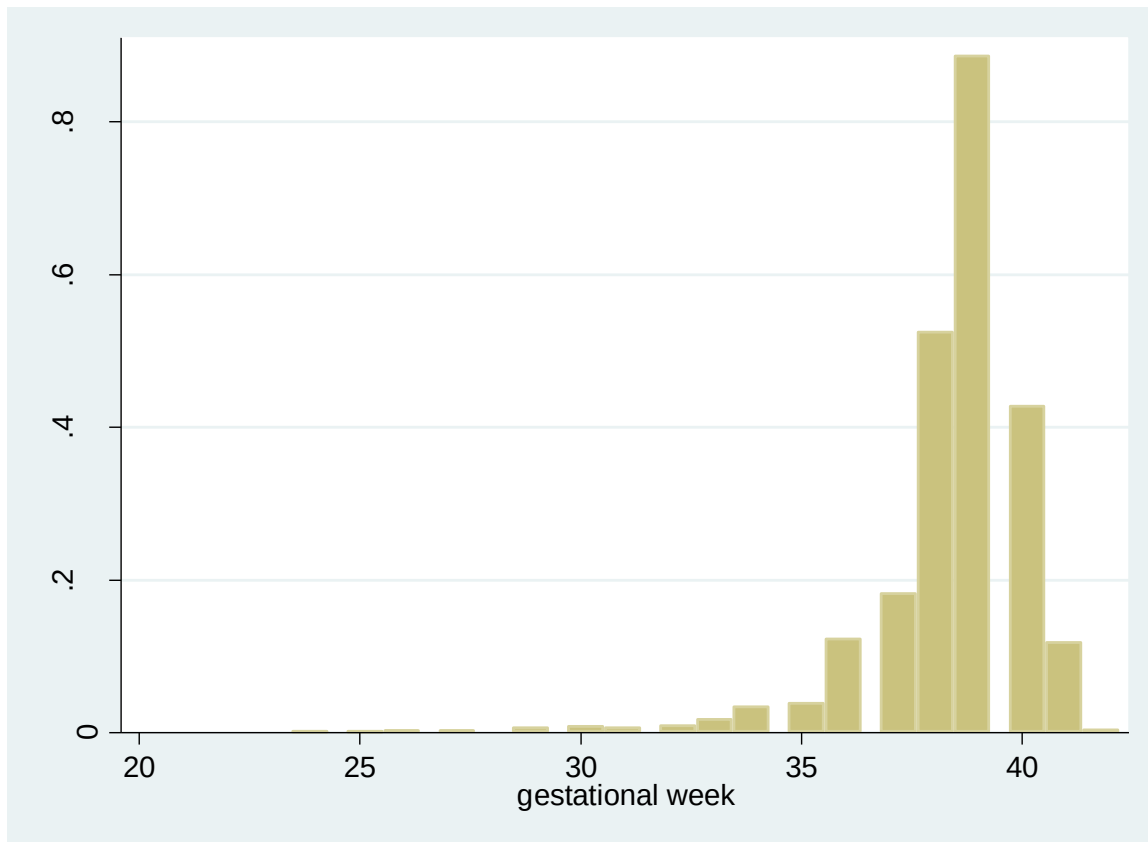


Figure 2
Gestational Age in Weeks

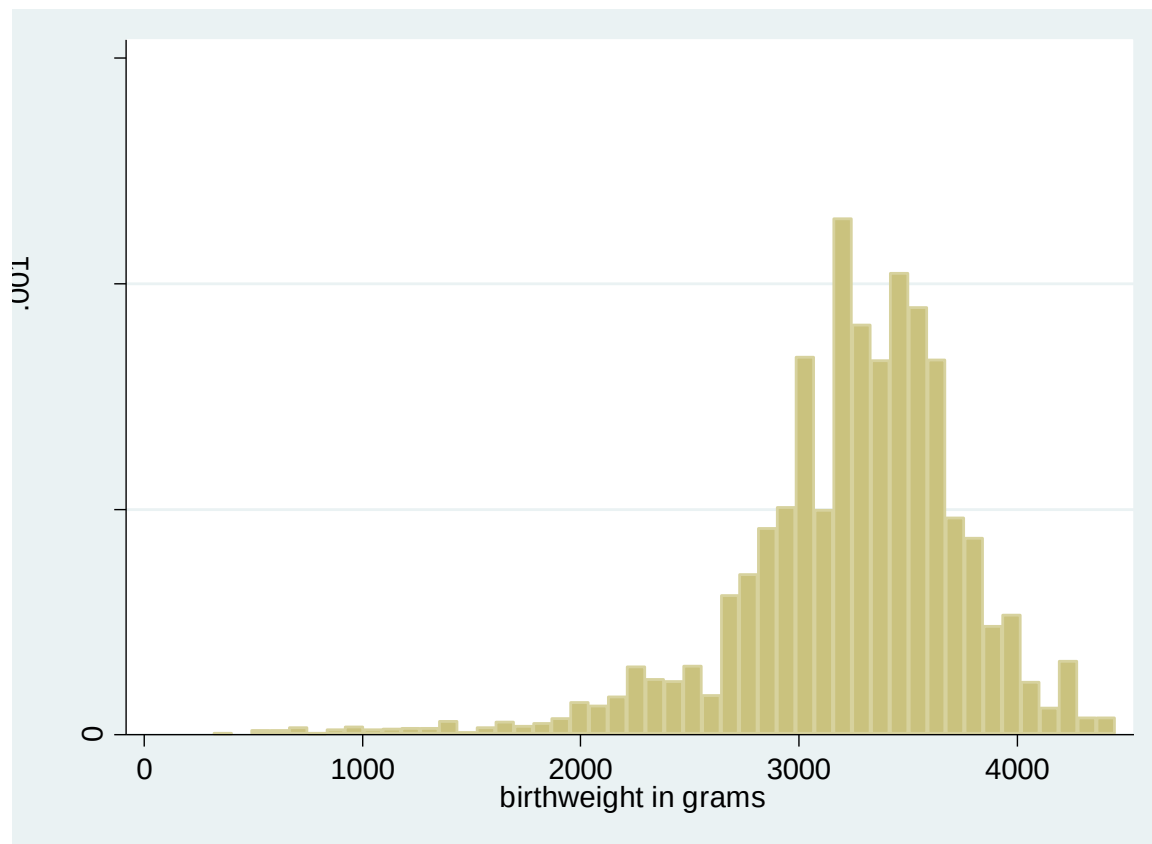


Figure 3
Unadjusted Birthweights in Grams

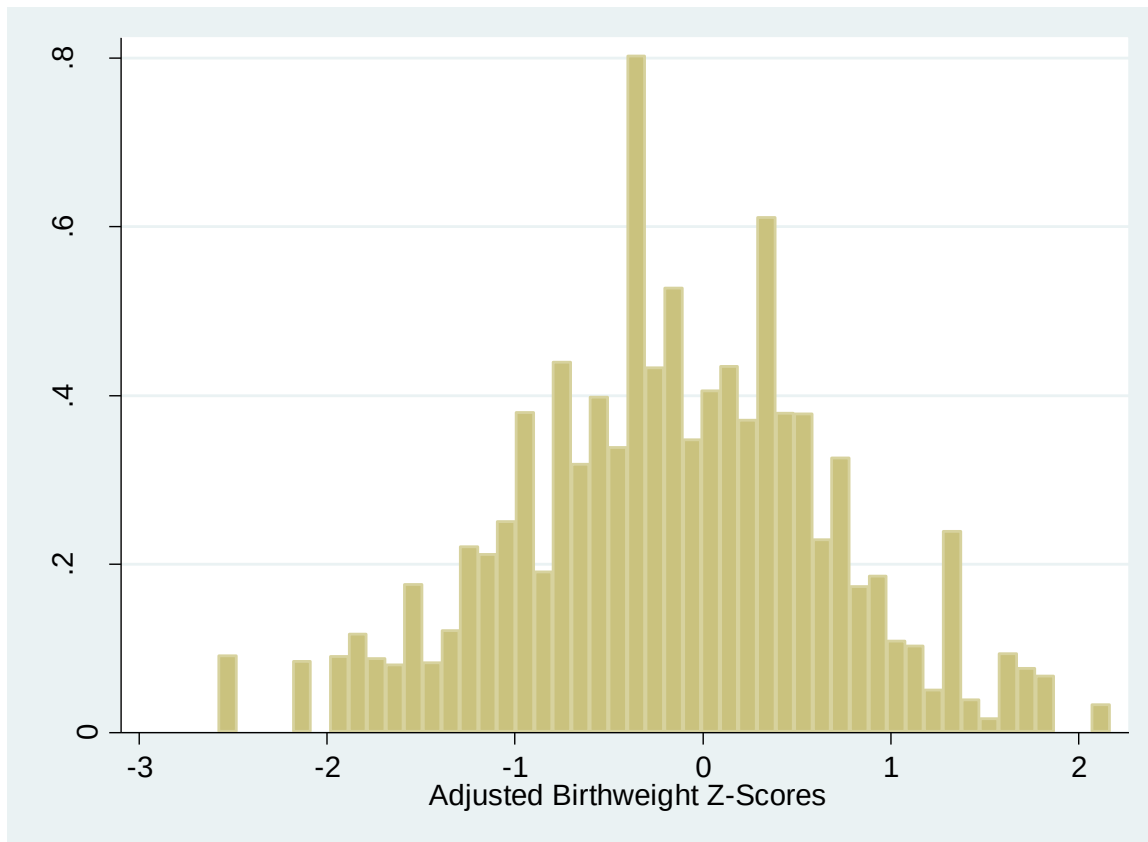


Figure 4
Adjusted Birthweight Z-Scores

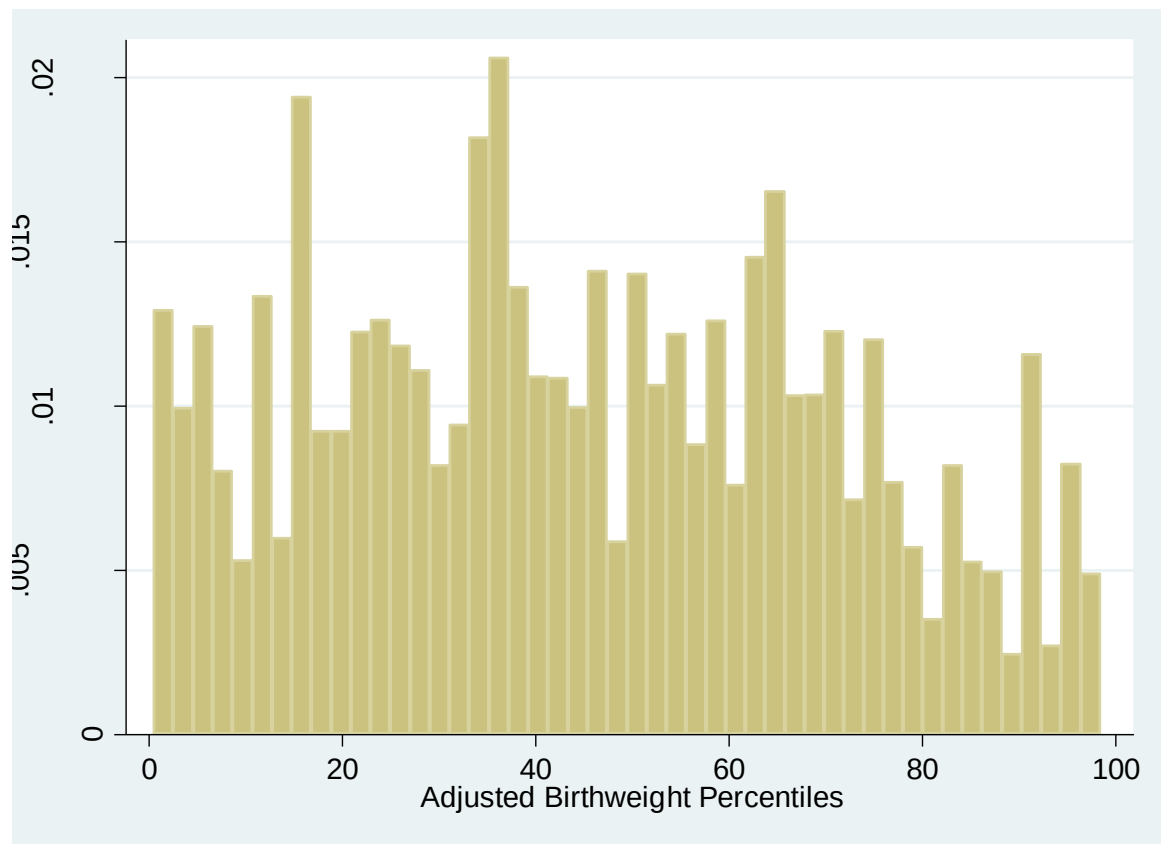


Figure 5
Adjusted Birthweight Percentiles

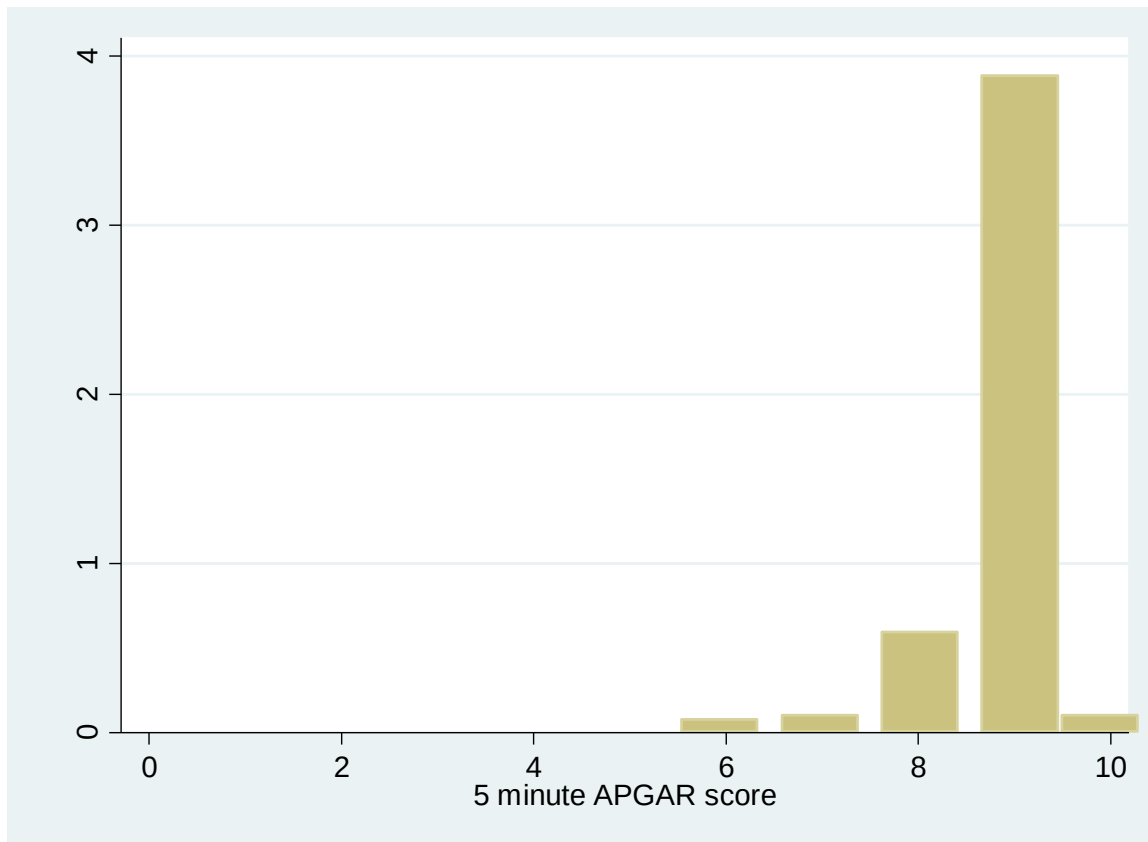


Figure 6
5-Minute APGAR Scores

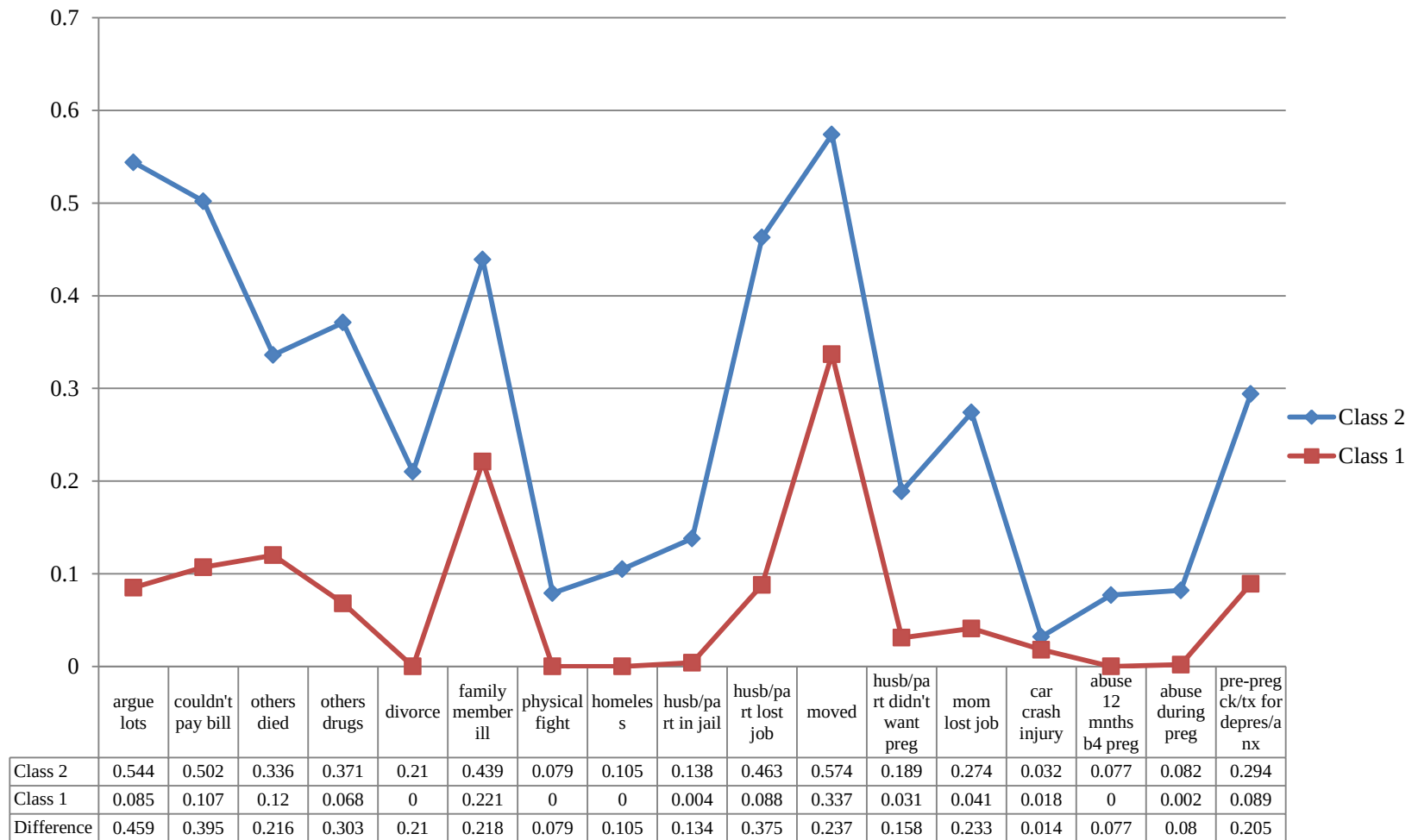


Figure 7
Proportion of Stressors Experienced by Class

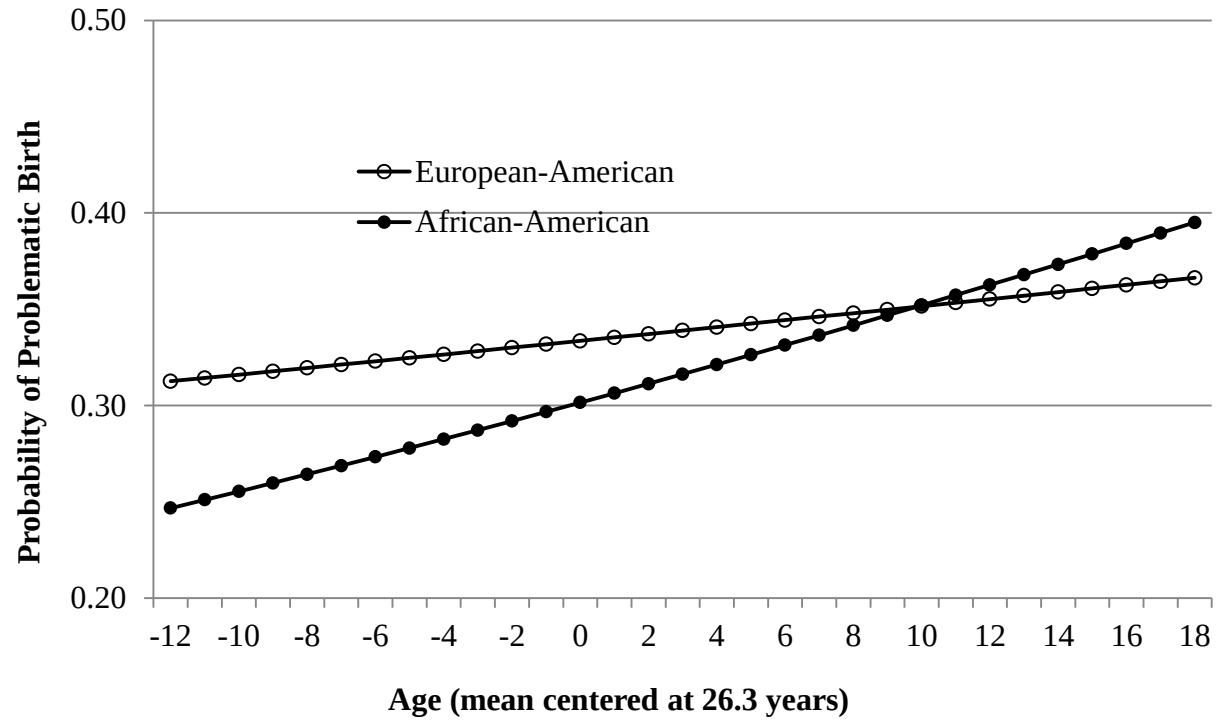


Figure 8
Interaction between Age and Problematic Birth by Race

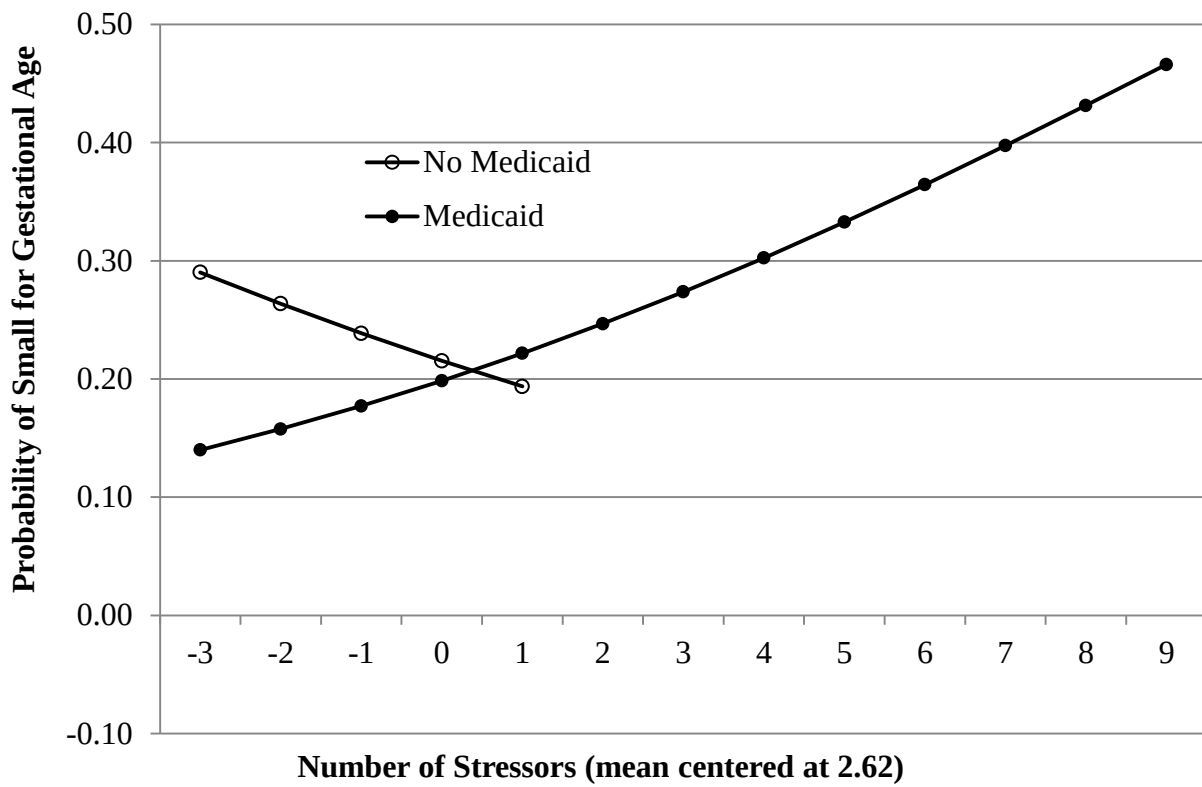


Figure 9
Interaction between Number of Stressors and SGA by Medicaid Status

Appendix A: PRAMS Questionnaire

Pregnancy Risk Assessment Monitoring System (PRAMS)

Phase 6 Core Questionnaire

1/28/09

First, we would like to ask a few questions about *you* and the time before you got pregnant with your new baby.

1. At any time during the 12 months before you got pregnant with your new baby, did you do any of the following things? For each item, circle **Y** (Yes) if you did it or **N** (No) if you did not.

	No	Yes
a. I was dieting (changing my eating habits) to lose weight	N	Y
b. I was exercising 3 or more days of the week.....	N	Y
c. I was regularly taking prescription medicines other than birth control.....	N	Y
d. I visited a health care worker to be checked or treated for diabetes	N	Y
e. I visited a health care worker to be checked or treated for high blood pressure.....	N	Y
f. I visited a health care worker to be checked or treated for depression or anxiety.....	N	Y
g. I talked to a health care worker about my family medical history	N	Y
h. I had my teeth cleaned by a dentist or dental hygienist.....	N	Y

2. During the month before you got pregnant with your new baby, were you covered by any of these health insurance plans? Check all that apply

Health insurance from your job or the job of your husband, partner, or parents

Health insurance that you or someone else paid for (not from a job)

Medicaid (or *state Medicaid name*)

TRICARE or other military health care

State-specific option (IHS, etc.)

State-specific option (state name for indigent care)

State-specific option (SCHIP or CHIP program name)

Other source(s) => Please tell us

[BOX]

I did not have any health insurance before I got pregnant

3. During the month before you got pregnant with your new baby, how many times a week did you take a multivitamin, a prenatal vitamin, or a folic acid vitamin?

I didn't take a multivitamins, prenatal vitamins, or folic acid vitamins at all

1 to 3 times a week

4 to 6 times a week

Every day of the week

Insertion point for Standard question(s) G

4. *Just before you got pregnant with your new baby, how much did you weigh?*

[BOX] Pounds OR [BOX] Kilos

5. **How tall are you without shoes?**

[BOX] Feet [BOX] Inches
OR [BOX] Meters

6. **What is your date of birth?**

[BOX] / [BOX] / [19]
Month Day Year

Insertion point for Standard question(s) L10

Insertion point for Standard question(s) L17, L18

7. **Before you got pregnant with your new baby, were you ever told by a doctor, nurse, or other health care worker that you had Type 1 or Type 2 diabetes?** This is not the same as gestational diabetes or diabetes that starts during pregnancy.

No
Yes

Insertion point for Standard question(s) L11

8. **Before you got pregnant with your new baby, did you ever have any other babies who were born alive?**

No ≡ Go to Question 11
Yes

9. **Did the baby born just before your new one weigh more than 5 pounds, 8 ounces (2.5 kilos) at birth?**

No
Yes

10. **Was the baby just before your new one born more than 3 weeks before his or her due date?**

No
Yes

Insertion point for Standard question FF4

Insertion point for Standard question K1

The next questions are about the time when you got pregnant with your new baby.

11. Thinking back to *just before* you got pregnant with your *new* baby, how did you feel about becoming pregnant? Check one answer

I wanted to be pregnant sooner
I wanted to be pregnant later
I wanted to be pregnant then
I didn't want to be pregnant then or at any time in the future

Insertion point for Standard question(s) Q4

12. When you got pregnant with your new baby, were you trying to get pregnant?

No
Yes ≡ **Go to Question 15**

Insertion point for Standard question(s) Q7

13. When you got pregnant with your new baby, were you or your husband or partner doing anything to keep from getting pregnant? (Some things people do to keep from getting pregnant include not having sex at certain times [natural family planning or rhythm] or withdrawal, and using birth control methods such as the pill, condoms, vaginal ring, IUD, having their tubes tied, or their partner having a vasectomy.)

No
Yes ≡ **Go to Question 15**

14. What were your reasons or your husband's or partner's reasons for not doing anything to keep from getting pregnant? Check all that apply

I didn't mind if I got pregnant
I thought I could not get pregnant at that time
I had side effects from the birth control method I was using
I had problems getting birth control when I needed it
I thought my husband or partner or I was sterile (could not get pregnant at all)
My husband or partner didn't want to use anything

Other ≡ Please tell us:

[BOX]

Insertion point for Standard question(s) E3

Insertion point for Standard question(s) A1–A2, A4–A5

The next questions are about the prenatal care you received during your most recent pregnancy. Prenatal care includes visits to a doctor, nurse, or other health care worker before your baby was born to get checkups and advice about pregnancy. (It may help to look at the calendar when you answer these questions.)

15. How many weeks or months pregnant were you when you were sure you were pregnant? (For example, you had a pregnancy test or a doctor or nurse said you were pregnant.)

[BOX] Weeks OR [BOX] Months

I don't remember

16. How many weeks or months pregnant were you when you had your first visit for prenatal care? Do not count a visit that was only for a pregnancy test or only for WIC (the Special Supplemental Nutrition Program for Women, Infants, and Children).

[BOX] Weeks OR [BOX] Months

I didn't go for prenatal care → **Go to Question 18**

17. Did you get prenatal care as early in your pregnancy as you wanted?

No

Yes → **Go to Question 19**

18. Did any of these things keep you from getting prenatal care at all or as early as you wanted? For each item, circle **T** (True) if it was a reason that you didn't get prenatal care when you wanted or circle **F** (False) if it was not a reason for you or if something does not apply to you

	True	False
a. I couldn't get an appointment when I wanted one	T	F
b. I didn't have enough money or insurance to pay for my visits	T	F
c. I had no transportation to get to the clinic or doctor's office.....	T	F
d. The doctor or my health plan would not start care as early as I wanted	T	F
e. I had too many other things going on	T	F
f. I couldn't take time off from work or school.....	T	F
g. I didn't have my Medicaid (or <i>state Medicaid name</i>) card.....	T	F
h. I had no one to take care of my children.....	T	F
i. I didn't know that I was pregnant.....	T	F
j. I didn't want anyone else to know I was pregnant.....	T	F
k. I didn't want prenatal care	T	F

If you did not go for prenatal care, go to Page #, Question 21.

Insertion point for Standard question(s) R1

19. Did any of these health insurance plans help you pay for your *prenatal care*? Check all that apply

Health insurance from your job or the job of your husband, partner, or parents

Health insurance that you or someone else paid for (not from a job)

Medicaid (or *state Medicaid name*)

TRICARE or other military health care

State-specific option (IHS, or tribal/state name)

State-specific option (state name for indigent care)

State-specific option (CHIP or SCHIP program)

Other source(s) = Please tell us:

[BOX]

I did not have health insurance to help pay for my prenatal care

20. 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? Please count only discussions, not reading materials or videos. For each item, circle **Y** (Yes) if someone talked with you about it or circle **N** (No) if no one talked with you about it.

	No	Yes
a. How smoking during pregnancy could affect my baby	N	Y
b. Breastfeeding my baby	N	Y
c. How drinking alcohol during pregnancy could affect my baby.....	N	Y
d. Using a seat belt during my pregnancy.....	N	Y
e. Medicines that are safe to take during my pregnancy.....	N	Y
f. How using illegal drugs could affect my baby	N	Y
g. Doing tests to screen for birth defects or diseases that run in my family.....	N	Y
h. The signs and symptoms of preterm labor (labor more than 3 weeks before the baby is due)....	N	Y
i. What to do if my labor starts early.....	N	Y
j. Getting tested for HIV (the virus that causes AIDS)	N	Y

- | | | |
|--|---|---|
| k. What to do if I feel depressed during my pregnancy or after my baby is born | N | Y |
| l. Physical abuse to women by their husbands or partners..... | N | Y |

Insertion point for Standard question(s) R1

Insertion point for Standard question(s) R3, R4, R18, R5

Insertion point for Standard question(s) R12, R2, R17, R16

Insertion point for Standard question(s) K4, R13

Insertion point for Standard question(s) R14

Insertion point for Standard question(s) R9–R11

Insertion point for Standard question(s) R6–R8

21. At any time during your most recent pregnancy or delivery, did you have a test for HIV (the virus that causes AIDS)?

No
Yes
I don't know

Insertion point for Standard question(s) I7

Insertion point for Standard question(s) I4–I6

Insertion point for Standard question(s) I2–I3

Insertion point for Standard question(s) G5

Insertion point for Standard question(s) G1–G4

Insertion point for Standard question(s) L12–L15

22. During your most recent pregnancy, were you on WIC (the Special Supplemental Nutrition Program for Women, Infants, and Children)?

No
Yes

Insertion point for Standard question(s) B7-B8

23. During your most recent pregnancy, were you told by a doctor, nurse, or other health care worker that you had gestational diabetes (diabetes that started during this pregnancy)?

No
Yes

Insertion point for Standard question(s) N7, N6

24. Did you have any of the following problems during your most recent pregnancy? For each item, circle **Y** (Yes) if you had the problem or circle **N** (No) if you did not.

	No	Yes
a. Vaginal bleeding	N	Y
b. Kidney or bladder (urinary tract) infection	N	Y
c. Severe nausea, vomiting, or dehydration	N	Y
d. Cervix had to be sewn shut (cerclage for incompetent cervix)	N	Y
e. High blood pressure, hypertension (including pregnancy-induced hypertension [PIH]), preeclampsia, or toxemia.....	N	Y
f. Problems with the placenta (such as abruptio placentae or placenta previa)	N	Y
g. Labor pains more than 3 weeks before my baby was due (preterm or early labor)	N	Y
h. Water broke more than 3 weeks before my baby was due (premature rupture of membranes [PROM])	N	Y
i. I had to have a blood transfusion	N	Y
j. I was hurt in a car accident	N	Y

Insertion point for Standard Question(s) N8, N5

Insertion point for Standard question(s) N1–N4

Insertion point for Standard question(s) L4–L7

The next questions are about smoking cigarettes around the time of pregnancy (before, during, and after).

25. Have you smoked any cigarettes in the *past 2 years*?

No ≡ **Go to Question 29**

Yes

26. In the *3 months before* you got pregnant, how many cigarettes did you smoke on an average day?
(A pack has 20 cigarettes.)

41 cigarettes or more

21 to 40 cigarettes

11 to 20 cigarettes

6 to 10 cigarettes

1 to 5 cigarettes

Less than 1 cigarette

I didn't smoke then

27. In the *last 3 months* of your pregnancy, how many cigarettes did you smoke on an average day?
(A pack has 20 cigarettes.)

41 cigarettes or more

21 to 40 cigarettes

11 to 20 cigarettes

6 to 10 cigarettes

1 to 5 cigarettes

Less than 1 cigarette

I didn't smoke then

Insertion point for Standard question AA6

Insertion point for Standard question AA1

28. How many cigarettes do you smoke on an average day *now*? (A pack has 20 cigarettes.)

41 cigarettes or more

21 to 40 cigarettes

11 to 20 cigarettes

6 to 10 cigarettes

1 to 5 cigarettes

Less than 1 cigarette

I don't smoke now

29. Which of the following statements best describes the rules about smoking *inside* your home *now*?

Check one answer

No one is allowed to smoke anywhere inside my home
Smoking is allowed in some rooms or at some times
Smoking is permitted anywhere inside my home

Insertion point for Standard question U1-U2

**The next questions are about drinking alcohol around the time of pregnancy
(before, during, and after).**

30. Have you had any alcoholic drinks in the past 2 years? A drink is 1 glass of wine, wine cooler, can or bottle of beer, shot of liquor, or mixed drink.

No = **Go to Question 33**
Yes

31a. During the 3 months before you got pregnant, how many alcoholic drinks did you have in an average week?

14 drinks or more a week
7 to 13 drinks a week
4 to 6 drinks a week
1 to 3 drinks a week
Less than 1 drink a week
I didn't drink then => **Go to Question 32a**

31b. During the 3 months before you got pregnant, how many times did you drink 4 alcoholic drinks or more in one sitting? A sitting is a two hour time span.

6 or more times
4 to 5 times
2 to 3 times
1 time
I didn't have 4 drinks or more in 1 sitting

32a. During the last 3 months of your pregnancy, how many alcoholic drinks did you have in an average week?

14 drinks or more a week

7 to 13 drinks a week
 4 to 6 drinks a week
 1 to 3 drinks a week
 Less than 1 drink a week
 I didn't drink then => **Go to Question 33**

32b. During the last 3 months of your pregnancy, how many times did you drink 4 alcoholic drinks or more in one sitting? A sitting is a two hour time span.

6 or more times
 4 to 5 times
 2 to 3 times
 1 time
 I didn't have 4 drinks or more in 1 sitting

Pregnancy can be a difficult time for some women. These next questions are about things that may have happened before and during your most recent pregnancy.

33. This question is about things that may have happened during the 12 months before your new baby was born. For each item, circle **Y** (Yes) if it happened to you or circle **N** (No) if it did not. (It may help to look at the calendar when you answer these questions.)

	No	Yes
a. A close family member was very sick and had to go into the hospital	N	Y
b. I got separated or divorced from my husband or partner	N	Y
c. I moved to a new address.....	N	Y
d. I was homeless.....	N	Y
e. My husband or partner lost his job	N	Y
f. I lost my job even though I wanted to go on working	N	Y
g. I argued with my husband or partner more than usual.....	N	Y
h. My husband or partner said he didn't want me to be pregnant	N	Y
i. I had a lot of bills I couldn't pay.....	N	Y
j. I was in a physical fight	N	Y
k. My husband or partner or I went to jail	N	Y
l. Someone very close to me had a problem with drinking or drugs.....	N	Y
m. Someone very close to me died	N	Y

Insertion point for Standard question(s) P14, P17, P15–P16

Insertion point for Standard question(s) BB1

34. During the 12 months before you got pregnant with your new baby, did your husband or partner push, hit, slap, kick, choke, or physically hurt you in any other way?

No
Yes

Insertion point for Standard question(s) Z5, Z3, Z7

35. During your most recent pregnancy, did your husband or partner push, hit, slap, kick, choke, or physically hurt you in any other way?

No
Yes

Insertion point for Standard question(s) Z6, Z4

The next questions are about your labor and delivery. (It may help to look at the calendar when you answer these questions.)

36. When was your baby due?

[BOX] / [BOX] / 20__ [BOX]
Month Day Year

37. When did you go into the hospital to have your baby?

[BOX] / [BOX] / 20__ [BOX]
Month Day Year

I didn't have my baby in a hospital

Insertion point for Standard question(s) K5

38. When was your baby born?

[BOX] / [BOX] / 20__ [BOX]
Month Day Year

Insertion point for Standard question(s) K9-K10

Insertion point for Standard question(s) K8, K3, K7, K6

39. When were you discharged from the hospital after your baby was born?

[BOX] / [BOX] / 20__ [BOX]
Month Day Year

I didn't have my baby in a hospital

Insertion point for Standard question(s) II1

40. Did any of these health insurance plans help you pay for the *delivery* of your new baby? Check all that apply

Health insurance from your job or the job of your husband, partner, or parents

Health insurance that you or someone else paid for (not from a job)

Medicaid (or *state Medicaid name*)

TRICARE or other military health care

State-specific option (IHS, or tribal/state name)

State-specific option (state name for indigent care)

State-specific option (SCHIP or CHIP program)

Other source(s) = Please tell us:

[BOX]

I did not have health insurance to help pay for my delivery

The next questions are about the time since your new baby was born.

41. After your baby was born, was he or she put in an intensive care unit?

No

Yes

I don't know

42. After your baby was born, how long did he or she stay in the hospital?

Less than 24 hours (less than 1 day)

24 to 48 hours (1 to 2 days)

3 to 5 days

6 to 14 days

More than 14 days

My baby was not born in a hospital

My baby is still in the hospital = **Go to Question 45**

Insertion point for Standard question(s) K11, K12

43. Is your baby alive now?

No ≡ **Go to Question 51**
Yes

44. Is your baby living with you now?

No ≡ **Go to Question 51**
Yes

Insertion point for Standard question(s) B4

45. Did you ever breastfeed or pump breast milk to feed your new baby after delivery, even for a short period of time?

No ≡ **Go to Question 4**
Yes

Insertion point for Standard question(s) B1

46. Are you currently breastfeeding or feeding pumped milk to your new baby?

No
Yes ≡ **Go to Question 48a**

47. How many weeks or months did you breastfeed or pump milk to feed your baby?

[BOX] Weeks OR [BOX] Months

Less than 1 week

Insertion point for Standard question(s) B2

Insertion point for Standard question(s) B3

48a. How old was your new baby the first time he or she drank liquids other than breast milk (such as formula, water, juice, tea, or cow's milk)?

[BOX] Weeks OR [BOX] Months

My baby was less than 1 week old

My baby has not had any liquids other than breast milk

48b. How old was your new baby the first time he or she ate food (such as baby cereal, baby food, or any other food)?

[BOX] Weeks OR [BOX] Months

My baby was less than 1 week old

My baby has not eaten any foods

Insertion point for Standard question(s) B5–B6

If your baby is still in the hospital, go to Page ##, Question 51.
--

49. In which *one* position do you most often lay your baby down to sleep now? Check one answer

On his or her side

On his or her back

On his or her stomach

Insertion point for Standard question(s) F1, F3

50. Was your new baby seen by a doctor, nurse, or other health care worker for a one week check-up after he or she was born?

No

Yes

Insertion point for Standard question(s) X6

Insertion point for Standard Question(s) X9

Insertion point for Standard question(s) X7

Insertion point for Standard question(s) X8

Insertion point for Standard question(s) X4

Insertion point for Standard question(s) X1–X2

Insertion point for Standard question(s) X3

Insertion point for Standard question(s) X5

Insertion point for Standard question(s) T4–T5

Insertion point for Standard question(s) T1, T3, T2

Insertion point for Standard question(s) H1–H4

51. Are you or your husband or partner doing anything *now* to keep from getting pregnant? (Some things people do to keep from getting pregnant include not having sex at certain times [natural family planning or rhythm] or withdrawal, and using birth control methods such as the pill, condoms, vaginal ring, IUD, having their tubes tied, or their partner having a vasectomy.)

No

Yes ≡ **Go to Question 53**

52. What are your reasons or your husband's or partner's reasons for not doing anything to keep from getting pregnant *now*? Check all that apply

I am not having sex

I want to get pregnant

I don't want to use birth control

My husband or partner doesn't want to use anything

I don't think I can get pregnant (sterile)

I can't pay for birth control

I am pregnant now

Other ≡ Please tell us:

[BOX]

Insertion point for Standard question(s) E1

Insertion point for Standard question(s) E2

Insertion point for Standard question(s) L8–L9

Insertion point for Standard question(s) L16

Insertion point for Standard question(s) O1–O3

53. Below is a list of feelings and experiences that women sometimes have after childbirth. Read each item to determine how well it describes your feelings and experiences. Then, write on the line the number of the choice that best describes how often you have felt or experienced things this way since your new baby was born. Use the scale when answering:

1	2	3	4	5
Never	Rarely	Sometimes	Often	Always
a. I felt down, depressed, or sad		[BOX]		
b. I felt hopeless		[BOX]		
c. I felt slowed down		[BOX]		

Insertion point for Standard question(s) M12

The next questions are on a variety of topics.

[STATE-SPECIFIC SECTION (Standards without insertion points and state-developed questions)]

The last questions are about the time during the 12 months before your new baby was born.

Insertion point for Standard Question(s) P18

53. During the 12 months before your new baby was born, what was your yearly total household income before taxes? Include your income, your husband's or partner's income, and any other income you may have received. (All information will be kept private and will not affect any services you are now getting.)

Less than \$10,000
\$10,000 to \$14,999
\$15,000 to \$19,999
\$20,000 to \$24,999
\$25,000 to \$34,999
\$35,000 to \$49,999
\$50,000 or more

Note: States can add additional categories as long as the categories are collapsible back to the existing core categories (i.e. may add upper or lower ranges beyond what is provided or split out existing categories into sub-categories)

54. During the 12 months before your new baby was born, how many people, including yourself, depended on this income?

[BOX] People

55. What is today's date?

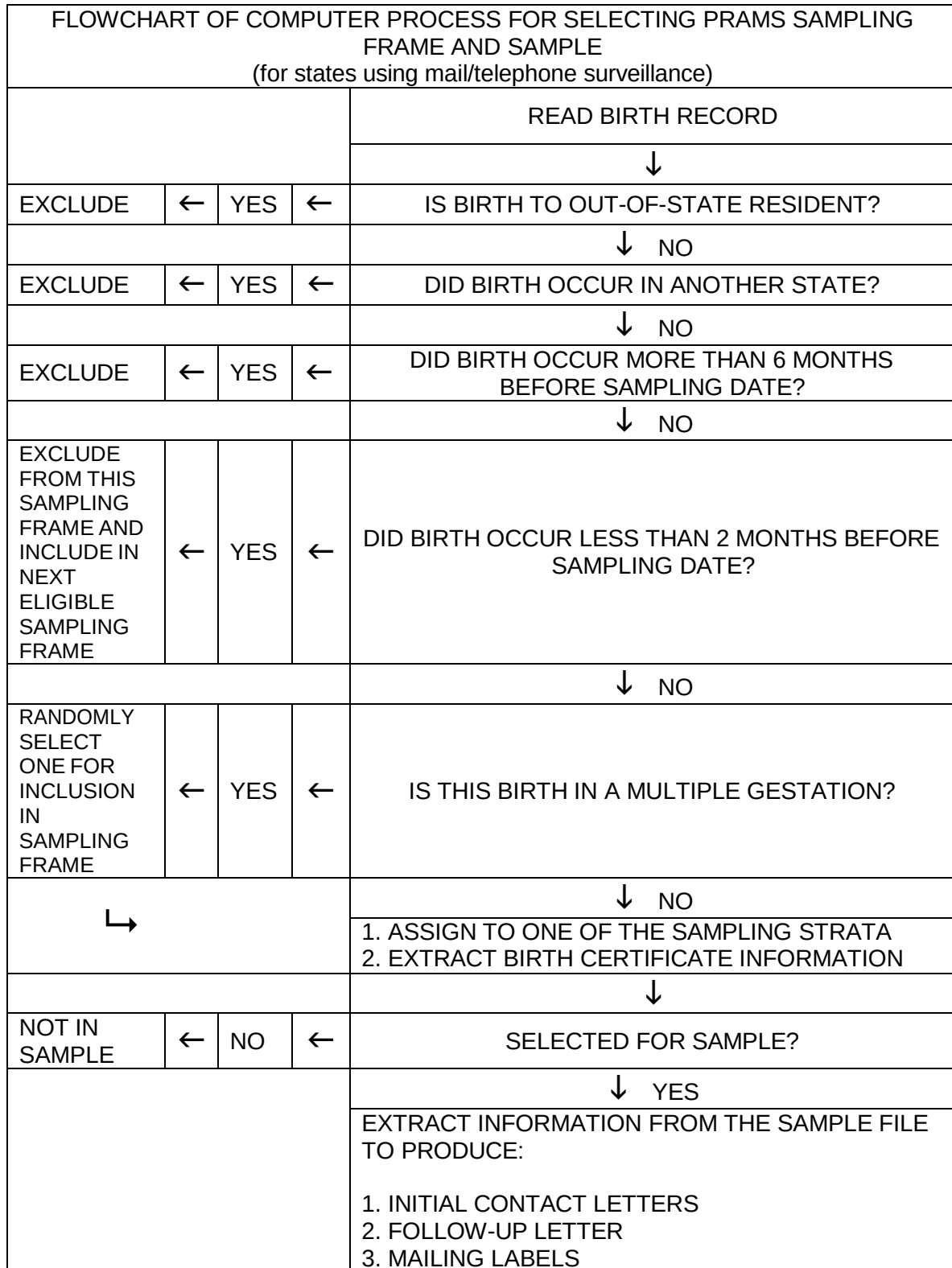
[BOX] / [BOX] / 20__ [BOX]
Month Day Year

Please use this space for any additional comments you would like to make about
the health of mothers and babies in State.

Thanks for answering our questions!

Your answers will help us work to make State mothers and babies healthier.

Appendix B: PRAMS Sampling Frame



Appendix C: PRAMS Non-Response Methodology

Nonresponse adjustment factors attempt to compensate for the tendency of women having certain characteristics (such as being unmarried or of lower education) to respond at lower rates than women without those characteristics. Where multivariate analysis shows that these characteristics affect the propensity to respond in a particular stratum, the adjustment factor is the ratio of the sample size in that category to the number of respondents in the category. If analysis shows that no characteristic distinguishes respondents from nonrespondents, the adjustment factor is the ratio of the sample size in that stratum to the number of respondents in the stratum. In the first case, each category so identified has an adjustment factor; in the second, there is a single factor for the whole stratum.

The rationale for applying nonresponse weights is the assumption that nonrespondents would have provided similar answers, on average, to respondents' answers for that stratum and adjustment category. So that cells with few respondents are not distorted by a few women's answers, small categories are collapsed until each cell contains at least 25 respondents. The magnitude of the adjustment for nonresponse depends on the response rate for a category. If 80% (or 4/5) of the women in a category respond, the nonresponse weight is 1.2 (or 5/4). Categories with lower response rates have higher nonresponse weights.

Frame omission studies are carried out to look for problems that occur during frame construction. The frame noncoverage weights are derived by comparing frame files for a year of births to the calendar year birth tape that states provided to CDC. Omitted records are usually due to late processing and are evenly scattered across the state, but sometimes they are clustered by particular hospitals or counties or even times of the year. The effect of the noncoverage weights is to bring totals estimated from sample data in line with known totals from the birth tape. In mail/telephone surveillance, the magnitude of noncoverage is small (typically from 1% to 5%), so the adjustment factor for noncoverage is not much greater than 1.

Multiplying together the sampling, nonresponse, and noncoverage components of the weight yields the analysis weight. The weight can be interpreted as the number of women like herself in the population that each respondent represents.

Appendix D: Example of U.S. Birth Certificate

U.S. STANDARD CERTIFICATE OF LIVE BIRTH			
LOCAL FILE NO.			BIRTH NUMBER:
C H I L D	1. CHILD'S NAME (First, Middle, Last, Suffix)	2. TIME OF BIRTH (24 hr)	3. SEX
	5. FACILITY NAME (If not institution, give street and number)	6. CITY, TOWN, OR LOCATION OF BIRTH	7. COUNTY OF BIRTH
M O T H E R	8a. MOTHER'S CURRENT LEGAL NAME (First, Middle, Last, Suffix)	8b. DATE OF BIRTH (Mo/Day/Yr)	
	8c. MOTHER'S NAME PRIOR TO FIRST MARRIAGE (First, Middle, Last, Suffix)	8d. BIRTHPLACE (State, Territory, or Foreign Country)	
	9a. RESIDENCE OF MOTHER-STATE	9b. COUNTY	9c. CITY, TOWN, OR LOCATION
	9d. STREET AND NUMBER	9e. APT. NO.	9f. ZIP CODE
			9g. INSIDE CITY LIMITS? ☐ Yes ☐ No
F A T H E R	10a. FATHER'S CURRENT LEGAL NAME (First, Middle, Last, Suffix)	10b. DATE OF BIRTH (Mo/Day/Yr)	10c. BIRTHPLACE (State, Territory, or Foreign Country)
C E R T I F I E R	11. CERTIFIER'S NAME: TITLE: ☐ MD ☐ DO ☐ HOSPITAL ADMIN. ☐ CNM/CM ☐ OTHER MIDWIFE ☐ OTHER (Specify) _____	12. DATE CERTIFIED ____/____/____ MM DD YYYY	13. DATE FILED BY REGISTRAR ____/____/____ MM DD YYYY
INFORMATION FOR ADMINISTRATIVE USE			
M O T H E R	14. MOTHER'S MAILING ADDRESS: 9 Same as residence, or: State: _____ City, Town, or Location: _____ Street & Number: _____ Apartment No.: _____ Zip Code: _____		
	15. MOTHER MARRIED? (At birth, conception, or any time between) IF NO, HAS PATERNITY ACKNOWLEDGEMENT BEEN SIGNED IN THE HOSPITAL? ☐ Yes ☐ No	16. SOCIAL SECURITY NUMBER REQUESTED FOR CHILD? ☐ Yes ☐ No	17. FACILITY ID. (NPI)
	18. MOTHER'S SOCIAL SECURITY NUMBER: _____ 19. FATHER'S SOCIAL SECURITY NUMBER: _____		
INFORMATION FOR MEDICAL AND HEALTH PURPOSES ONLY			
M O T H E R	20. MOTHER'S EDUCATION (Check the box that best describes the highest degree or level of school completed at the time of delivery) ☐ 8th grade or less ☐ 9th - 12th grade, no diploma ☐ High school graduate or GED completed ☐ Some college credit but no degree ☐ Associate degree (e.g., AA, AS) ☐ Bachelor's degree (e.g., BA, AB, BS) ☐ Master's degree (e.g., MA, MS, MEng, MEd, MSW, MBA) ☐ Doctorate (e.g., PhD, EdD) or Professional degree (e.g., MD, DDS, DVM, LLB, JD)	21. MOTHER OF HISPANIC ORIGIN? (Check the box that best describes whether the mother is Spanish/Hispanic/Latina. Check the "No" box if mother is not Spanish/Hispanic/Latina) ☐ No, not Spanish/Hispanic/Latina ☐ Yes, Mexican, Mexican American, Chicana ☐ Yes, Puerto Rican ☐ Yes, Cuban ☐ Yes, other Spanish/Hispanic/Latina (Specify) _____	22. MOTHER'S RACE (Check one or more races to indicate what the mother considers herself to be) ☐ White ☐ Black or African American ☐ American Indian or Alaska Native (Name of the enrolled or principal tribe) _____ ☐ Asian Indian ☐ Chinese ☐ Filipino ☐ Japanese ☐ Korean ☐ Vietnamese ☐ Other Asian (Specify) _____ ☐ Native Hawaiian ☐ Guamanian or Chamorro ☐ Samoan ☐ Other Pacific Islander (Specify) _____ ☐ Other (Specify) _____
F A T H E R	23. FATHER'S EDUCATION (Check the box that best describes the highest degree or level of school completed at the time of delivery) ☐ 8th grade or less ☐ 9th - 12th grade, no diploma ☐ High school graduate or GED completed ☐ Some college credit but no degree ☐ Associate degree (e.g., AA, AS) ☐ Bachelor's degree (e.g., BA, AB, BS) ☐ Master's degree (e.g., MA, MS, MEng, MEd, MSW, MBA) ☐ Doctorate (e.g., PhD, EdD) or Professional degree (e.g., MD, DDS, DVM, LLB, JD)	24. FATHER OF HISPANIC ORIGIN? (Check the box that best describes whether the father is Spanish/Hispanic/Latino. Check the "No" box if father is not Spanish/Hispanic/Latino) ☐ No, not Spanish/Hispanic/Latino ☐ Yes, Mexican, Mexican American, Chicano ☐ Yes, Puerto Rican ☐ Yes, Cuban ☐ Yes, other Spanish/Hispanic/Latino (Specify) _____	25. FATHER'S RACE (Check one or more races to indicate what the father considers himself to be) ☐ White ☐ Black or African American ☐ American Indian or Alaska Native (Name of the enrolled or principal tribe) _____ ☐ Asian Indian ☐ Chinese ☐ Filipino ☐ Japanese ☐ Korean ☐ Vietnamese ☐ Other Asian (Specify) _____ ☐ Native Hawaiian ☐ Guamanian or Chamorro ☐ Samoan ☐ Other Pacific Islander (Specify) _____ ☐ Other (Specify) _____
Mother's Name			
Mother's Medical Record No.			
	26. PLACE WHERE BIRTH OCCURRED (Check one) ☐ Hospital ☐ Freestanding birthing center ☐ Home Birth: Planned to deliver at home? 9 Yes 9 No ☐ Clinic/Doctor's office ☐ Other (Specify) _____	27. ATTENDANT'S NAME, TITLE, AND NPI NAME: _____ NPI: _____ TITLE: ☐ MD ☐ DO ☐ CNM/CM ☐ OTHER MIDWIFE ☐ OTHER (Specify) _____	28. MOTHER TRANSFERRED FOR MATERNAL MEDICAL OR FETAL INDICATIONS FOR DELIVERY? ☐ Yes ☐ No IF YES, ENTER NAME OF FACILITY MOTHER TRANSFERRED FROM: _____

REV. 11/2003

MOTHER	29a. DATE OF FIRST PRENATAL CARE VISIT MM / DD / YYYY ☐ No Prenatal Care		29b. DATE OF LAST PRENATAL CARE VISIT MM / DD / YYYY		30. TOTAL NUMBER OF PRENATAL VISITS FOR THIS PREGNANCY _____ (If none, enter A0".)	
	31. MOTHER'S HEIGHT (feet/inches) _____		32. MOTHER'S PREPREGNANCY WEIGHT (pounds) _____		33. MOTHER'S WEIGHT AT DELIVERY (pounds) _____	
	35. NUMBER OF PREVIOUS LIVE BIRTHS (Do not include this child) 35a. Now Living Number _____ ☐ None		36. NUMBER OF OTHER PREGNANCY OUTCOMES (spontaneous or induced losses or ectopic pregnancies) 36a. Other Outcomes Number _____ ☐ None		37. CIGARETTE SMOKING BEFORE AND DURING PREGNANCY For each time period, enter either the number of cigarettes or the number of packs of cigarettes smoked. IF NONE, ENTER A0". Average number of cigarettes or packs of cigarettes smoked per day: # of cigarettes OR # of packs Three Months Before Pregnancy _____ OR _____ First Three Months of Pregnancy _____ OR _____ Second Three Months of Pregnancy _____ OR _____ Third Trimester of Pregnancy _____ OR _____	
MEDICAL AND HEALTH INFORMATION	35c. DATE OF LAST LIVE BIRTH MM / YYYY		36b. DATE OF LAST OTHER PREGNANCY OUTCOME MM / YYYY		39. DATE LAST NORMAL MENSES BEGAN MM / DD / YYYY	
	41. RISK FACTORS IN THIS PREGNANCY (Check all that apply) Diabetes ☐ Prepregnancy (Diagnosis prior to this pregnancy) ☐ Gestational (Diagnosis in this pregnancy) Hypertension ☐ Prepregnancy (Chronic) ☐ Gestational (PIH, preeclampsia) ☐ Eclampsia ☐ Previous preterm birth ☐ Other previous poor pregnancy outcome (Includes perinatal death, small-for-gestational age/intrauterine growth restricted birth) ☐ Pregnancy resulted from infertility treatment-If yes, check all that apply: ☐ Fertility-enhancing drugs, Artificial insemination or Intrauterine insemination ☐ Assisted reproductive technology (e.g., in vitro fertilization (IVF), gamete intrafallopian transfer (GIFT)) ☐ Mother had a previous cesarean delivery If yes, how many _____ ☐ None of the above		43. OBSTETRIC PROCEDURES (Check all that apply) ☐ Cervical cerclage ☐ Tocolysis External cephalic version: ☐ Successful ☐ Failed ☐ None of the above		38. PRINCIPAL SOURCE OF PAYMENT FOR THIS DELIVERY ☐ Private Insurance ☐ Medicaid ☐ Self-pay ☐ Other (Specify) _____	
	42. INFECTIONS PRESENT AND/OR TREATED DURING THIS PREGNANCY (Check all that apply) ☐ Gonorrhea ☐ Syphilis ☐ Chlamydia ☐ Hepatitis B ☐ Hepatitis C ☐ None of the above		44. ONSET OF LABOR (Check all that apply) ☐ Premature Rupture of the Membranes (prolonged, ≥12 hrs.) ☐ Precipitous Labor (<3 hrs.) ☐ Prolonged Labor (≥20 hrs.) ☐ None of the above		46. METHOD OF DELIVERY A. Was delivery with forceps attempted but unsuccessful? ☐ Yes ☐ No B. Was delivery with vacuum extraction attempted but unsuccessful? ☐ Yes ☐ No C. Fetal presentation at birth ☐ Cephalic ☐ Breech ☐ Other D. Final route and method of delivery (Check one) ☐ Vaginal/Spontaneous ☐ Vaginal/Forceps ☐ Vaginal/Vacuum ☐ Cesarean If cesarean, was a trial of labor attempted? ☐ Yes ☐ No	
		45. CHARACTERISTICS OF LABOR AND DELIVERY (Check all that apply) ☐ Induction of labor ☐ Augmentation of labor ☐ Non-vertex presentation ☐ Steroids (glucocorticoids) for fetal lung maturation received by the mother prior to delivery ☐ Antibiotics received by the mother during labor ☐ Clinical chorioamnionitis diagnosed during labor or maternal temperature ≥38°C (100.4°F) ☐ Moderate/heavy meconium staining of the amniotic fluid ☐ Fetal intolerance of labor such that one or more of the following actions was taken: in-utero resuscitative measures, further fetal assessment, or operative delivery ☐ Epidural or spinal anesthesia during labor ☐ None of the above		47. MATERNAL MORBIDITY (Check all that apply) (Complications associated with labor and delivery) ☐ Maternal transfusion ☐ Third or fourth degree perineal laceration ☐ Ruptured uterus ☐ Unplanned hysterectomy ☐ Admission to intensive care unit ☐ Unplanned operating room procedure following delivery ☐ None of the above		
NEWBORN INFORMATION						
NEWBORN	48. NEWBORN MEDICAL RECORD NUMBER		54. ABNORMAL CONDITIONS OF THE NEWBORN (Check all that apply) ☐ Assisted ventilation required immediately following delivery ☐ Assisted ventilation required for more than six hours ☐ NICU admission ☐ Newborn given surfactant replacement therapy ☐ Antibiotics received by the newborn for suspected neonatal sepsis ☐ Seizure or serious neurologic dysfunction ☐ Significant birth injury (skeletal fracture(s), peripheral nerve injury, and/or soft tissue/solid organ hemorrhage which requires intervention) 9 None of the above		55. CONGENITAL ANOMALIES OF THE NEWBORN (Check all that apply) ☐ Anencephaly ☐ Meningocele/Spina bifida ☐ Cyanotic congenital heart disease ☐ Congenital diaphragmatic hernia ☐ Omphalocele ☐ Gastroschisis ☐ Limb reduction defect (excluding congenital amputation and dwarfing syndromes) ☐ Cleft Lip with or without Cleft Palate ☐ Cleft Palate alone ☐ Down Syndrome ☐ Karyotype confirmed ☐ Karyotype pending ☐ Suspected chromosomal disorder ☐ Karyotype confirmed ☐ Karyotype pending ☐ Hypospadias ☐ None of the anomalies listed above	
	49. BIRTHWEIGHT (grams preferred, specify unit) _____ 9 grams 9 lb/oz					
	50. OBSTETRIC ESTIMATE OF GESTATION: _____ (completed weeks)					
	51. APGAR SCORE: Score at 5 minutes: _____ If 5 minute score is less than 6, Score at 10 minutes: _____					
	52. PLURALITY - Single, Twin, Triplet, etc. (Specify) _____					
53. IF NOT SINGLE BIRTH - Born First, Second, Third, etc. (Specify) _____						
56. WAS INFANT TRANSFERRED WITHIN 24 HOURS OF DELIVERY? 9 Yes 9 No IF YES, NAME OF FACILITY INFANT TRANSFERRED TO: _____		57. IS INFANT LIVING AT TIME OF REPORT? ☐ Yes ☐ No ☐ Infant transferred, status unknown		58. IS THE INFANT BEING BREASTFED AT DISCHARGE? ☐ Yes ☐ No		

Mother's Name

Mother's Medical Record No.

Appendix E: IRB Exemption Form A

FORM A

Certification for Exemption from IRB Review for Research Involving Human Subjects

A. PRINCIPAL INVESTIGATOR(s) and/or CO-PI(s) (For student projects, list both the student and the advisor.):
Tess Lefmann, MSSW & Terri Combs-Orme, Ph.D.

B. DEPARTMENT:
College of Social Work

C. COMPLETE MAILING ADDRESS AND PHONE NUMBER OF PI(s) and CO-PI(s):
P.O. Box 3265, Knoxville, TN 37927; (949) 878-2175
204 Henson Hall, 1618 Cumberland Ave., Knoxville, TN 37996; (865) 805-0137

D. TITLE OF PROJECT:
Prenatal Stress, Poverty, and Birth Outcomes
EXTERNAL FUNDING AGENCY AND ID NUMBER (if applicable):
N/A

E. GRANT SUBMISSION DEADLINE (if applicable):
N/A

F. STARTING DATE (NO RESEARCH MAY BE INITIATED UNTIL CERTIFICATION IS GRANTED.):
Upon IRB approval and receipt of the data
ESTIMATED COMPLETION DATE (Include all aspects of research and final write-up):
May 1, 2013

G. RESEARCH PROJECT
Please see attached page for the following

1. **Objective(s) of Project** (Use additional page, if needed.):
2. **Subjects** (Use additional page, if needed.):
3. **Methods or Procedures** (Use additional page, if needed.):
4. **CATEGORY(s) FOR EXEMPT RESEARCH PER 45 CFR 46** (See instructions for categories.):
4

H. CERTIFICATION: The research described herein is in compliance with 45 CFR 46.101(b) and presents subjects with no more than minimal risk as defined by applicable regulations.

Principal Investigator: TESS LEFMANN Name Tessa Lefmann Signature 7/26/2013 Date

Student Advisor: Terri Combs Orme Name Terri Combs Orme Signature 8/2/13 Date

Department Review Committee Chair: Rebecca Bolen Name [Signature] Signature 8/15/13 Date

APPROVED:
Department Head: Karen Sowers Name [Signature] Signature 8/15/2013 Date

COPY OF THIS COMPLETED FORM MUST BE SENT TO COMPLIANCE OFFICE IMMEDIATELY UPON COMPLETION.

Rev. 01/2005

Vita

Teresa Anne Lefmann received her Master's of Science in Social Work degree from the University of Tennessee at Knoxville in 2010. During her time of study there, a required neurodevelopment course taught by Dr. Terri Combs-Orme inspired her to continue her education and attain new knowledge within the field. She began her doctoral studies under Dr. Combs-Orme's tutelage in 2010 at the University of Tennessee and will be receiving her Doctor of Philosophy in Social Work degree in 2014.