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# Trauma Exposure, Posttraumatic Stress, And Depression In A Community Sample Of First-Time Mothers

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**TRAUMA EXPOSURE, POSTTRAUMATIC STRESS, AND DEPRESSION IN A  
COMMUNITY SAMPLE OF FIRST-TIME MOTHERS**

by

**MICHELLE IRENE SPERLICH, M. A., M. S. W., C. P. M.**

**DISSERTATION**

Submitted to the Graduate School

of Wayne State University,

Detroit, Michigan

in partial fulfillment of the requirements

for the degree of

**DOCTOR OF PHILOSOPHY**

2014

MAJOR: SOCIAL WORK (Infant Mental Health)

Approved by:

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Advisor

Date

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## **DEDICATION**

*This dissertation is dedicated to my life partner, Scott Keith Sperlich. I have been free to pursue my passions in life because I am grounded in his support, sacrifice, and abiding love.*

## ACKNOWLEDGMENTS

*I wish to acknowledge and express my gratitude to Dr. Arlene Weisz. Arlene was my academic advisor throughout my doctoral education, and also served as my dissertation committee chair. She guided and encouraged and supported me every step of the way. Her insight has been crucial in helping me make the transition from midwifery, and I have been truly inspired by her dedication to the field of social work and commitment to feminist ideals.*

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## Chapter 1 Introduction

The overall goals of this dissertation project are to increase understanding of the comorbidity of trauma, posttraumatic stress disorder (PTSD) and major depressive disorder (MDD) in a childbearing sample, explore the extent to which trauma is associated with depression, determine outcomes for women with such challenges, and suggest implications for possible intergenerational transmission of trauma and psychiatric vulnerability.

Women in the US report high rates of lifetime exposure to trauma (Kessler, Sonnega, Bromet, Hughes, & Nelson, 1995; Resnick, Kilpatrick, Dansky, Saunders, & Best, 1993) and in particular are at high risk for child and adult sexual assault (Breslau, 2009; Kessler et al., 1995; Tjaden & Thoennes, 2000). Estimates of the number of US women who report a history of child sexual abuse range from 10% to 51% (Briere & Elliot, 2003; Cogle, Timpano, Sachs-Ericsson, Keough, & Riccardi, 2010; Gorey & Leslie, 1997; Kendler, Bulik, Silberg, Hettema & Prescott, 2000; Pereda, Guilera, Forns & Gómez-Benito, 2009; Stoltenborgh, van IJzendoorn, Euser, & Bakersmans-Kranenburg, 2011). Child abuse survivors are at twice the risk for revictimization in adulthood compared to women who did not experience childhood abuse (Barnes, Knoll, Putnam, & Trickett, 2009). The consequences of child sexual abuse are far ranging, placing survivors at risk for medical, psychological, behavioral, and sexual disorders, and it has been suggested that sexual abuse should thus be considered as a general, non-specific risk factor for psychopathology (Maniglio, 2009).

Women bring trauma history and risk for adverse effects with them into pregnancy and early motherhood. Focus on the childbearing year as a sensitive period for trauma survivors is beginning; researchers have begun quantifying the prevalence of trauma and posttraumatic stress during pregnancy and the early postpartum period (Loveland Cook et al, 2004; Seng, Low,

Sperlich, Ronis, & Liberzon, 2009; Seng, Sperlich, Kane Low, Ronis, Muzik, & Liberzon, 2013). The prevalence of current (acute) PTSD in a nationally representative sample of women is 4.6% (Resnick et al., 1993); evidence is accumulating that pregnant women have nearly twice that risk, with rates reaching 8% (Loveland Cook et al, 2004; Seng et al., 2009), and 13-20% exhibiting clinically significant depression (Gold & Marcus, 2008). PTSD due to childhood maltreatment (including child sexual abuse) appears to particularly affect perinatal mental health and bonding outcomes (Seng et al, 2013), and also appears to be associated with prematurity and low birth weight (Morland et al., 2007; Rogal et al., 2007; Seng, Kane Low, Sperlich, Ronis, & Liberzon, 2011).

Postpartum depression has been shown to increase rates of impaired mother/infant bonding and insecure attachments (Beck, 1995; Murray & Cooper, 1997; Seng et al, 2013), poor infant cognitive performance, and maternal perception of infants as more difficult temperamentally (Whiffen & Gotlib, 1989). Depression also increases rates of compromised caregiving, including feeding practices, sleep routines, well-child visits, vaccinations, and safety practices (Field, 2010; Zajicek-Farber, 2009), and is also likely a factor in the 20% of postpartum maternal deaths attributable to suicide (Lindahl, Pearson, & Colpe, 2005).

Researchers have noted the difficulty of diagnosis and treatment for pregnant women affected by comorbidity of PTSD and depression (Silverstein, Feinberg, Sauder, Egbert, & Stein, 2010). Currently, researchers and clinicians addressing PTSD and MDD in childbearing women appear to be operating in distinct “silos.” They are not specifically addressing *either* the comorbidity between the two, *or* the possibility that some depression may be related to unresolved trauma. Studies are needed to elucidate needed steps in the development of targeted interventions for women with history of trauma and psychiatric vulnerability. To address this gap

in knowledge, the goal of this dissertation research is to examine the trajectory of psychopathology for childbearing women by exploring the comorbidity between trauma, PTSD and MDD.

### **Dissertation Research Overview**

**Organizing framework.** A conceptual framework which highlights the role of lifetime violence and posttraumatic stress in the relation between violence trauma and adverse childbearing outcomes (Seng, 2002) has been used to organize salient theoretical perspectives (see Chapter 2) under one rubric. Seng's framework builds on earlier work by the Centers for Disease Control and Prevention (CDC) which addressed research on violence occurring during the childbearing years (Petersen et al., 1996). Seng adapted this framework to include lifetime history of abuse trauma as a contributing factor for adverse outcomes and posited a greater contributory role for PTSD as a plausible mechanism for adverse outcomes.

**Sample and overall design.** Assessment for PTSD per the *Diagnostic and Statistical Manual of Mental Disorders (DSM-IV*; American Psychiatric Association [APA], 1994) (and the *DSM-5*; American Psychiatric Association [APA], 2013) begins with determining whether a person meets the *A1* criterion, meaning that they endorse having experienced or witnessed a major event or events that involved actual or threatened death or serious injury, or a threat to the physical integrity of self or others (see Appendix A for full diagnostic PTSD criteria). The second criterion for trauma, *A2*, is whether the person experienced fear, helplessness, or horror. The focus of my dissertation is determining the extent to which it is warranted to integrate perinatal mental health care to combine attention to depression *and* PTSD, and exploring the implications of the changes to trauma criteria following the publication of the *DSM-5* (APA, 2013). I undertake this by examining whether trauma (as defined by the *A1* and *A2* criterion) is

associated *not only* with perinatal PTSD *but also* with perinatal MDD, at least in a subset of cases. This concept of “Trauma-Exposed Perinatal Depression (T-EPD)” is tested through secondary quantitative analysis of diagnostic psychiatric and demographic interview data and medical record abstraction data from Dr. Julia Seng’s NIH-funded prospective study of a community sample of first-time mothers, housed at University of Michigan, and entitled “Psychobiology of PTSD & Adverse Outcomes of Childbearing” (NIH/NINR R01 NR008767; otherwise known as the “STACY” project). The STACY study recruited participants from 2005 to 2008 at three large health systems’ in Michigan. Eligible women were 18 and older, initiating prenatal care, expecting a first child, and able to speak English. Data were collected via telephone interviews using established instruments to assess trauma history, PTSD, depression, comorbidity reflective of affect dysregulation and interpersonal reactivity, pregnancy substance use, postpartum depression, parenting sense of competence, quality of life, bonding, and sociodemographics. The available data include pregnancy interviews ( $n=1581$ ), 6-week postpartum interviews ( $n=566$ ) and medical chart abstractions ( $n=839$ ).

It is important to note that the STACY data were collected using assumptions inherent to the diagnostic formulation of PTSD as outlined in the *DSM-IV* (APA, 1994). Now that the PTSD diagnostic criteria have changed with the publication of the *DSM-5* (APA, 2013), analyses will proceed in a manner which will address both diagnostic criteria in tandem to the extent possible within the limitations of the data collected.

**Study aims and hypotheses.** I explore the comorbidity of trauma, PTSD and MDD and the concept of “Trauma-Exposed Perinatal Depression (T-EPD)” through analyses guided by the following specific aims and hypotheses.

***Aim I: To determine the extent to which it is warranted to integrate perinatal mental health care to combine attention to depression, trauma, and posttraumatic stress.***

*Hypothesis Ia: Some women who meet diagnosis for major depression will meet the criterion for index trauma exposure (“Trauma-Exposed Perinatal Depression” [T-EPD]), and will be subthreshold for PTSD (will have at least three symptoms of PTSD but not meet the full diagnostic criteria).*

*Hypothesis Ib: Antenatally depressed women with index trauma exposure (T-EPD) will differ from antenatally depressed women without index trauma exposure by type of traumas endorsed overall (index or not), sociodemographic risk factors, and other comorbid conditions.* (Note: Women in all categories reported traumas – but not all endorsed traumas were identified as “index” – meaning that the woman appraised a particular trauma as being the “worst” or “second worst” of the traumas endorsed.)

*Hypothesis Ic: Symptoms of PTSD and the number of traumas endorsed by women will be better predictors of postpartum depression than symptoms of depression in pregnancy.*

The first wave of STACY survey data represents an unselected sample and provides excellent opportunity to establish prevalence rates for trauma, PTSD, and MDD. The remaining waves of survey data and medical record data represent three cohorts selected if they met any of the following criteria: 1) trauma exposure and PTSD, 2) trauma exposure without PTSD diagnosis, and 3) non-exposed controls. Participants who did not fit these criteria (women with trauma exposures but who did not meet diagnostic criteria for PTSD) were not followed longitudinally. Published reports of the STACY data have already demonstrated marked comorbidity between major depression and PTSD (Seng et al., 2009). However, the rates of those reporting significant trauma exposure and depression in the absence of PTSD have not yet



been reported, and it is as yet unknown whether trauma exposure and/or posttraumatic symptomatology in the absence of diagnosis is related to postpartum depression. This is important to explore because women with depression may still have challenges related to their past traumatic experiences that may be impacting their current experience. Identifying such women would increase clinician opportunity to provide resources, including trauma-informed interventions, to women who may need them, regardless of whether they meet the psychiatric diagnostic criteria for PTSD.

Aim I comparison groups have been established *a priori* via participant endorsement of significant trauma exposure and diagnosis of PTSD and MDD for each of the 1581 cases in the sample, using standardized instruments, as elucidated in Chapter 3. Statistical techniques employed for Aim 1 include *multinomial logit* and *receiver-operator growth curve modeling* techniques (described in Chapter 3) to examine the extent to which different predictor variables can differentiate between the comparison groups and predict postpartum depression.

***Aim II: To explore the implications of DSM-5 (APA, 2013) changes in qualifying trauma criteria in relation to perinatal mental health.***

*Hypothesis IIa: Constraining the trauma criteria per DSM-5 guidelines will reduce the rate of PTSD prevalence in the STACY sample, and will thereby reduce the rate of comorbidity between PTSD and MDD.*

*Hypothesis IIb: Those individuals who no longer meet PTSD diagnostic criteria per DMS-5 will have similar levels of postpartum depression.*

*Hypothesis IIc: Those individuals who no longer meet PTSD diagnostic criteria per DMS-5 will have similar levels of bonding impairment to those individuals who do meet DSM-5 criteria.*

Given the extent of trauma exposures and symptoms in perinatal populations and the links to intergenerational patterns and adverse outcomes, we must now look at the changes to *DSM* trauma history criteria. The *one-way analysis of variance* (ANOVA) analyses planned for Aim II are responsive to considering the implications of the changes in the *DSM-5* (APA, 2013) PTSD diagnostic formulation.

### **Summary**

The hypotheses outlined have allowed for two broad areas of investigation. First, it allows us to explore whether trauma exposure alone is associated with depression in a meaningful way, and whether the association centers on sub-threshold PTSD comorbid with MDD. Secondly, it explores implications of the changing diagnostic criteria related to the ability of clinicians to predict which mothers are at-risk for postpartum depression and also impairments in mother/infant bonding. Women with depression combined with subthreshold PTSD may be of genuine clinical concern, and clinicians may be missing opportunity to intervene on their behalf.

The STACY dataset available for this secondary analysis is large, uses gold-standard measures, and is quite diverse. In the next chapter I will review the literature related to trauma, PTSD, and MDD in the childbearing year and compare the STACY study to other studies of perinatal mental health. I will demonstrate the uniqueness of the STACY study and its applicability to the dissertation aims and analyses. However, there are also limitations to using the STACY dataset, including those common to secondary analysis in general. These include the inability to access potentially important variables, especially related to the STACY cohort design which means that some of my subjects of interest were not followed longitudinally, the inability to make temporal predictions regarding trauma exposure and the development of psychopathology, and the fact that a truly dimensional measure of depression is not available

(overall diagnosis is available but due to skip-patterns in the instrument only probability scores, rather than symptom counts, are available). Given these limitations, any nosological implications of these efforts will require evidence of construct, discriminant, and incremental validity relative to established national or international diagnostic categories.

Despite limitations, these analyses *do* provide preliminary evidence about key factors related to the comorbidity of trauma exposure, PTSD, and depression and adverse postpartum outcomes. This may in turn help researchers and clinicians alike to understand *who* is at risk for *what* in perinatal populations, and may have implications for health care for pregnant women by bringing attention to the comorbidity present in clinic and community populations, and highlighting linkages between trauma and depression specifically. Extending the Seng conceptual model to address depression as well will encourage social workers, healthcare professionals, infant mental health specialists and others who provide mental health services to pregnant women to consider trauma-informed and trauma-specific modalities for working with depression during the childbearing year. The conceptual design of these analyses is consistent with the ecological framework of the social work discipline and brings specificity to the identification of structural inequalities such as gender, race, and economic status which undergird the development of poor mental health for pregnant women. Overall, the goal of this project is to increase our understanding of the relative effects of trauma, posttraumatic stress, and depression for childbearing women, and suggest implications for possible intergenerational transmission of trauma and psychiatric vulnerability through the mechanism of impairments in mother/baby bonding.

## **Chapter 2 A Review of the Relevant Literature**

I study mental health during the childbearing year because I view pregnancy as a time of great opportunity for identifying women who might benefit from therapeutic intervention which could help protect them and their babies from adverse outcomes. Understanding the ways in which psychological and physical health are related and may confer intergenerational risk involves translating a great deal of literature and theoretical perspectives across several disciplines. This chapter will review the literature on trauma and stress exposure for childbearing women, perinatal posttraumatic stress disorder (PTSD), perinatal depression (MDD), and the comorbidity between posttraumatic stress and depression and will highlight a few important theoretical perspectives on understanding the differential susceptibility to PTSD and MDD for women. I will also briefly review hypothesized mechanisms of intergenerational transference of vulnerability. Understanding the literature in these areas is important for conceptualization of the design of my dissertation analyses and operationalizing specific factors salient to the examination of the comorbidity of trauma, posttraumatic stress, and depression.

### **Expanding Seng's Conceptual Framework**

The CDC model to guide future research on violence during the childbearing year (Peterson et al., 1996) posited that violence exposure would lead to trauma/stress, which would then be related to physiological mechanisms, and ultimately to adverse maternal and fetal outcomes, after considering mediating and moderating factors such as physiological mechanisms, psychological states, personal disposition, social support, and health behaviors. Seng adapted this very practical model (Figure 1) to add cumulative life trauma to current trauma/stress and specify PTSD as a plausible mechanism for the relationship between trauma/stress and adverse outcomes (Seng, 2002). Seng also codified a bidirectional relationship

between behavioral alterations and neuroendocrine relationships and reconceptualized “health behaviors” to include “modifiable health care related factors,” suggesting a role for both intervention and risk reduction. Both the CDC and Seng models are informed by trauma theory

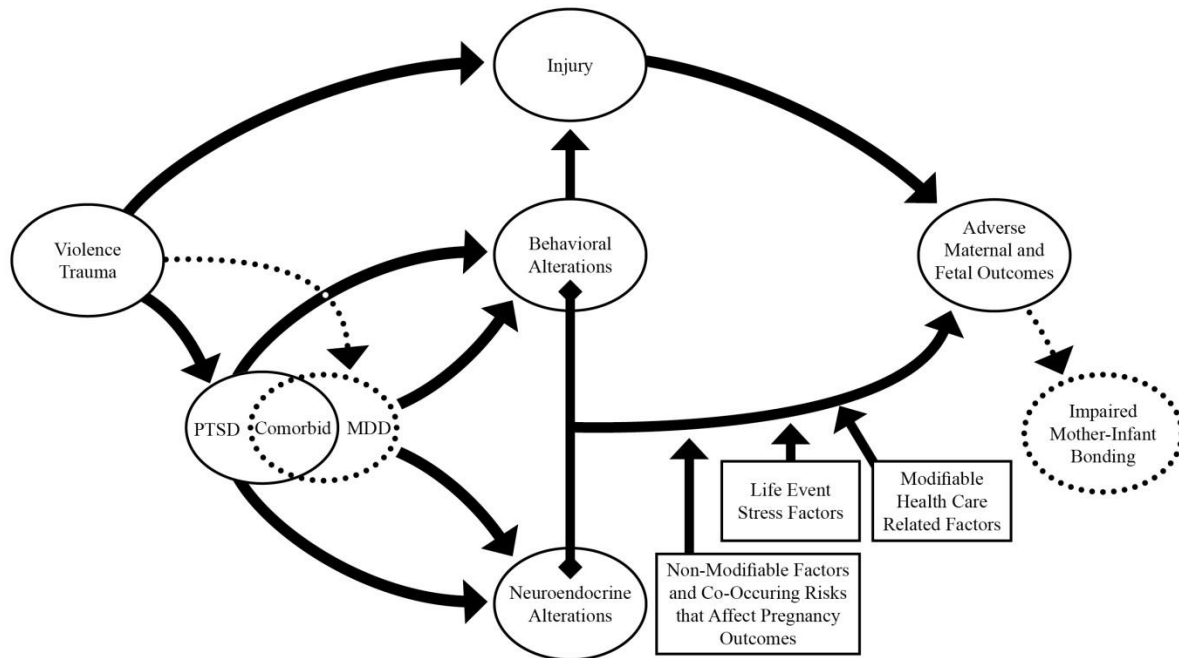


Figure 1. Conceptual framework highlighting PTSD as a factor in studying the relationship between violence trauma and adverse childbearing outcomes. Proposed modifications to include MDD and bonding impairment represented by dotted lines.

in general, which is an “umbrella” theory, commonly associated with Judith Herman’s work (1992), which strives to explain the ways in which humans adapt to major repeated traumatic exposures and may as a result develop a complex form of PTSD. Complex PTSD may also be comorbid with other disorders and present a picture of symptom complexity as the individual attempts to integrate these experiences over time (Cloitre et al, 2009).

I propose to use Seng’s model as a guide to explore whether depression, in addition to PTSD, might also be a pathway by which trauma/stress increases risk for adverse outcomes. I am also suggesting modification to the Seng model to specify that adverse outcomes are exemplified

not only by poorer mental health for mothers and prematurity (for example) for infants, but also by impairments to the mother/infant dyadic relationship through a mother's sense of feeling bonded to her infant, or not. With the STACY data I cannot look specifically at observational measures of dyadic functioning with laboratory attachment paradigms, and am constrained to looking only at the mother's appraisal of her level of feeling bonded to her baby. Yet, owing to findings for the association between postpartum depression and attachment (e.g., Kuscu et al., 2008), bonding impairment suggests an additional pathway to adversity. Mothers who report a lack of bonding may be more likely to have postpartum depression (Kokubu, Okano, & Suguyama, 2012), more likely to have an ambivalent attachment with their babies (Kuscu et al., 2008), and less likely to exhibit positive parenting behaviors (Muzik et al., 2013).

I believe that these changes to the Seng model are consonant with stress-vulnerability models which posit that negative life events lead to general negative affect and ultimately to depressive diagnosis (Hankin & Abramson, 2001). It is also aligned with the biopsychosocial model (Engel, 1977), advanced by the social work profession in general, which posits that each person's life is lived contextually across several domains and that our understanding of how a person might react to a significant stressor such as intimate partner violence is dependent on their appraisal of the event, personal characteristics, and coping capacities (Nurius & Macy, 2010). Adding depression and bonding impairment to the Seng model also portrays a heterogeneity of response to stress, and in that regard is aligned with work by Bonanno and others to characterize multiple trajectories of adjustment to "potentially traumatic events," including chronic distress, recovery, resilience, and delayed reactions (e.g., Bonanno & Mancini, 2011).

## Defining Trauma and Stress

One of the challenges in comparing outcomes of studies of mental health in the perinatal period related to adversity is the variety of ways in which trauma and/or stress have been characterized and measured. An exposure to trauma is part of the diagnostic formulation of PTSD, and the *DSM-5* (APA, 2013) defines it as “(e)xposure to actual or threatened death, serious injury, or sexual violence.” (p. 271). Yet there is provision within the *DSM-5* for indirect exposure as well, including learning that such traumas have happened to a loved one, or experiencing repeated or extreme exposures to aversive details of such traumas through work as a first responder to a traumatic incident or through repeated exposure to child abuse details on the part of law enforcement officials (and presumably others). As such, the very definition of trauma, even within the diagnostic formulation, is somewhat complex. The placement of PTSD within the pantheon of mental disorders also reflects a changing nosological landscape with the publication of the *DSM-5*; PTSD, previously classified as an “Anxiety” disorder, is now listed in the company of “Trauma and Stressor-Related Disorders.” Although categorized as potentially related to stress as well, the diagnostic formulation itself is entirely dependent on a “traumatic” rather than a “stress” exposure. Conversely, the current diagnostic formulation for PTSD in the international diagnostic “bible,” the World Health Organization’s International Classification of Mental Disorders and Related Health Problems (*ICD 10*; World Health Organization, 2008), depends on a person’s “response to a stressful event or situation (either short-or long lasting) of an exceptionally threatening or catastrophic nature, which is likely to cause pervasive distress in almost anyone (p.120).” This may seem like “splitting hairs” to point out these distinctions, since after all, we are reliant exclusively on an affected individual’s *appraisal* of an event in order to

define such exposures. However, this becomes an important consideration which bears influence on instrumentation and subsequent analysis when looking at exposures during the childbearing year.

For example, in a recent systematic analysis of the various psychometric instruments utilized in perinatal studies, reviewers found that 115 publications in the ten-year period between 1999 and 2009 used 43 different instruments to assess psychosocial stress during pregnancy (Nast, Bolten, Meinlschmidt, & Hellhammer, 2013). Nast and colleagues organized these instruments into seven categories, including *anxiety, depression, daily hassles, aspects of psychological symptomatology which were not reduced to anxiety or depression, life events, specific socio-environmental stressors and stress related to pregnancy and parenting itself*. The authors suggest that this diversity of measurement hampers efforts to compare results across research studies. Similarly, there exist many different instruments which assess for trauma exposure; and different studies use different measures or truncate their measurement to items of specific interest, like combat exposure, or child sexual abuse, for example. The National Center for PTSD, for example, lists 13 different trauma exposure measurements on their website which are available for clinicians to use (<http://www.ptsd.va.gov/professional/pages/assessments/list-trauma-exposure-measures.asp>). They are working on updating several of these measures in order to be consistent with *DSM-5* (APA, 2013).

Trauma/stress has also been conceptualized as “adverse events,” following on the publication of analyses of the Adverse Childhood Experiences (ACE) study (Felitti et al., 1998). Often cited, the ACE study queried over 13,000 adult members of a large HMO in California about “adverse childhood experiences,” including psychological, physical, or sexual abuse; violence against mother; and living with household members who were substance abusers,



mentally ill or suicidal, or ever imprisoned. They found a “dose-response” pattern of vulnerability to many adult health conditions, including alcoholism, drug abuse, depression, suicide attempt, smoking, and many serious medical conditions like cancer, lung disease, skeletal fractures, and liver disease. Subsequent to these findings the study authors have made the “ACE score” freely available on their website ([http://acestudy.org/ace\\_score](http://acestudy.org/ace_score)) and many clinicians in primary practice settings avail themselves of this relatively easy to use measure. However, the ACE items do not map onto *DSM-5* (APA, 2013) diagnostic requirements for trauma exposure; for instance, there is no provision in the *DSM-5* for “psychological trauma” as an index trauma (meaning the participant deemed it the “worst or “second worst” in their life) for the PTSD diagnosis.

The American College of Obstetricians and Gynecologists (ACOG), in a committee opinion about perinatal screening, recommended that all women be screened for “psychosocial stressors,” which they define as “barriers to care, unstable housing, unintended pregnancy, communication barriers, nutrition, tobacco use, substance use, depression, safety, intimate partner violence, and stress (ACOG, 2006).” In practice, it appears that in clinical settings that the terms ‘stress’ and ‘trauma’ are used interchangeably without consideration of qualitative differences which might influence reactions to these various events or stability of such reactions over time. Yet this presents a problem for research settings where the lability of definitions disinhibits direct comparisons; one may reasonably ask whether a study of ‘daily hassles,’ for instance, might be readily compared with one which considers domestic violence, for example, or childhood sexual abuse. So, while I cannot provide a unified definition for either ‘stress’ or ‘trauma,’ I will make transparent which specific exposures are being referenced whenever possible in the following review.

## **Trauma, Stress, and Childbearing**

Researchers have looked at direct links between various trauma/stress exposures and adverse outcomes during pregnancy and the early postpartum period, exclusive of any mediating effect of resultant or concurrent psychopathology. The following section reviews findings for various trauma/stress exposures, including childhood sexual abuse, intimate partner violence, psychosocial stressors, and intergenerational effects related to trauma/stress exposure. There are additional bodies of literature which look at the effects of traumatic injuries (including motor vehicle accidents), and natural and manmade disasters (including military deployment) which happen *during* the childbearing year; however, because the focus of this dissertation research is on the effect of trauma exposures that women bring into pregnancy with them, I will not be reviewing these particular areas of investigation.

**Childhood sexual abuse.** As mentioned in the introductory chapter, the rates of women reporting childhood sexual abuse are high, ranging from 10% to 51% (Briere & Elliot, 2003; Cogle, Timpano, Sachs-Ericsson, Keough, & Riccardi, 2010; Gorey & Leslie, 1997; Kendler, Bulik, Silberg, Hettema & Prescott, 2000; Pereda, Guilera, Forns & Gómez-Benito, 2009; Stoltenborgh, van IJzendoorn, Euser, & Bakersmans-Kranenburg, 2011). In a random sample of 1835 women in Israel, a higher percentage of pregnant women who disclosed a history of childhood sexual abuse also reported higher rates of “gynecological problems” (Yampolsky, Lev-Wiesel, & Ben-Zion, 2010). Two case-control studies conducted in Germany have also found increased complications during pregnancy; a study of 118 women found that prenatal medical complications and infant complications in the postpartum period were increased for childhood sexual abuse survivors (Möhler et al., 2008), and another study of 155 women also

found significantly increased risk for pregnancy and infant complications, including pregnancy hospitalizations, more episodes of premature labor contractions, cervical insufficiency, and premature birth (Leeners, Stiller, Block, Görres, & Rath, 2010). Although not a focus of this dissertation study, it is important to note that several studies have identified strong links between childhood sexual abuse and adolescent pregnancy; meta-analysis of these studies show that a history of child sexual abuse conferred twice the risk for adolescent pregnancy (Noll, Shenk & Putnam, 2009).

**Adult sexual abuse.** Investigations of adverse childbearing outcomes specifically related to adult-only sexual abuse are lacking. This may be because there are high rates of both childhood and adult revictimization among women who experienced childhood sexual abuse (Barnes, Knoll, Putnam, & Trickett, 2009), and as such it may be an arbitrary distinction to separate the trauma by epoch. We do know that there is also considerable overlap between physical and sexual abuse in intimate partner relationships (e.g., McFarlane et al., 2005; Tjaden & Thoennes, 2000), however, and a relatively small body of research has begun investigating outcomes of pregnancy as a result of rape (Nerum, Halvorsen, Straume, Sørli, & Ølan, 2012).

**Intimate partner violence.** The prevalence of intimate partner violence (IPV) within the last year prior to pregnancy ranges from 3% to 9% (Gazmarian et al., 1996). In a population-based sample representing 16 states and using data from the Pregnancy Risk Assessment Monitoring System (PRAMS) developed by the Centers for Disease Control (CDC), Saltzman, Johnson, Gilbert and Goodwin (2003) found that 8.7% of women experience partner violence occurring around the time of pregnancy. Intimate partner violence during pregnancy presents a range of problems, including higher rates of depression, suicide attempts, substance use, and pregnancy and birth complications (Kiely, El-Mohandes, El-Khorazaty, & Gantz, 2010). Meta-

analysis shows a three-fold increase in the odds of developing PPD after experiencing IPV during pregnancy (Howard, Oram, Galley, Trevillion & Feder, 2013), and Ludermir and colleagues found that psychological violence during pregnancy by an intimate partner is strongly associated with PPD independently of physical or sexual violence (Ludermir, Lewis, Valongueiro, de Araújo, & Araya, 2010). Not surprisingly, a recent study of 120 women found that both child maltreatment and IPV during pregnancy were significantly related to the development of posttraumatic stress symptomatology, as well (Huth-Bocks, Krause, Ahlfs-Dunn, Gallagher, & Scott, 2013). Last, but not least, the risk of pregnancy-related suicide and homicide is concentrated in relationships involving IPV; in analysis of data from the National Violent Death Reporting System between 2003 to 2007 researchers found a suicide rate of 2.0 per 100,000 live births and a homicide rate of 2.9 deaths per 100,000 live births (Palladino, Singh, Campbell, Flynn, & Gold, 2011). They also found that homicide victims were more likely to be African American and at the extremes of the age range for pregnancy, and that 54.3% of suicides and 45.3% of homicides were associated with intimate partner violence.

Taken as a whole, this review suggests that interpersonal trauma, whether childhood sexual abuse/maltreatment, adult sexual abuse, or IPV, is associated with both posttraumatic stress *and* perinatal depression. Yet abuse trauma is not happening in a vacuum; the following section of this review will consider the broader psychosocial context and the independent and cumulative risk that is conferred by exposure to psychosocial stressors including race, poverty, low education, not living with a partner, and neighborhood effects.

**Psychosocial stress.** In an effort to account for the observed increases for African American women in relation to adverse birth outcomes, Arline Geronimus (1996) hypothesized that African American women experience a “weathering” effect, which describes

the cumulative and compounding effects of social inequality. Geronimus tested this hypothesis through analysis of over 50,000 birth and infant death certificates in Michigan and augmented these data with census-based economic indicators. She found that for low-income African American women, the odds of having a low birth weight infant were increased 3-fold, and that the odds of having a *very* low birth weight infant were increased 4-fold. Her data further suggested that this disparity was related to increasing age of African American mothers, lending fuel to her hypothesis for a weathering effect. Additionally, Geronimus found increased rates of smoking during pregnancy for African American mothers, which in and of itself may have accounted for some of the increase in prematurity. Geronimus' analysis, while robust in size and influential in nature, was hampered by likely under-reporting of health conditions via birth certificate data, and lack of any mental health data on the women included in her study. However, researchers have continued to find support for linkages between socioeconomic disparities, including racial disparity specifically, related to prematurity and infant mortality (Blumenshine, Egerter, Barclay, Cubbin, & Braveman, 2010; Chen et al., 2012; MacDorman, 2011), as well as increased tobacco usage and underutilization of prenatal care for African American women (Dailey, 2009). There are also a number of studies making direct links between self-report discrimination with premature birth and low birth weight (e.g., Collins, David, Handler, Wall, & Andes, 2004; Mustillo et al., 2004). It may be that 'weathering' denotes effects related not only to discrimination but also to structural inequalities such as income, education, and access to both physical and mental health care, and that adverse health effects arise from a combination of these conditions. The term 'intersectionality' (Crenshaw, 1991) has been used to describe how multiple aspects of one's personal identity intersect to confer risk for adverse health outcomes. The intersecting effects

of race, class, gender, pregnancy status, age, religion, etc. may combine to confer additional stress which may in turn have adverse impacts on childbearing. In an exploratory analysis using an interpersonal-level approach to modeling intersectionality in a quantitative way, STACY researchers considered the role that discrimination played in the development of posttraumatic stress and also quality of life by considering not only the number of discriminatory experiences reported but also the woman's attribution for why she experienced that discrimination (Seng, Lopez, Sperlich, Hamama, & Meldrum, 2012). Using these attributions as a proxy for an "intersecting identities" increased the predictive power for explaining disparity in both posttraumatic symptoms and quality of life scores, and may suggest a pathway to bridge the qualitative work being done looking at intersectionality with the quantitative research which considers multiple levels of structural inequalities.

In addition to the effects of socioeconomic inequalities, it appears that African American women are at increased risk for a history of lifetime traumatic exposures, which is associated with adverse prenatal health via concurrent risk for tobacco use, premature rupture of membranes, and longer hospital stays (Dailey, Humpheys, Rankin, & Lee, 2011). Analysis of the STACY data also found increased risk for history of trauma exposure for the 709 first-time African American mothers studied, which was concurrent with increased PTSD diagnosis and symptoms, comorbidity with depression, and pregnancy substance use including tobacco and alcohol (Seng, Kohn-Wood, McPherson, & Sperlich, 2011). However, an important finding of another 2011 STACY analysis was that, when considered in the same multivariate regression models, PTSD subsequent to childhood maltreatment was a stronger predictor of shorter gestation than being African American, and a nearly equal predictor of low birth weight (Seng, Kane Low, Sperlich, Ronis, & Liberzon, 2011). What this suggests is that

although at the bivariate level African American women are at greater risk for premature birth, when specific interpersonal traumas and any resultant PTSD, together with coping behaviors such as substance usage are taken into consideration, we can start to “unpack” some of the weathering process. We can then see that it may be *not only* socioeconomic inequalities which contribute to weathering but also the differential exposure to trauma/stress and additional risk inherent to maladaptive coping behaviors that women sometimes develop to cope with trauma/stress and environmental risks.

Gavin, Nurius and Logan-Greene (2012) considered other mediators of the relationship between women experiencing social disadvantages (poverty, low education, and not living with a partner), and adverse birth outcomes in a community-based sample of 2,168 pregnant women. They found evidence of a mediated pathway through maternal health conditions in pregnancy, including chronic medical conditions such as asthma, hypertension, diabetes, etc. Curiously, although the authors of this study reference the ‘weathering’ hypothesis, there is no reporting of the racial/ethnic diversity of the sample other than to say it is “diverse,” and race itself is not considered in analytic models.

A study of over 3,000 women which did explicitly consider racial and ethnic differences in determinants of preterm birth examined the extent to which neighborhood disadvantage played a role (Reagan & Salsberry, 2005). Reagan and Salsberry found that neighborhood disadvantage; in particular neighborhood poverty rates and housing vacancy rates, increased the rates of very preterm birth for African Americans. Rates of preterm birth for Hispanic women were increased for those who were residing in female-headed households, and rates of preterm birth among white women decreased with increasing rates of employment. Specification of the concept of neighborhood disadvantage is underway; seen in

preliminary work to develop an instrument to assess racial residential segregation and its effects on birth outcomes and thereby establish black isolation as a proxy for concentrated socioeconomic disadvantage (Anthopolos, James, Gelfand, & Miranda, 2011). It is also seen in the establishment of scales to measure physical incivility, territoriality and social spaces, all in an effort to capture important aspects of neighborhood characteristics which may exert influence on the social context of pregnancy (Laraia et al., 2006).

**Intergenerational implications of maternal abuse history.** I've already alluded to the role of trauma/stress in the development of preterm birth and low birth weight. These represent severe adverse outcomes that are readily evident in terms of intergenerational transmission of health risk. Also evident so far in this review is the role of risk behaviors on the part of a mother who is potentially self-medicating with tobacco, alcohol, or other substances. The prevalence of smoking during pregnancy, as determined from PRAMS data from 2008 and birth certificate data from 2003 has been shown to be between 11.3 to 15.2% (Tong, Dietz, Farr, D'Angelo, & England, 2013); pregnancy smokers were more likely to be aged 25-29 years, of non-Hispanic white race, unmarried, enrolled in Medicaid or uninsured, and participating in WIC services. Researchers at the CDC calculated the average annual percentage of any alcohol use on the part of pregnant women to range from 10.2 to 16.2% (Denny, Tsai, Floyd, & Green, 2009). Despite increasing awareness of the effects of alcohol consumption and the link to fetal alcohol syndrome, Denny and colleagues found that the prevalence of alcohol use during pregnancy has not changed substantially since 1991.

Although researchers have established links between risk behaviors such as smoking and drinking during pregnancy, the exact mechanisms by which the risk behaviors translate into poor fetal growth and adverse outcomes are still being sought. Such investigations look at



potential mechanisms using the lens of *fetal programming, or fetal origins of disease* (Barker, 1990, 1995), which suggests that adverse effects on infant and adult health may originate in compromised fetal health status related to nutritional impoverishments in the uterine environment, with more recent emphasis on potential genetic polymorphisms or other epigenetic changes caused by the teratogenic effects of substance exposure in utero (e.g., Suter, Anders, & Aagard, 2013). It is beyond the scope of this dissertation work to contribute specifically to the literature on fetal programming. Yet it is important to move in the direction of understanding what the relative contributions to the variance in risk to offspring are for maternal trauma exposure, maternal psychiatric sequelae, and maternal risk behavior - both unrelated and related to trauma and psychopathology.

It is also important to state explicitly that many mothers who face trauma/stress and psychological challenges to such exposures are quite resilient and go on to be “good enough” mothers (Winnicott, 1953). For those who have experienced maltreatment as a child, there appear to be specific protective factors such as perceived parental care, adolescent peer relationships, quality of adult love relationships, and specific personality characteristics which contribute to good prognosis (Collishaw et al., 2007). Yet associations have been found between mothers with history of childhood abuse related to increases in intrusive parenting style (Moehler, Biringen, & Poustka, 2007), anxieties about intimate parenting (Douglas, 2000), poorer behavioral trajectories for children at ages 4-7 (Collishaw et al., 2007), and more punitive disciplinary style (DiLillo & Damashek, 2003; Schuetze & Eiden, 2005). Disruptions that result from a mother’s inability to interact with her infant in a healthy pattern have been described as *early relational trauma* (Schoore, 2009), with the infant at risk due to the mother’s frightened, threatening, or dissociative interactions or from the unpredictability of these

extreme maternal responses (Hesse & Main, 2006), particularly for disorganized attachment (e.g., Lyons-Ruth, Bronfman, & Parsons, 1999) and later behavioral problems (e.g., Fearon, Bakermans-Kranenburg, van IJzendoorn, Lapsley, & Roisman, 2010). A child who has a mother who was abused is at risk for maltreatment, whether by the mother herself (Spieker, Bensley, McMahon, Fung, & Ossiander, 1996; Sroufe, Egeland, Carlson, & Collins, 2005), or by an intimate partner or member of the mother's family-of-origin (McCloskey & Bailey, 2000). In a comparison of abused children and non-abused children, PTSD and depression were among the diagnoses found to occur more often among mothers of abused children (Famularo, Kinscherff, & Fenton, 1992). Analysis of a community sample of 499 women suggest that there is a mediated pathway to offspring victimization for mothers who are victims of abuse themselves and who have substance abuse problems (Appleyard, Berlin, Rosanbalm, & Dodge, 2011). It is important to point out here that there are several methodological problems related to studying the intergenerational effect of child maltreatment which may hamper our understanding; Newcomb & Locke (2001) suggest that it is important to be specific in measuring the type of child maltreatment exposure that parents have experienced in order to better understand specific risk to children. In their community sample of 383 parents, they found a moderately strong effect for parental history of child maltreatment to poor parenting for both mothers and fathers; however, these effects differed by maltreatment history. For mothers a history of neglect by itself led to poor parenting, and sexual abuse history led to aggressive parenting; whereas for fathers a history of sexual abuse led to rejecting parenting practices. Yet another important obvious consideration for intergenerational transmission is the extent to which those parents who have experienced child maltreatment maintain connection with potential abusers in their family of origin or broader

social structure, which may be driven by both economic dependency and lack of agency that may result from psychological adaptations to trauma/stress.

### **Maternal Psychopathology**

The following review is constrained to addressing the prevalence, risk factors and outcomes related to PTSD, depression, and the comorbidity between the two. Although women do experience other psychiatric challenges in the childbearing year, it is beyond the scope of this investigation and available data to adequately address mental health conditions other than those directly salient to the planned analyses. Furthermore, many of the studies which consider “anxiety in pregnancy” make no distinction between the different anxiety disorders, which often include PTSD, which is problematic going forward when taking into consideration the *DSM-5* (APA, 2013) reclassification of PTSD as a trauma-related rather than an anxiety-related disorder.

### **PTSD**

PTSD may result from directly experiencing or witnessing a traumatic event such as actual or threatened death, serious injury, or sexual violence. Its hallmark feature is the re-experiencing of the trauma, through intrusive memories, nightmares, and dissociative reactions such as “flashbacks” (feeling that the trauma is happening all over again). Additional diagnostic features include avoidance of reminders of the trauma, negative alterations in cognitions and mood, marked alterations in physical arousal and reactivity, all of which last longer than one month in duration and cause clinically significant impairment in social or occupational functioning unrelated to any effects of substance use or a medical condition (APA, 2013). Diagnostic symptom clusters and criteria for PTSD have changed recently owing to the release of the *DSM-5* (APA, 2013); this change will affect methodology related to my dissertation since the STACY study dataset available for secondary analysis was collected under assumptions of

the previous edition of the *DSM-IV* (APA, 1994). I will specify such effects in the chapter on methodology.

Another issue relevant to this study is the phenomenon of ‘subthreshold’ (or ‘partial’ or ‘subsyndromal’) PTSD. These distinctions have been used to characterize those individuals who have symptoms of PTSD but do not meet the full diagnosis. Although the literature on subthreshold PTSD is not as developed as for full PTSD, a recent publication of the National Center for PTSD (2014) finds that there are commonly held definitions that include the exposure, duration and distress/impairment elements of the *DSM-IV* (1994) PTSD formulation, together with some combination of symptoms. The National Center for PTSD’s review finds that despite the variability in definitions that subthreshold PTSD has been found to be associated with both psychiatric and medical comorbidity. They point to work by Ruscio, Ruscio, & Keane (2002) which suggests that the latent structure of PTSD is a dimensional one, with the full diagnosis at the high end of a continuum of stress response, and several studies demonstrating functional impairment for those with subthreshold PTSD as compared to those with trauma exposure and no PTSD (e.g., Breslau, Kucia, & Davis, 2004, Pietrzak et al., 2012). Although more studies are needed, those that do exist suggest that the subthreshold diagnosis has clinical relevance, and should continue to be a focus of research.

**Prevalence and risk of PTSD for childbearing women.** It has been demonstrated that women are at twice the risk for PTSD following trauma exposure compared to men (Breslau, Davis, Andreski, & Peterson, 1991; Kessler et al., 1995). Childbearing women are also at increased risk for PTSD, with incidence rates in pregnancy converging at 8% (Seng et al., 2009), which is nearly double the incidence for women in the general population (Resnick, et al., 1993). High levels of posttraumatic stress symptoms are observed among pregnant women

with history of violence-related trauma (Harris-Britt, Martin, Li, Casanueva, & Kupper, 2004), which in turn is associated with subsequent postpartum posttraumatic stress symptomatology (Lev-Wiesel, Chen, Daphna-Tekoah, & Hod, 2009). Investigations into PTSD in the childbearing year have looked at the traumagenic potential for the birth experience itself (Ayers, Harris, Sawyer, Parfitt, & Ford, 2009; Denis, Parant, & Callahan, 2010; Söderquist, Wijma, Thorbert, & Wijma, 2009), PTSD in relation to childbirth pain (Boudou et al., 2007), operative delivery (Söderquist, Wijma & Wijma, 2002), and in relation to the experience of having a sick or premature infant (Shaw et al., 2009; Vanderbilt, Bushley, Young, & Frank, 2009).

**Differential susceptibility to PTSD.** Empirically supported models which establish the etiology of risk for differential susceptibility to PTSD are nascent. One possible reason for increased risk of PTSD may be the initial response to a trauma; one study which looked at PTSD following motor vehicle accidents found that *peritraumatic dissociation* (distorted perceptions of time, person, and place) may be experienced more by females and may partially account for gender differences in the development of posttraumatic symptoms six weeks post-accident (Irish et al., 2011). Another review of the literature also suggests that women may be more susceptible because of the phenomenon of peritraumatic dissociation, and also due to the specific types of trauma to which they are exposed, their stronger perceptions of threat and loss of control, insufficient social support services, and greater use of alcohol to manage trauma-related symptoms (Olf, Langeland, Draijer, & Gersons, 2007).

**PTSD and childbearing outcomes.** Infants born to mothers with PTSD during pregnancy subsequent to childhood maltreatment are at increased risk for low birth weight and preterm birth, after controlling for depression (Seng et al., 2011). As mentioned, previous to

the publication of the *DSM-5* (APA, 2013) PTSD was considered as an anxiety disorder. As a result several studies considered anxiety during pregnancy in the aggregate, including PTSD, generalized anxiety, state-trait anxiety, and phobic disorders in some cases. One study found that ‘anxious’ mother/baby dyads (measured as generalized anxiety, state/trait anxiety, phobias) scored less optimally than controls on measures of maternal sensitivity and infant social engagement (Feldman et al., 2009). Another study found that antenatal phobic anxiety but *not* antenatal depression (depression during pregnancy) was associated with behavioral and emotional problems in children at age four (O’Connor, Heron, & Glover, 2002). A 2005 review found fourteen prospective studies with evidence linking the effects of antenatal anxiety (including panic disorder, ‘daily hassles’, state-trait anxiety, chronic stress, life event stress, phobias) to cognitive, behavioral and emotional problems in children (Van den Bergh, Mulder, Mennes, & Glover, 2005). Several of these studies also looked at depression, and in fact in recent years there has been a shift among some researchers to calling the comorbidity between these various types of anxiety combined with depression “perinatal mood and anxiety disorders” (e.g., Bradley & Hill, 2011). This is likely also in part an artifact of the frequent use of the *Edinburgh Postnatal Depression Scale (EPDS)*; (Cox & Holden, 2003), which has subscales for both depression and anxiety.

### **Perinatal Depression**

The risk of major depression is 1.70 to 2.70 times higher for women in the United States than for men (Kessler, McGonagle, Swartz, Blazer, & Nelson, 1993; Marcus et al., 2008; Weissman, et al., 1996). This risk peaks during a woman’s childbearing years (Kessler et al., 1993). Research suggests that the prevalence of perinatal depression may not be higher during pregnancy or the first year postpartum than for non-pregnant women (Cox & Holden, 2003;

Gavin et al., 2005). However, because there appears to be an increased rate of *onset* in pregnancy and the postpartum period, this represents opportunity for detection and intervention since women are also accessing healthcare during this time (Stowe, Hostetter & Newport, 2005). Recent investigations have also begun exploring the links between postpartum depression and later conversion to bi-polar disorder. For example, a Danish study of over 120,000 women linking health records with psychiatric records found that a postpartum onset of psychiatric symptoms in the first 14 days after delivery significantly predicted conversion to bi-polar disorder within the 15-year follow-up period (Munk-Olsen, Laursen, Meltzer-Brody, Mortensen, & Jones, 2012).

Due to the often devastating consequences for mothers, babies and families, including infanticide and suicide, perinatal depression has been a frequent target of research and intervention, particularly for postpartum depression, and more recently for antenatal depression. The *DSM-IV* (APA, 1994) or the *DSM-5* (APA, 2013) does not designate a special type of perinatal depression but rather lists several categories of conditions for which there may be the specifier “with postpartum onset” if they occur in the first four weeks after birth, including *major depressive disorder, manic or mixed episode in major depressive disorder, bipolar disorder, or brief psychotic disorder*.

Depression may develop at various points during the childbearing year. Of women referred to specialty care for depression, one study found that 11.5% reported onset in the prenatal period, 22.0% had late postpartum onset, and 66.5% had early postpartum onset (Stowe, Hostetter, & Newport, 2005). These researchers urge that, because the risk is spread over the childbearing year, it is necessary to screen mothers throughout prenatal care, at postpartum check-ups, and at pediatric visits during the first six months postpartum.

**Differential susceptibility to depression.** In attempting to explain the discrepancy in depression between genders, Hankin and Abramson (2001) elaborated on previous cognitive *vulnerability-stress* models for understanding the emergence of gender differences in depression. Their model posits that negative events lead to initial elevations of general negative affect, and then generic cognitive vulnerability factors moderate the likelihood that the initial negative affect will progress to depressive diagnosis. The model is transactional in that depression can then itself lead to more self-generated negative life events and thus begin the causal chain again. In this developmentally sensitive model, a traumatic life experience, such as child sexual abuse, plays a contributory role in the formation of negative affect and risk for further negative life events. Studies which report significantly more severe and non-severe negative events for women prior to onset of depression than for men (Harkness et al., 2010) present good evidence to support this model.

*Biological or medical models* which emphasize genetic vulnerability or abnormalities of brain and biochemical processes affecting regulation of neurotransmitters and reactions to stress are prevalent, ubiquitous explanations (if not overtly identified theories) for depression in general. They hold sway today as evidenced by the common usage of antidepressant medication. It has also been suggested from a *feminist perspective* that the chronic stressors associated with the traditional female role in society have led to a higher prevalence of depression in women (Mirowsky & Ross, 1995; Rosenfield 2000). Because the rates of depression rise at a time that coincides with the onset of puberty (Essau, Lewinsohn, Seeley, & Sasagawa, 2010), some theorists have endorsed an ovarian *hormonal dysregulation* explanation (*estrogen* and *progesterone*) for the greater prevalence of depression for women (Moos et al., 1969). However, owing to the fact that other epochs of hormonal changes in a woman's life (pregnancy,



menopause, use of oral contraceptives, use of hormone replacement therapy) have not been associated with increased risk for depression over that of men, purely hormonal explanations have been largely abandoned (Kessler, 2003).

More recent models look at the hormone *oxytocin*, a hypothalamic neuropeptide which plays a key role in mammalian female reproductive function and affiliation. It has been found to be dysregulated in depressed women (Cyranowski et al., 2008) and in women who are trauma abuse survivors and who have posttraumatic stress and pelvicvisceral dysregulation (Seng, 2010).

Other explanations for the increased levels of depression in women center on findings related to the insidious relationship between rumination and depression, which is a prominent feature of Nolen-Hoeksema's *response styles* theory (Nolen-Hoeksema & Morrow, 1991). Rumination is a mode of responding to distress that involves repetitively and passively focusing on the symptoms of distress and on the possible causes and consequences of the symptoms, and was proposed as an explanation of women's greater vulnerability to depression (Nolen-Hoeksema, Wisco, & Lyumomirsky, 2008). The response styles theory was originally thought to predict the duration of depression; however, newer evidence suggests it predicts onset rather than duration of depressed mood, as well as other comorbidities including anxiety, binge eating, binge drinking, and self-harm (Nolen-Hoeksema, Wisco, & Lyumomirsky, 2008). The negative content of ruminative thought is conceptually similar to the automatic thoughts, schema, and negative cognitive styles extensively studied by *cognitive theorists* (e.g., Beck, 1976), which are the target of many CBT treatments for depression in women.

Various theoretical perspectives have been identified as prominent in the discourse, specifically for *postpartum* depression, including the *medical model*, *feminist theory*, *attachment*

*theory, interpersonal theory, and self-labeling theory* (Beck, 2002). With the medical model there is a recent shift in focus to looking not only at neuroendocrine function, but also at psychoneuroimmune function as an etiological explanation for postpartum depression, particularly examining the role of proinflammatory cytokine dysregulation and its interaction with glucocorticoids (Kendall-Tackett, 2010).

Proponents of feminist theory as it relates to postpartum depression and other mental states oppose the medical model approach and the propensity to label women by their “disease,” when many women may be simply trying to cope with the impossible standards of the motherhood mystique imposed by the medical model and society at large (Lazarre, 1997; Mauthner, 1993). It has also been suggested that medical control of childbirth reinforces helplessness in women and may contribute to postpartum depression, as well (Oakley, 1980).

**Prevalence of antenatal depression.** A systematic review found that estimates of the prevalence of antenatal depression range from 5.0% to more than 25%, depending on the population sampled, with rates from 7.4% in the first trimester to 12.8% in the second trimester and 12.0% in the third (Bennett, Einarson, Taddio, Koren, & Einarson, 2004). A competing review conversely found overall lower rates during pregnancy; 11% in the first trimester, and a drop to 8.5% in the second and third trimesters (Gavin et al., 2005). A meta-analytic review by the same researchers found that as many as 14.5% of pregnant women have a new episode of major or minor depression during pregnancy (Gaynes et al., 2005). A longitudinal study since the 2004 and 2005 reviews finds that the proportion of women with depressive *symptoms* was 15% in the first trimester, 14% in the second trimester, and 30% in the third trimester (Setse et al., 2009). Recent research also suggests that the prevalence of antenatal depression is increased for ethnic minority populations; a Canadian study found rates as high as 29.5% among Aboriginal

women (Bowen, Stewart, Baetz, & Muhajarine, 2009), and 25% among African American women (Luke et al., 2009).

**Risk factors specific to antenatal depression.** A number of potential risk factors for antenatal depression have been identified in the literature. These include poor overall health, greater alcohol use consequences, smoking, single motherhood, unemployment and lower educational attainment (Bolton, Hughes, Turton, & Sedgwick, 1998; Marcus, Flynn, Blow, & Barry, 2003). Other risk factors include history of prior depression (Marcus, et al., 2003), previous miscarriage (Rubertsson, Waldenström & Wickberg, 2003), and history of childhood sexual abuse (Rodgers, Lang, Twamley, & Stein, 2003). Meta-analytic review suggests that domestic violence, life stress, and lack of social support are contextual factors associated with the development of *antenatal depression* (Lancaster, Gold, Flynn, Yoo, Marcus, & Davis, 2010). A recent German study of over 1,000 women confirms the effect of contextual factors, including educational status, monthly income, residential property status, and partnership status on depression symptoms both in the antenatal and postpartum period (Hein et al., 2013).

A 2011 study of two separate large samples of women in the Boston area ( $n= 2128, 1509$ ) found that maternal history of both physical and sexual abuse were independently associated with antenatal depression in the combined samples, with odds ratios of 1.48 (physical abuse) and 1.68 (sexual abuse) (Rich-Edwards et al., 2011). A past history of psychiatric disorder and domestic abuse during pregnancy have been implicated as contributing to later entry to prenatal care, and inadequacy of utilization (Kim et al., 2006). Antenatal depression is also associated with high levels of state-trait anxiety in pregnancy (Field et al., 2003; Leigh & Milgrom, 2008), stressful/adverse life events during pregnancy (Chung, Mathew, Elo, Coyne, & Culhane, 2008; Lancaster et al., 2010; Rubertsson, Waldenström & Wickberg, 2003), domestic violence

(Lancaster, et al., 2010), low self-esteem (Leigh & Milgrom, 2008; Ritter, Hobfoll, Lavin, Camron, & Hulsizer, 2000), low social support (Da Costa, Larouche, Dritsa, & Brender, 2000; Field et al., 2003; Lancaster, et al., 2010; Leigh & Milgrom, 2008; Rubertsson, Waldenström, & Wickberg, 2003), and poor relationship status with the woman's partner or infant's father (Glazier, Elgar, Goel, & Holzapfel, 2004; Zelkowitz et al., 2004).

**Outcomes related to antenatal depression.** Much more research to date has been directed to elucidating outcomes for mother and baby related to postpartum depression than antenatal depression. However, evidence has begun accumulating for antenatal depression as an independent risk factor for poor prenatal care, medical and obstetric complications, substance use, impaired bonding, and suicide (Andersson, Sundström-Poromaa, Wulff, Åström, & Bixo, 2004; Raudzus & Misri, 2008). Studies linking antenatal depression (exclusive of antidepressant medication exposure) to low birth weight and premature birth have been mixed; with one meta-analysis finding that antenatal depression does increase the risk, but the magnitude of the effect widely varies depending on the type of depression measurement, country location, and US socioeconomic status (Grote et al., 2010). A more recent meta-analysis finds evidence of links between antenatal depression and both premature delivery and decreases in breastfeeding initiation, but with modest effects (Grigoriadis et al, 2013).

There has been surprisingly little research looking at the effects of antenatal depression on development in children relative to the volume of studies which consider postpartum depression (Yonkers, et al, 2009). There has been one study finding depressive symptoms during pregnancy predicting children's high externalizing and total behavior problems (Luoma et al., 2001); these researchers also found evidence for exposure leading to antisocial behaviors in children (Hay, Pawlby, Waters, Perra, & Sharp, 2010). A recent article also finds that antenatal

depression predicted worse physical health for children, including immediate health issues during postnatally or during infancy, increased healthcare utilization in infancy, hospitalizations, physical limitations, or chronic illness by age 5 (Raposa, Hammen, Brennan, & Najman, in press). A small study found that the risk of depression for adolescents was 4.7 times greater if their mothers were depressed during pregnancy (Pawlby, Hay, Sharp, Waters, & O’Keane, 2009). This finding has been corroborated by a recently published report from the Avon Longitudinal Study of Parents and Children (ALSPAC) in the former Avon Health Authority in the west of England, which shows that antenatal depression may be an independent risk factor for offspring depression at age 18 (Pearson et al., 2013). Odds of offspring depression at 18 were 1.28 for antenatal depression and 1.26 for mothers with postpartum depression and who had low level of education; maternal education did not moderate the effect of antenatal depression. The authors suggest that treating depression during pregnancy therefore has the potential to prevent offspring depression as well.

What is clear from this review of antenatal depression is that there has not been the same sort of attention directed to depression in pregnancy as to postpartum depression, despite demonstrated adverse effects. Salient to this dissertation work, however, is the question of whether some amount of these effects might be attributed to the comorbidity between depression and posttraumatic stress, owing to the fact that both are not often considered simultaneously.

**Prevalence of postpartum depression.** Up to 80% of new mothers who have recently given birth develop a transitory postpartum depressive syndrome commonly referred to as the “*baby blues*” (Hansen, 1990). Although assessment and intervention may be appropriate for such women, most of these women will not go on to develop major depression. Some women may develop a much more serious condition known as *postpartum psychosis*, a rare disorder

occurring in 1-2 out of 1000 childbearing women, typically within the first 2-4 weeks after delivery (Sit, Rothschild, & Wisner, 2006). Postpartum psychosis may include paranoid delusions, mood swings, bizarre thinking, and grossly disorganized behavior (Sit et al., 2006; Wisner, 2006). I am not considering prevalence, assessment, or intervention for either the *baby blues* or *postpartum psychosis* in this dissertation analysis, and will instead focus on major depression and depressive symptomatology.

After childbirth, a 2005 review of studies found that the rate of depression was highest in the third month postpartum (12.9%), showed a slight decline in the fourth through seventh months (9.9-10.6%), and a decline to 6.5% after seven months (Gavin et al., 2005). A technical report and meta-analysis by many of these same authors (Gaynes et al., 2005) found lower rates of prevalence when studies utilizing self-report measures alone were excluded from analysis; they found that for major depression alone estimates ranged from 1.0% to 11% at different times during the postpartum period. Importantly, when looking at the limited data available regarding incidence of depression, these researchers found that as many as 14.5% of women had a new episode of major or minor depression during the first three months postpartum. Other researchers have found this increased risk for new onset in the early postpartum period; one study found that the odds of a new episode of major depression were three times that of a comparison group (Cox, Murray, & Chapman, 1993). A recent study regarding prevalence of postpartum depression found a 14% rate of women screening positive on self-report (Wisner et al., 2013).

**Risk factors related to postpartum depression.** Researchers have examined antenatal depression as a logical risk factor for postpartum depression. A large prospective study found a higher percentage (13.5%) of participants with scores on *EPDS* (Cox & Holden, 2003) above threshold for probable depression than for those same women during the early postpartum period

(Evans, Heron, Francomb, Oke, & Golding, 2001). Other studies have also shown that antenatal depression is a risk factor for postpartum depression (Da Costa et al., 2000; Evans, et al., 2001; Gotlib, Whiffen, Mount, Milne, & Cordy, 1989; Kim, Hur, Kim, Oh, & Shin, 2008; Leigh & Milgrom, 2008), particularly when co-morbid with posttraumatic stress (Seng, et al., 2013; Söderquist, Wijma, Thorbert, & Wijma, 2009). Other risk factors for postpartum depression include antenatal anxiety ('worry', state-trait anxiety) (Austin, Tully, & Parker, 2007), stressful life events during the pregnancy, low levels of social support, and a previous history of depression (Robertson, Grace, Wallington, & Stewart, 2004). Two recent studies have found that unintended pregnancy increased the risk for postpartum depression (McCrorry & McNally, 2013; Mercier, Garrett, Thorp, & Siega-Riz, 2013). Similarly to antenatal depression, a lifetime history of sexual or physical abuse has also been found to be associated with the development of *postpartum depression (PPD)* (Leeners et al., 2006; Records & Rice, 2009), although these studies did not measure PTSD, so we cannot know the contributory effect of any comorbidity.

**Outcomes related to postpartum depression.** Postpartum depression has been associated with adverse outcomes for infants and children, as well as mothers. A meta-analysis of antenatal depression found increased risk for pre-term birth and low birth weight, with the magnitude varying based on depression measurement, country location and US socioeconomic status (Grote et al., 2010). PPD has been shown to increase rates of impaired mother/infant bonding (Seng et al., 2013), insecure mother/baby attachments (Beck, 1995; Murray & Cooper, 1997), disturbed early mother/infant interactions (Field, 2010), poor infant cognitive performance on developmental assessments, and maternal perception of infants as more difficult temperamentally (Whiffen & Gotlib, 1989). Infants of depressed mothers score more poorly on social engagement measures and exhibit less mature regulatory behaviors (Feldman et al., 2009),

and are less responsive to faces and voices and show less negative response to still-face procedure at 3-5 months of age (Field, Diego, Hernandez-Reif, 2009). Affected dyads show lower quality of bonding (Moehler, Brunner, Wiebel, Reck, & Resch, 2006), ambivalent attachment (Kuscu et al., 2008), interactional disturbances (Hipwell, Goosens, Melhuish, & Kumar, 2000), and insecure attachment (McMahon, Kowalenko, & Tennant 2006; Shin, Park, & Kim, 2006). Longitudinal studies have demonstrated enduring effects of PPD, with children exhibiting more behavioral problems to age 9 (Luoma et al., 2001), lower IQ scores, more attentional problems, and difficulties in math reasoning at age 11 (Hay et al., 2001). Several caregiving activities appear to be compromised among depressed mothers as well, including feeding practices (particularly breastfeeding), sleep routines, well-child visits, vaccinations, and safety practices (Field, 2010; Zajicek-Farber, 2009). Maternal depression also appears to be highly correlated with depression in children, through age 15 (Hammen & Brennan, 2003) and age 18 (Pearson et al., 2013). Last, but certainly not least, an extremely adverse outcome for mothers, babies and their families alike is maternal suicide; which accounts for about 20% of postpartum maternal deaths (Lindahl et al., 2005). A recent study of 10,000 women showed that among those who screened positive for postpartum depression (1396 women = 14.0%), fully 19.3% endorsed self-harm ideation (Wisner et al., 2013).

Given the severely adverse outcomes related to postpartum depression for both mothers, babies and children, it is imperative that we increase the capacity of clinicians to predict which women might be at risk. This is a focus of this dissertation effort.



## Comorbidity of PTSD and Depression

The following section will review the comorbidity of PTSD and depression in the childbearing year, including diagnostic considerations and a thorough review of studies that measure both PTSD and depression in during pregnancy, specifically.

**Diagnostic considerations.** High rates of comorbidity between PTSD and depression have been noted in samples of veterans (e.g., 45% per Gros, Simms, & Acierno, 2010, in women with forcible rape history (69% per Zinzow et al., 2012), and in a nationally representative sample of US citizens (47.9%; Kessler et al., 1995). Explanations for the high rates of comorbidity between PTSD and depression have been advanced, centering on diagnostic symptom overlap, and shared latent negative emotional associations between the disorders, particularly for the dysphoria (feeling unwell or unhappy) items of the PTSD diagnosis (Biehn et al, 2013). Several studies reported results of factor analysis investigations in the run-up to the publication of the *DSM-5* (APA, 2013), and in fact these analyses are a significant part of the reason that the symptom clusters for PTSD have changed, favoring a 4-factor solution (versus the *DSM IV* (APA, 1994) three cluster solution) that includes intrusive re-experiencing, avoidance/numbing, negative alterations in cognitions and mood (a new category), and hyperarousal (Friedman, Resick, Bryant, & Brewin, 2011). Although the 4-factor solution was favored for the *DSM-5*, current analyses are instead favoring a 5-factor solution that splits the arousal cluster into two separate clusters which posits that difficulty sleeping, irritability, and concentration difficulties represent a “dysphoric arousal” cluster and that the remaining arousal items, like an exaggerated startle response, represent an “anxious arousal” cluster (Armour, Carragher, & Elhai, 2013). The *DSM-5* is being touted as a “living

document” that should be more open to incremental change than were the past versions, so it is likely that the diagnosis will continue to evolve.

Research into the factor structure of the major depression diagnosis is more limited; so far there appears to be support for a two-factor model that recognizes somatic and non-somatic factors (Krause, Reed, & McArdle, 2010; Elhai et al., 2012). The *DSM-5* (APA, 2013) has retained the *DSM-IV* (APA, 1994) criteria for diagnosis of major depressive disorder; although as more work is done to understand the latent constructs which underlie the comorbidity between PTSD and depression, this may also have implications for future *DSM-5* revisions (Contractor et al., 2013). New research posits that the trauma-depression link may be partially mediated by adult attachment style. This finding has implications for treatment when considering the role that adult attachment may play in establishing a therapeutic alliance between therapist and client (Fowler, Allen, Oldham, & Frueh, 2013).

As mentioned briefly in chapter one, clinicians who treat pregnant women have called for better understanding of the comorbidity between PTSD and depression for pregnant women. In a study of 190 urban mothers, 56 (29%) screened positive for depression, and 32 (17%) had symptoms consistent with PTSD (Silverstein et al, 2010). Of the women with depression, 14 (25%) had symptoms consistent with PTSD, and 31 of the women had both positive depression screens and a reported history of significant trauma. Of those women, 45% had symptoms consistent with PTSD. The authors of this brief report stress that the comorbidity between depression, trauma, and PTSD makes depression more difficult to diagnose, and more refractory to treatment. The Seng et al. (2009) STACY study found that major depression was in fact rare among those women who did not have PTSD; and has led to further investigation into this comorbidity via this dissertation analysis. In the overall STACY

sample of 1581 women, fully 84.5% of the women with antenatal depression were comorbid with full or partial PTSD. It is my goal to unpack this figure via dissertation analyses.

Diagnosis is important for researchers for establishing risk factors, outcomes, and interrelationships with comorbidities. It is also important in clinical settings where discovery of a diagnosis might foster understanding of a person's otherwise seemingly disparate constellation of symptoms and garner support for targeted treatment. Yet many clinicians may wonder about the relative value of insisting on full diagnostic criteria in order to develop a treatment plan for a symptomatic individual; and instead choose to respond to the person's distress, regardless of whether their symptom count meets a diagnostic threshold. In the course of a therapeutic relationship, or even at the beginning with a good therapist, distress might emerge more fully than it does when a woman is responding to a survey; many women will no doubt choose not to reveal their deepest pain on a survey or even a research interview. Therefore, it seems reasonable that any symptoms readily accounted as part of the research interview should be taken as clinically relevant. For this reason, I consider symptom counts for PTSD and the other continuous measures in the STACY dataset. As Chapter 5 will demonstrate, there is a case to be made that trauma-informed interventions should be developed that address trauma, posttraumatic stress, and depression simultaneously, and that there may be specific intergenerational implications in family context related to safety that should be taken into consideration.

**Comorbidity during pregnancy.** Following is an explication of studies (including the STACY study) which have considered comorbidity during pregnancy and included diagnostic measurement of both PTSD and depression. Table 1 illustrates pertinent information regarding selected studies.

Table 1  
*Studies which Measured Trauma, PTSD diagnosis, and Major Depression Diagnosis in a Perinatal Sample*

Author(s)	<i>n</i>	Current PTSD overall (%)	MDD overall (%)	Comorbid overall (%)
Kim et al., 2013	745	6.6	18.4	57.1
Cook et al., 2004	744	7.7	10.8	42.2
Morland et al., 2007	101	16.0	43.4	37.5
Rodriguez et al., 2008	210	23.9	59.9	n/a
Seng et al., 2009	1581	7.9	12.3	50.0
Smith et al., 2006	948	3.5	n/a	66.7
Söderquist et al., 2004	951	2.3	5.7	9.1

*Note:* n/a = not available

Kim, Harrison, Godecker and Muzyka (2013) recruited pregnant women at three clinics in Minnesota which served as Healthy Start sites, which are Federally Qualified Health Care Centers with a specific mandate to improve outcomes for communities with elevated risk for infant mortality. They recruited a diverse sample of 745 women, which included 58.7% African American women, 6.2% American Indian women, 7.9% Asian American women, 7.9% Hispanic women, and 10.2% white women. The primary purpose of the overall study was to validate an instrument to assess for overall prenatal risk, which they termed the “PRO.” They specifically sought to validate the depression, alcohol use, and drug use domains of the PRO against corresponding diagnoses as measured by the *Structured Clinical Interview for DSM-IV* (SCID; First, Spitzer, Gibbon, & Williams, 2005); and did find good evidence for validity of this instrument (Harrison, Godecker, & Sidebottom, 2012). However, the researchers also assessed for PTSD, and were therefore positioned to report on the comorbidity of PTSD and depression in a subsequent analysis (Kim et al., 2013). The study participants completed both the PRO and the SCID, which revealed that 43.8% of the sample reported trauma but had insufficient symptoms for meeting a diagnostic category. As seen in Table 1, 6.6% of the sample met the diagnostic criteria for PTSD, and another 4.2% met criteria for

“subthreshold” PTSD, meaning that they endorsed a total of 3 rather than 6 symptoms. Combining those who were subthreshold with those who met diagnosis showed that more than half of the sample (54%) reported a trauma which met PTSD criteria. The rate of comorbidity with depression was 57.1%, with risk for several items assessed as part of the PRO, including not only depression but also housing instability, drug use disorder (cigarettes), and involvement with child protection services, statistically significantly increased for those with PTSD. Importantly, this study suggests that the symptom profile for women with subthreshold PTSD were clinically similar to those who met full diagnostic criteria for PTSD, and suggests that the distinction between the two may not be clinically relevant.

The Kim (et al., 2013) is similar to the STACY study in that it assessed for trauma and both depression and PTSD in a racially and ethnically diverse pregnancy sample. Both were community samples and also assessed for other psychosocial risk factors. Yet the studies differ in design – the Kim et al. study was cross-sectional in nature, and reported findings for pregnancy risk alone, whereas the STACY study was a longitudinal cohort design, and has been thus able to report on longitudinal analyses that encompass birth outcomes and psychiatric status in the postpartum period as well. The Kim et al. study also differs from the STACY study because there was a mixture of first-time mothers (primiparous) with mothers having subsequent children (multiparous) in the Kim et al. study whereas the STACY study restricted recruitment to primiparous mothers in order to control for the effect of traumatic previous births or psychosocial stressors related specifically to parenting. Additionally, the Kim et al. study did not use a specific trauma measurement to assess trauma exposures, relying instead on open-ended questions; the STACY study did use standardized instruments. Another difference between the studies is that the Kim et al. interviews were conducted in person by

one research assistant, whereas the STACY participants were interviewed by phone by one of a team of interviewers. Moving forward, the Kim et al. study has implications for my dissertation analyses, including the importance of looking at housing instability to the extent possible in the STACY study as an influential factor, and the advisability of looking at subthreshold PTSD as a clinically important category.

Cook and colleagues (2004) also endeavored to assess the prevalence of PTSD in a sample of 744 African American (57.5%) and white (42.5%) women in the St. Louis area who attended services at the Women, Infants, and Children Supplemental Nutrition Program (WIC) sites in St. Louis and the surrounding rural counties. Women were assessed for psychiatric disorders, pregnancy history and environmental stressors. Cook et al. (2004) found that 7.7% of women in the study met diagnostic criteria for PTSD, that 10.8% met diagnosis for depression, and that another 8.6% of women were nicotine dependent. A total of 42.2% of women had PTSD comorbid with depression. Although assessments used in this study were similar to the STACY study, and the authors report that their design is prospective in nature, there are not as yet reports in the literature for any longitudinal analyses with which to make direct comparisons to the STACY study. An important finding of the Cook et al. study is that although many women met diagnostic criteria for PTSD, very few had undergone any treatment related to that condition.

Another study which looked specifically at the comorbidity between PTSD and depression in a childbearing sample is the Morland et al. (2007) study. This study of 101 Hawaiian pregnant women assessed for PTSD and depression using standardized measures. Their sample included Asian American women (40%), Caucasian women (20%), and Native Hawaiian/Pacific Islander women (39%) recruited in early pregnancy as part of a longitudinal

study (preliminary findings were first reported in Goebert, Frattarelli, Onoye, & Matsu, 2007). Overall, Morland et al. (2007) showed a rate of 16.0% PTSD diagnosis, 43.4% with depression, and 37.5% women experiencing comorbid PTSD/depression. The major limitation of this study is the sample size, which did not permit multivariate analysis of childbirth outcomes; however, this team of researchers did find that those women in the sample with either PTSD or “subclinical” PTSD had a three-fold risk of having PTSD in the postpartum period, and those women were also at increased risk for “stress” (73%), anxiety (64%), and depression (73%) in the postpartum period (between 4 to 8 weeks postpartum) (Onoye, Goebert, Morland, Matsu, & Wright, 2009). Although little explanation is given for the reason why, these researchers also collapsed their “subclinical” or subthreshold PTSD cases together with those meeting full diagnosis. Importantly, the researchers found that women with history of interpersonal violence were twice as likely to have postpartum PTSD.

Another study which looked at PTSD and depression in an ethnically diverse sample was the Rodriguez et al. (2008) study. This sample of 210 pregnant Latina women in Los Angeles, California was selected for a two-cohort analysis divided between those who did and those who did not have histories of IPV exposure to complete assessments for IPV, trauma exposures, PTSD, depression, resilience, chronic stress, alcohol and tobacco usage. Not surprisingly, the research team found that those women who had experienced IPV were more at risk for PTSD and depression, controlling for age, language of interview, and site effects. Sample size did not permit complicated analysis; although logistic regressions were performed, they predicted PTSD and depression separately. Therefore, information regarding the specific rate of comorbidity is not available in the published report.

Another study with a high percentage of Latina women (52.7%) was a study of those who received care at one of three federally-funded obstetric clinics in the New Haven, Connecticut area (Smith, Poschman, Cavaleri, Howell, & Yonkers, 2006). Spearheaded by Yale University researcher Yonkers, this study compared the psychiatric status of pregnant women with selected results for non-pregnant traumatized women using data from the National Comorbidity Study (Kessler et al., 1995). Using standardized instruments, the research team found an incidence rate of 3.5% (current PTSD) and a prevalence rate of 29.3% (lifetime PTSD). Depression was reported in relation to PTSD diagnosis only, so it is not known what the overall rate of depression was in the sample; however, 66.7% of those with PTSD also had depression, which represented a four-fold risk. Other factors associated with PTSD were suicidality, panic disorder, sexual molestation, and physical attacks/assaults. In direct comparisons with the National Comorbidity non-pregnant sample, the Smith et al. (2006) study found that pregnant women were less likely to endorse re-experiencing symptoms of PTSD, and were more likely to report “loss of interest” and “irritability” symptoms. The study continued to recruit subjects; in a subsequent paper the research team reported enrollment of 1,326 women total and analysis of available outcomes data for 1,100 of the sample (Rogal et al., 2007). Postpartum analyses centered on risk for prematurity, finding a positive but non-statistically significant risk for PTSD but a significant risk for minor depression (OR = 1.82). Low rates of psychiatric morbidity have been found in other studies of Hispanics (e.g., Grant et al, 2004); and the comparatively lower rates of PTSD in this sample may have resulted in limited power to realize statistical significance.

A Swedish study also assessed trauma, PTSD, and depression in a single longitudinal design; however, the measurements that were used restricted the diagnosis of PTSD to whether



or not the woman was symptomatic with traumatic stress related to the forthcoming delivery (Söderquist, Wijma, & Wijma, 2004). Remarkably, the researchers found that 2.3% (1.1% for nulliparous women, 3.2% for multiparous women) of 951 women assessed met *DSM-IV* (APA, 1994) diagnostic criteria for PTSD related to the impending birth; another 5.8% (3.2% nulliparous, 7.5% multiparous) were subthreshold for PTSD. A more specific measurement of fear of impending childbirth was also administered, and 13.5% of women endorsed “severe” fear of childbirth. Rates of depression were lower in this study than others, with 5.7% of the sample reporting depression in early pregnancy. The rate of comorbidity between PTSD diagnostic criteria and depression was 9.1%. There was also a strong correlation with high trait anxiety scores. The authors suggest that the symptoms seen in this study suggest a kind of “pre” traumatic stress related to childbirth. However, although this study did assess for trauma, PTSD and depression in the same sample, there is very little sociodemographic information provided with which to make direct comparisons to the STACY sample. The sample reports no racial ethnic diversity, and appears to be largely composed of well-educated women who were either married or cohabitating with a partner.

Taken together, the STACY study appears to be unique among studies which have undertaken simultaneous measurement of trauma, PTSD, and depression in pregnancy in that it a) recruited a sample large enough to permit multivariate analyses, b) was longitudinal, c) was diverse, d) included “gold standard” measures of trauma, PTSD, and depression, including the full range of types of trauma exposure, e) collected many sociodemographically relevant variables and f) controlled for the effect of previous traumatic birth by recruiting only nulliparous women. Shortcomings of the STACY study include its cohort design which resulted in dismissal of women with subthreshold PTSD from longitudinal data collection,

lack of a dimensional measure of depression (diagnosis or probability scores are available, but not symptom scores), lack of a measure of housing instability, and limited ability to model markers of specificity in regards to neighborhood disadvantage like physical incivility, territoriality and social spaces (Laraia et al., 2006).

### **Summary of Literature on Trauma, Stress, and Mental Health during the Childbearing Year**

Yehuda, McFarlane, & Shalev (1998) suggest that the notion that PTSD is but one possible outcome of trauma exposure will not surprise mental health workers. However, the contributory role of trauma to depression diagnosis and recovery has not been fully explored. As previously discussed, there is considerable variability in the very definition of trauma and/or stress as it relates to childbearing women. Despite some calls for combining depression with ‘anxiety’ measures, there is also considerably wide interpretation as to how anxiety is defined in any given study, and considerable inconsistency in how trauma/stress/adversity is measured. Moreover, studies which examine depression and PTSD together are still rare; it does still seem to be the case that trauma and PTSD are treated in one silo of research whereas stress and depression (albeit with the more recent addition of disorders like phobias and state/trait anxiety) are treated in another silo.

Yet there are studies which suggest that it is time to start to apply a trauma-informed lens more specifically to depression. For instance, the Yehuda et al. (1998) study found that cortisol response to acute trauma in emergency rooms may lead to a psychiatric disorder other than PTSD, including depression. Kendler, Gardner, & Prescott (2002) presented findings of structural equation models for MDD that suggests that the development of the risk for the disorder results from three broad pathways, including internalizing symptoms, externalizing

symptoms, and psychosocial adversity. However, findings from the Breslau, Davis, Peterson & Schultz (2000) epidemiologic study of young adult members of a health maintenance organization contrasted with the Yehuda et al. (1998) study. They found an increased risk for major depression for persons with PTSD, but not in trauma-exposed persons without PTSD. However, these were not perinatal samples and did not address what specific trauma exposures were related to current diagnosis.

Overall, this review of the literature suggests that both PTSD and depression have strong linkages with interpersonal (or “relational”) trauma specifically, and with cumulative trauma (“weathering”), and that it is worthwhile to consider specificity of trauma exposures related to comorbidity and to *T-EPD*, including childhood sexual abuse/maltreatment, and IPV. The literature also has identified several poor outcomes for women and babies, particularly related to prematurity and poor postpartum mental health, which has implications for infant mental health, mother/baby dyadic attachment, and development of children. I have used this review as a basis for consideration of choice of variables for analysis, and will outline those choices in the next chapter, focused on dissertation methodology.

### **Chapter 3 Methodology**

This chapter will provide more information about the STACY study upon which this secondary analysis is based and will explicate the methodology utilized for each of the study's aims and hypothesis testing.

#### **Human Subjects Review**

The STACY study is governed by the institutional review boards of the hospital and university settings where the data were collected. Prior to beginning dissertation analysis I submitted an additional application to the Wayne State University Institutional Review Board for human subjects review. Because these data have been de-identified, and the confidentiality of its participants is not threatened, and because the STACY study has continually been under review, the Wayne State University Institutional Review Board concurs that this research is exempt from additional review (See Appendix B).

#### **STACY Study**

**Sample.** The data for this secondary analysis comes from a community sample of 1581 sociodemographically diverse pregnant women who were participants in Dr. Julia Seng's NIH-funded project, "Psychobiology of PTSD & Adverse Outcomes of Childbearing;" NIH/NINR R01 NR008767; known as the "STACY" (Stress, Trauma and the Childbearing Year) project. The mean age of the participants was 26 years old; and 42.6% of participants reported that they did not reside with a husband or other domestic partner. Table 2 provides additional sociodemographic characteristics of the study participants, including race/ethnicity, educational attainment, and household income. Of particular note is the high level of diversity in the sample, including nearly 45% African American participants, and the fact that over 57% of the sample reported a household income less than \$50,000 per year.

Table 2  
*STACY Participant Sociodemographic Characteristics (n = 1581)*

Characteristic	%	Frequency
<b>Race/Ethnicity</b>		
Latina	4.2	67
Middle Eastern	2.3	36
African American	44.8	709
European American	45.9	725
Asian American	7.1	113
Native Hawaiian/Pacific Islander	0.4	7
American Indian/Alaska Native	1.5	23
Other	3.2	51
<b>Education Level</b>		
< High School	6.5	103
High School Equivalency	39.7	628
Associate Degree	8.7	137
Bachelor Degree	21.3	337
Master Degree	16.4	259
Professional Degree/Doctorate	7.1	112
Refused	0.3	5
<b>Household Income</b>		
<&15,000 per year	22.6	357
\$15,000-\$25,000	17.0	268
\$25,000-\$50,000	17.5	277
>\$50,000	37.9	599
Refused	5.1	80

**Data available for analysis.** The purpose of the STACY study was to test the hypothesis that PTSD is associated with adverse outcomes of childbearing. It was a prospective three-cohort study combining data from three structured interviews in early pregnancy, late pregnancy, and six weeks postpartum. Details of data collection procedures and methodology are available in the prevalence report (Seng et al., 2009) and are summarized here as well.

Participants were recruited from August, 2005 to May, 2008 at three large health systems in Michigan. Eligible women were 18 and older, initiating prenatal care, expecting their first child, and able to speak English. The first wave of interview data was collected on an unselected sample of women who responded to an invitation at their intake prenatal visit to participate in a survey about “stressful things that happen to women, emotions, and pregnancy.” The project then

followed three cohorts longitudinally: women with lifetime diagnosis of PTSD (PTSD cases), those with trauma exposure who did not develop PTSD (trauma-exposed controls), and those with no trauma exposures (non-exposed controls). A fourth cohort ( $n=532$ ) included women who did not match one of the three cohort definitions and were not followed longitudinally.

Outcomes data include 6-week postpartum interviews ( $n=564$ ) and medical chart abstractions ( $n=839$ ). Salivary cortisol samples were collected and analyzed for a sub-sample of participants ( $n=395$ ), and DNA samples were also collected for a genetics supplementary grant and are still being analyzed.

Table 3 depicts those STACY measures used for dissertation analyses; it is organized to show which measures are providing data to represent the factors in the conceptual framework outlined in the preceding chapters. Several measures were used during the early prenatal interview. These include The Life Stressor Checklist (Wolf & Kimerling, 1997), which was used to assess trauma exposure. It was chosen for the STACY study because it is considered to have high sensitivity to trauma among women, and has demonstrated test-retest reliability from 79% to 98% for individual items (Norris & Hamblen, 2004). The National Women's Study PTSD Module (NWS-PTSD; Kilpatrick, Resnick, Saunders, & Best, 1989) was used to assess PTSD symptoms and both lifetime and current (acute) diagnosis. This measure has been compared favorably with the face-to-face clinician-administered Structured Clinical Interview for DSM-III-R, attaining a sensitivity of 0.99 and specificity of 0.79 (Kilpatrick et al., 1994; Resnick et al., 1993). Diagnosis for major depressive disorder and generalized anxiety disorder were ascertained via the Composite International Diagnostic Interview short form (CIDI; Wittchen, 1994). The CIDI is a structured diagnostic tool designed for use by trained interviewers who are not clinicians, and is supported by extensive field trial data on cross-national reliability and

validity (Wittchen, 1994). The Symptom Checklist-90-Revised (SCL-90-R; Derogatis, 1977/2000) is a self-report inventory which was used to reflect somatization and interpersonal sensitivity. The SCL-90-R has been used extensively worldwide in over 2,000 published studies and has been translated into at least 30 languages, with multiple analyses providing evidence of construct validity and reliability (Derogatis & Unger, 2010). The Dissociative Experiences Scale-Taxonomic Version (DES-T; Bernstein & Putnam, 1986) was used to ascertain symptoms of dissociation. Psychometric evidence for the DES-T includes excellent convergent validity with other dissociation measures across studies and impressive predictive validity concerning dissociative disorders and traumatic experiences (van IJzendoorn & Schuengel, 1996). The STACY study utilized the Perinatal Risk Assessment Monitoring Survey to assess sociodemographic indicators including income, education, age, and race/ethnic identity (Beck et al., 2002) and used the 2000 Federal Bureau of Investigation uniform crime report for each woman's ZIP code using Simply Maps ([simplymaps.com](http://simplymaps.com), retrieved May 20, 2009).

Measures used from the STACY postpartum interview for these dissertation analyses include the Postpartum Bonding Questionnaire (PBQ; Brockington et al., 2001) and the Postpartum Depression Screening Scale (PDSS; Beck & Gable, 2002). PTSD was also reassessed at this interview, using the NWS-PTSD (Kilpatrick et al., 1989) previously described. The PBQ was used to screen for mother's bonding impairment, with a sensitivity of 0.93 and specificity of 0.99, compared with structured diagnostic interview (Brockington et al., 2001). The PDSS was the measure used to screen for postpartum depression. Psychometric evidence for the PDSS includes sensitivity of 0.78 and specificity of 0.99, with overall positive predictive value of 0.93 as compared with structured diagnostic interviews (Beck & Gable, 2002).

Table 3  
*STACY Measures and Variables Included in the Dissertation Study*

Factor	Instrument and reference	Variables created
Trauma exposure	Life Stressor Checklist (Wolfe & Kimerling, 1997)	Child abuse prior to age 16 defined as completed rape, molestation, physical abuse, emotional abuse/neglect, or physical neglect
PTSD	National Women's Study PTSD Module (Resnick et al., 1993)	Current and lifetime PTSD diagnoses and symptom counts assessed pre- and also post- delivery for new trauma
MDD	World Health Organization's Composite International Diagnostic Interview (CIDI) depression module (Wittchen, 1994)	Major depression in the past year
Somatization	Symptom Checklist-90-Revised (SCL-90-R) (Derogatis, 1977/2000)	Somatization and interpersonal sensitivity scale scores
Dissociation	Dissociative Experiences Scale-Taxonomic version (DES-T) Bernstein & Putnam, 1986)	Dissociative symptomatology
Anxiety	World Health Organization's Composite International Diagnostic Interview (CIDI) generalized anxiety module (Wittchen, 1994)	Generalized anxiety diagnosis (GAD)
Life event stress factors	CDC Perinatal Risk Assessment Monitoring System (PRAMS) survey items (Beck et al., 1999); Intake zip code analysis/FBI uniform crime report (simplymaps.com, retrieved May 20, 2009)	Risk factors for adverse obstetric outcomes identified in the literature: African American race per self-report Poverty defined as income <\$15,000 Low level of education per self-report Age per self-report and per prenatal record Residence in high-crime neighborhood Partner status per self-report
Adverse maternal outcomes	Postpartum Bonding Questionnaire (PBQ) (Brockington et al., 2001)	Poor bonding with infant defined as lower scores on PBQ
	Postpartum Depression Screening Scale (PPSS) (Beck & Gable, 2002)	Postpartum Depression per symptom score
	National Women's Study PTSD Module (Resnick et al., 1993)	Postpartum PTSD

**Adequacy of the database for dissertation analysis.** Because many of the women with depression but not PTSD were not followed longitudinally, I was unable to create models which consider all aspects of Seng's (2002) conceptual model simultaneously. Not considered are *non-modifiable medical and obstetric risk factors* such as pregnancy complications and *behavioral alterations* such as substance use, which were collected at medical chart review for the longitudinal cohorts only, and *neuroendocrine alterations* in the form of calculated scores for



diurnal cortisol distributions collected from a longitudinal subsample only. I constrained analyses to data available at WAVE 1 in early pregnancy ( $n = 1581$ ) for variables related to trauma exposure, PTSD and MDD diagnoses and symptom counts, sociodemographic risk factors, and comorbidities including interpersonal sensitivity scores, dissociation scores, somatization scores, and generalized anxiety diagnosis, and used six-week postpartum WAVE 3 interviews ( $n = 564$ ) exclusively for the prediction of postpartum depression and bonding impairment.

Although the STACY team reported rates of PTSD, MDD, and their comorbidity (Seng et al, 2009), the team did not consider the specificity of trauma exposure in depressed women, which is a novel focus of this project. Preliminary analyses show that in 194 cases of antenatal depression *only 30 individuals* had no trauma exposure (*MDD-Only*). The remaining 84.5% ( $n = 164$ ) meet MDD diagnosis with trauma exposure but do not meet criteria for lifetime or current PTSD. This sub-group shows the hypothesized trauma-exposed perinatal depression (*T-EPD*).

## **Dissertation Study**

**Aims & hypotheses.** My *overall objective* is to bridge the gap between previous investigations of perinatal PTSD and MDD. The *overall strategy* is to highlight comorbid symptomatology and to test the concept of *T-EPD* via secondary analysis. Following I will restate the aims and hypotheses introduced in Chapter 1 and then provide an explanation of the analytic methodology for each set of analyses.

***Aim I: To determine the extent to which it is warranted to integrate perinatal mental health care to combine attention to depression, trauma, and posttraumatic stress.***

*Hypothesis Ia: Some women who meet diagnosis for major depression will meet the criterion for index trauma exposure (“Trauma-Exposed Perinatal Depression” [T-EPD]), and will be subthreshold for PTSD (will have at least three symptoms of PTSD but not meet the full diagnostic criteria).*

*Hypothesis Ib: Antenatally depressed women with index trauma exposure (T-EPD) will differ from antenatally depressed women without index trauma exposure by type of traumas endorsed overall (index or not), sociodemographic risk factors, and other comorbid conditions.*

*Hypothesis Ic: Symptoms of PTSD and the number of traumas endorsed by women will be better predictors of postpartum depression than symptoms of depression in pregnancy.*

**Methodology for Aim I, Hypothesis Ia.** Analyses for Aim I, Hypothesis Ia, are descriptive in nature; they portray frequencies and percentages for membership in the *a priori* trauma-based diagnostic category variable based on *DSM-IV* (APA, 1994) PTSD and MDD criterion, which also serves as the dependent variable for Hypothesis Ib. These analyses also allow us to see index trauma endorsement in each category of the variable *trauma-based diagnosis* based on rank order of endorsement. The procedure for creating the dependent variable follows.

First, I established whether or not each participant had endorsed a significant index trauma (the *DSM-IV* ‘A1’ criterion – their “worst” or “second worst” trauma), and whether or not they experienced “fear, helplessness, or horror” (the *DSM-IV* ‘A2’ criterion). An exception to this was made by the STACY study co-investigators and maintained for dissertation analyses - in the case of childhood physical or sexual abuse prior to age 16; the A2 criterion was not required. This is owing to the fact that many children are made to feel complicit in their abuse, and may not have characterized the abuse in this manner; this seems a particularly reasonable decision in the light of the fact that this criterion is no longer required at all in *DSM-5* (APA, 2013).

After establishing whether or not a participant endorsed an index trauma that met A1 and A2 criteria (with the exceptions just noted), I created the *trauma-based diagnosis* six-level categorical variable based on whether the individual met MDD or PTSD full criteria per *DSM-*

IV. Although the DSM-IV (APA, 1994) makes a distinction between lifetime PTSD (chronic) and current PTSD (acute), this distinction was eliminated in the DSM-5 (APA, 2013); I therefore included any participant who met lifetime PTSD (chronic) diagnosis. This resulted in 6 categories: *No-Trauma*, *Trauma-Only*, *MDD-Only*, *T-EPD*, *PTSD-Only*, and *Comorbid*.

It is important to clarify that membership in the *No-Trauma* group, which serves as the reference category for comparisons in the multinomial logit analyses, does not suggest that the individuals in this group endorsed no traumatic exposures in their lifetime, but rather denotes that whatever trauma they experienced did not in their estimation rise to the level of an index trauma consistent with the qualifying A1 and A2 criteria for PTSD diagnosis. Membership in the ‘Trauma-Only’ group is comprised of those individuals who *did* have an index trauma but did not meet diagnosis for either PTSD or MDD. Membership in the *MDD-Only* group is comprised of individuals who met MDD diagnosis, but did *not* have an index trauma and did *not* have PTSD diagnosis; members of the *T-EPD* (‘Trauma-Exposed Perinatal Depression’) group also met MDD diagnosis but not PTSD diagnosis but *did* have an index trauma. Members of the *PTSD-Only* group had an index trauma and met diagnosis for PTSD but *not* MDD; members of the *Comorbid* group had all three: index trauma, PTSD diagnosis, and MDD diagnosis. Table 4 reiterates these distinctions.

Table 4

*Basis for Determining Categories of the Dependent Variable 'Trauma-Based Diagnosis'*

Category	Endorsed index trauma per A1 & A2 criteria for PTSD per <i>DSM-IV</i>	Met all diagnostic criteria for PTSD per <i>DSM-IV</i>	Met all diagnostic criteria for MDD per <i>DSM-IV</i>
No-Trauma	-	-	-
Trauma-Only	X	-	-
MDD-Only	-	-	X
T-EPD	X	-	X
PTSD-Only	X	X	-
Comorbid	X	X	X

*Note: DSM-IV = Diagnostic and Statistical Manual of Mental Disorders (3<sup>rd</sup> ed.) (APA, 1994), PTSD = Posttraumatic Stress Disorder, MDD = Major Depressive Disorder*

After creating the dependent variable, I produced descriptives (frequencies, means, and standard deviations) for posttraumatic stress symptom counts across the six categories. Because I am interested in seeing whether distinctions between these groups center on subthreshold PTSD, I wanted to ascertain specifically whether members of the *T-EPD* group had levels greater than three symptoms, consistent with other published reports for sub-threshold PTSD (e.g., Breslau, Lucia, & Davis, 2004).

I then proceeded to exploring the specificity of index trauma exposures across the categories. I produced frequencies and percentages for index trauma exposures across all categories and for the sample as a whole.

**Methodology for Aim I, Hypotheses Ib.** For this hypothesis, I used a simultaneous multinomial logit procedure to examine associations in the sample ( $n= 1581$ ) based on elements of the Seng model using data from the early pregnancy interviews (Wave 1). Multinomial logit is a logistic regression procedure specific to analyses which have more than two categories of outcome. The procedure examines the contribution of individual predictors in order to predict membership in the six-category dependent variable.

*Variables used.* In order to see more distinctive features of the overall cumulative trauma exposure I used variables in the STACY dataset which divide the trauma exposures conceptually. Examination of the literature suggests that trauma exposures of an interpersonal nature such as childhood abuse (both physical and sexual abuse contact/penetration), adult rape (both contact and penetration), and emotional or physical neglect will be associated with both PTSD and depression. I therefore chose a variable representing *abuse trauma* (any endorsement of trauma exposures of an interpersonal nature such as childhood physical abuse and sexual abuse contact/penetration, adult sexual abuse contact/penetration, and emotional or physical neglect) to reflect interpersonal trauma of an abusive nature.

Because losses and separations are some of the most commonly endorsed traumatic experiences in the STACY dataset (see Chapter 4 results), I chose a variable representing *loss/separation trauma*, which includes death, sudden death, and separations (parents divorced, participant divorced, being fostered/adopted). Additionally, because this is a perinatal sample, I chose a variable representing *reproductive trauma*, meaning that the participant reported having had a traumatic experience with a reproductive loss (miscarriage or abortion) prior to this pregnancy. In order to see the contributions of other types of endorsed traumas not captured by *abuse trauma*, *loss/separation trauma* and *reproductive trauma*, I created a variable representing

a continuous count of the *other traumas* endorsed, including manmade or natural disaster, war zone exposure, seeing or being in an accident, seeing or being in a robbery, having a family member jailed or being jailed, caring for someone with a serious illness, undergoing a painful medical or ritual procedure, witnessing domestic violence, and “other (unspecified)” trauma.

To represent the effect of sociodemographic factors in analytic models, I used a five-level variable frequently used in STACY analyses to represent cumulative sociodemographic factors commonly associated with risk for adverse perinatal outcomes, including African American race, poverty, low educational attainment, being a teen (in this case constrained to being 18-20 years old, since adulthood was an inclusion criteria), and residence in a high-crime neighborhood. In addition to the cumulative five-level risk factor variable, I included a dichotomous variable representing whether or not the participant was living with a domestic partner in early pregnancy or not.

The remaining independent variables included in analyses were those for other comorbid conditions, including continuous counts of symptoms of *somatization*, *interpersonal sensitivity* and *dissociation*, and a dichotomous variable for meeting diagnostic threshold for *generalized anxiety disorder*.

*Data cleaning and model fitting.* In order to assess the relationships of the candidate variables for the multinomial logit procedure, I examined bivariate associations of predictor variables with my dependent variable *trauma-based diagnosis*. All predictor variables were found to be statistically significant at the bivariate level (See Table 5).

Table 5

*Point-Serial Correlations and Pearson Product-Moment Correlations Among and Descriptive Statistics for Key Study Variables*

	n (%)	DV	Abuse	Repro	Loss	Partner	GAD	IPS	Dissoc	Soma	PTSD	SES	Other
DV		1											
Abuse			1										
yes = 553 (35)													
no = 1028 (65)													
Repro				1									
yes = 220 (14)													
no = 1361 (86)													
Loss					1								
yes = 299 (82)													
no = 282 (18)													
Partner						1							
yes = 949 (60)													
no = 1512 (96)													
GAD							1						
yes = 69 (4)													
no = 1512 (96)													
	<u>M(SD)</u>												
IPS	4.76 (2.40)							1					
Dissoc	6.33 (5.54)								1				
Soma	6.33 (5.54)									1			
PTSD	4.17 (4.29)										1		
SES	1.78 (1.83)											1	
Other	2.42 (2.17)												1

*Note:* DV = dependent variable (trauma-based diagnosis), Abuse = abuse trauma, Repro = reproductive loss, GAD = generalized anxiety diagnosis, IPS = interpersonal sensitivity score, SOMA = somatization score, SES = index of cumulative socioeconomic risk, Other = all other trauma types. \*  $p < .05$ . \*\*  $p < .01$ .

Multinomial logistic regression does not require assumptions of normality, linearity, or homogeneity of variance for the independent variables. However, missing data and outliers are potentially problematic. The STACY data were extensively cleaned for missing data, so that is not an issue. The statistical program used, SPSS (version 22), does not calculate outlier diagnostics for multinomial logistic regression, so the general advice (e.g., Hosmer, Lemeshow, & Sturdivant, 2013) is to run a series of multiple binary logistic regressions with pairs of categories of the dependent variable and to test the exclusion of outliers or influential cases. In my case, because my dependent variable has 6 categories, this resulted in 15 separate logistic regressions. To assess outlier influence, a comparison is made of overall classification prediction with outliers in versus outliers out; if removing outliers improves classification by more than 2%, it is recommended that they be removed. Assessment of outliers for the preliminary model showed that the group 1 (*Trauma-Only*) to group 3 (*T-EPD*) had 1 case with a Cook's distance  $>1$  and 32 cases with standardized residuals  $>3$ . This represented just *under* a 2% change in the overall classification of the model (from 67.6% to 68.6%). But a separate analysis of the outliers showed that the 33 cases represented nearly half of the *T-EPD* cases, and so from a numerical and also a theoretical standpoint I determined it was important to leave these cases included in the analyses. No other bivariate logistic regressions produced significant classification accuracy change. Thus, I proceeded to examination of full model.

I first hand-calculated the proportional by-chance accuracy of the model to be .40339, or 40.34%. The threshold for assessing model classification accuracy is a rate 25% or greater than the proportional by-chance accuracy; making the proportional by-chance accuracy criteria 50.42%. With a model classification accuracy of 67.6%, this criterion was fulfilled.



The next criterion for model accuracy is a minimum ratio of valid cases to independent variables of at least 10 to 1, with a preferred ratio of 20 to 1. With 11 predictor variables and 5 categories calculated (the 6<sup>th</sup> category, *No-Trauma* is the reference category), this represents a total of 55 predictor variables. Thus with 1581 cases, a ratio of 29 cases to 1 independent variable was achieved, satisfying this requirement.

Multicollinearity in the model was assessed through examination of the standard errors of the b coefficients; standard errors  $> 2.0$  are indicators of numerical problems, as are improbable (very large) b coefficients. There were no standard errors  $> 2.0$  or improbable b coefficients.

In terms of predictive capacity, although overall classification of the model was a statistically significant improvement over by-chance accuracy (67.6% versus 40.34%), it is nonetheless unimpressive (i.e., predicting only 6.7% of *MDD-Only* cases and 1.5% of *T-EPD* cases, and 11.3% of *Comorbid* cases). While there was fairly good prediction for the *No-Trauma* (70.2%), *Trauma-Only* (79.2%), and *PTSD-Only* (77.5%) categories, there was poor prediction to the depressed categories. However, this is a customary finding of multinomial logit analyses, and does not disallow examination of the full, fitted, and statistically significant model, which I will undertake in the next chapter.

***Methodology for Aim I, Hypothesis 1c.*** For Aim I, Hypothesis 1c, I employed receiver-operator growth curve modeling (ROC) techniques to assess the degree to which knowing a pregnant woman's trauma history, PTSD, and depressive symptom load might predict meeting diagnostic criteria for postpartum depression. ROC modeling is based on the simultaneous measure of sensitivity ("true positive") and specificity ("true negative") for all possible cutoff points. Sensitivity and specificity pairs for each possible cutoff point are calculated, sensitivity is plotted on the y axis by (1-specificity) on the x axis. The curve that is created is called the

receiver operating characteristic (ROC) curve. The ‘*area under the curve*’ (AUC) represents the probability that the outcome for a randomly chosen positive case will exceed the result for a randomly chosen negative case; area under the ROC curve ranges from 0.5 and 1.0 with larger values indicative of better fit.

Preparatory to undertaking the ROC analyses, I created standard score variables for the continuous measures of cumulative trauma, posttraumatic stress symptoms, and depression probability scores. Results of ROC modeling will also be presented in the next chapter.

***Aim II: To explore the implications of DSM-5 (APA, 2013) changes in qualifying trauma criteria in relation to perinatal mental health.***

*Hypothesis IIa: Constraining the trauma criteria per DSM-5 guidelines will reduce the rate of PTSD prevalence in the STACY sample, and will thereby reduce the rate of comorbidity between PTSD and MDD.*

*Hypothesis IIb: Those individuals who no longer meet PTSD diagnostic criteria per DMS-5 will have similar levels of postpartum depression.*

*Hypothesis IIc: Those individuals who no longer meet PTSD diagnostic criteria per DMS-5 will have similar levels of bonding impairment to those individuals who do meet DSM-5 criteria.*

***Methodology for Aim II, Hypotheses IIa-IIb.*** Analyses for Aim II (Hypotheses IIa through IIc) beg the logical question in response to Aim I, “If trauma history and PTSD are central to perinatal mental health, what are the implications of DSM-5 (APA, 2013) changes in qualifying trauma criteria?”

The STACY data were collected under *DSM-IV* (APA, 1994) trauma/PTSD criteria; now that the *DSM-5* (APA, 2013) has been published we must consider how these data might have

salience for the future. This is particularly relevant to examining the association of trauma with depression (or PTSD for that matter), since the definition of a significant trauma has changed under *DSM-5* and has constricted the types of allowable traumas that are considered to be disturbing enough to lead to PTSD. An example is the death of a loved one; per *DSM-5* criteria this is no longer considered a trauma unless the death was accidental or violent.

*Establishing DSM-5 trauma criterion.* Fortunately, trauma measures used in the STACY study were comprehensive in nature and allow for reanalysis of diagnostic case-ness based on the *DSM-5* (APA, 2013) criteria. However, because of the wide variety of interpretations of trauma/stress evidenced in the literature (see Chapter 2), I prefer to not add to this general confusion and instead make the determination of ‘allowable *DSM-5* trauma’ in a manner which might demonstrate content validity for such a determination.

One way that many researchers have endeavored to establish content validity for various instruments is use of the ‘content validity index’ procedure (CVI), which involves establishing a panel of experts to evaluate your measure and provide a rating score for the items and then assessing percentage agreement among the experts to determine the most worthy items. Often attributed to an education specialist initially (Martuza, 1977), the procedure has been used frequently in the nursing literature (Polit & Beck, 2006). Polit & Beck (2006) have offered recommendations for using the CVI, including distinguishing between content validity at the item and at the scale levels, and reporting the range of values for items retained in the resultant scale. They also recommended standards for excellency for content validity that include 100% item agreement for panels comprised of 3 to 5 experts, 78% agreement for items with panels of 6 to 10 experts, and overall scale agreement of 90% for panels of any size.

As mentioned in Chapter 2, trauma measurements that will help clinicians and researchers to establish A1 criteria for PTSD per *DSM-5* (APA, 2013) are currently being constructed (there is no longer any ‘A2’ criteria per *DSM-5*). Because these measures are not yet available to use to make direct comparisons to the STACY trauma measure, I convened a CVI panel of professionals expert in PTSD and diagnostic criteria to examine the *Life Stressor Checklist* (Wolfe & Kimerling, 1997) items used by the STACY study and to rate whether each specific trauma would be “allowable” per DSM-5 trauma criteria. I defined “experts” as those clinicians or researchers who have published extensively on trauma and/or PTSD or are clinicians with more than five years of experience specifically working with trauma survivors and who use *DSM* diagnostic criteria on a regular basis. I provided them with a copy of the *DSM-5* PTSD trauma criteria and a list of potential traumas that the STACY used as part of the Life Stressor Checklist (Wolfe & Kimerling, 1997) and asked them to check a box beside each item to indicate whether or not they would consider that item to be an “allowable *DSM-5* trauma” (See Appendix C).

I used the items that the panel considered as allowable with 78% agreement, with two exceptions. A close reading of the diagnostic formulation in the *DSM-5* (APA, 2013) convinced me that although “sudden death” and “painful medical or ritual procedure” only had 71% agreement as allowable among the panel, that these two items should be nonetheless included. In fact, “sudden death” is used as the most frequent example of an allowable trauma in journal articles and on websites such as the National Center for PTSD, to point out that with the advent of *DSM-5* there is a concerted effort to distinguish between “death,” which many think leads more to depression than PTSD, and “sudden death,” which can certainly be traumatic. Similarly, the diagnostic differential for PTSD in *DSM-5* allows for medical procedures that are “sudden

catastrophic events” and as such, I made the determination to lower the threshold for agreement to 71% for these items. The next highest percentile “yes” agreement for other items was 57%.

*Establishing DSM-5 MDD criterion.* Although there have been changes to the PTSD diagnosis in the *DSM-5* (APA, 2013), the MDD diagnosis has remained unchanged. Therefore, no revisions were necessary for examination of antenatal depression respondent to *DSM-5*.

*Establishing DSM-5 PTSD criterion.* Broad changes to PTSD include its removal from the anxiety-related disorders to the trauma and stress-related disorders section, and the trauma criteria changes outlined previously. Additionally, a new dissociative subtype has been created, as well as a separate set of diagnostic criteria for diagnosing PTSD in children under six years old. A summary of changes to the PTSD cluster criteria is also included in Appendix A.

While the STACY data have several items to choose from in terms of trauma exposures, it is missing two of the new *DSM-5* (APA, 2013) PTSD D symptom cluster items, including ‘persistent distorted blame of self or others for causing the traumatic event or for resulting consequences’ and ‘persistent negative trauma-related emotions’ (e.g., fear, horror, anger, guilt, or shame). Although there is no data with which to approximate the ‘persistent distorted blame’ item, I was able to use one of the items from the STACY depression measure to approximate the ‘negative emotions’ item. There were two questions in the *CIDI depression module* (Wittchen, 1994) which addressed the ‘guilt’ and ‘shame’ aspects that were not part of the *DSM-IV* (1994) PTSD diagnostic criteria which asked respondent whether they ‘sometimes feel down on themselves, no good, or worthless.’ This question was asked twice; I counted endorsement of this item either time as a PTSD D cluster symptom per *DSM-5*.

Further PTSD criterion, including duration, and distress, were also available in the STACY data and included in the algorithm to create the *DSM-5* approximation.

After creating an algorithm for inclusion in the new *DSM-5* (APA, 2013) PTSD diagnosis, I also created a new variable with three categories representing 1) those individuals who did not meet PTSD diagnosis via either *DSM-IV* (APA, 1994) or *DSM-5*, 2) those who once met diagnosis via *DSM-IV* but no longer do, and 3) those who meet diagnosis via the new *DSM-5* criteria. I then used this variable to perform one-way ANOVA analyses comparing postpartum depression scores and bonding impairment scores across the three PTSD diagnostic categories.

**Strengths and limitations.** There are many areas of strength for the STACY study and for these dissertation analyses. As previously mentioned in Chapter 2, the STACY study appears to be unique among studies which have undertaken simultaneous measurement of trauma, PTSD, and depression in pregnancy and a) was theory-driven, b) recruited a sample large enough to permit multivariate analyses, c) was longitudinal, d) was diverse, e) included “gold standard” measures of trauma, PTSD, and depression, including the full range of types of trauma exposure, f) collected many sociodemographically relevant variables and g) controlled for the effect of previous traumatic birth by recruiting only nulliparous women. There is also strength in its diversity; oversampling of disadvantaged women fosters generalizability despite attrition. Strengths of the dissertation include highlighting the comorbidity between PTSD and MDD in a perinatal population and the novel focus on trauma association with depression in a context which attempts to alleviate some of the variability in defining trauma/stress.

There are limitations inherent to secondary analysis, including the inability to access potentially important variables. Although the STACY measures are extensive, a study designed at the outset to investigate *T-EPD* might include measures of cognition or response styles, including rumination (Nolen-Hoeksema et al., 2008). Another limitation is that I am only able to make statistical predictions regarding associations of trauma with depression and

not temporal prediction, since the exact timing of both the trauma and the depression cannot be determined. Also of concern is estimating level of error; this is minimized by the study's stringent data collection monitoring and inter-rater reliability procedures resulting in agreement of 94.4% for chart abstraction (Seng et al., 2011). Because STACY was concerned with obstetric effects of PTSD, it oversampled for sociodemographic risk, which means that there was more exposure to abuse trauma, and more PTSD. As a result, the proportion of cases with *MDD-Only* may be underestimated. However, rates of depression in the STACY sample do appear to mirror nationally representative rates among women (Substance Abuse and Mental Health Services Administration, 2009).

Other limitations of the STACY study include its cohort design, which resulted in dismissal of women with subthreshold PTSD from longitudinal data collection, lack of a dimensional measure of depression (diagnosis or probability scores are available, but not symptom scores), and lack of measures for other salient contextual factors such as housing instability, physical incivility, territoriality and social spaces (Laraia et al., 2006). Despite these limitations, there is a robust variety and richness of data with which to conduct secondary analyses.

## Chapter 4 Results

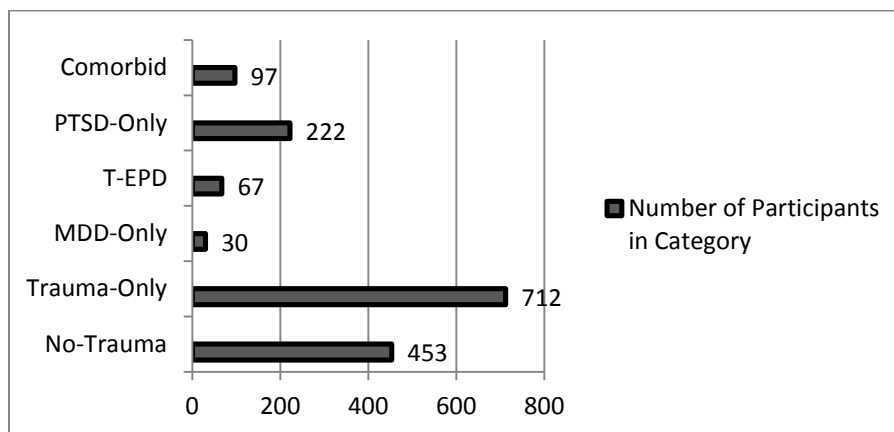
Following are the results of hypothesis testing; implications of these results will be discussed in Chapter 5.

### Aim I Analyses

**Aim I: To determine the extent to which it is warranted to integrate perinatal mental health care to combine attention to depression, trauma, and posttraumatic stress.**

*Hypothesis Ia: Some women who meet diagnosis for major depression will meet the criterion for index trauma exposure (“Trauma-Exposed Perinatal Depression” [T-EPD]), and will be subthreshold for PTSD (will have at least three symptoms of PTSD but not meet the full diagnostic criteria).*

This hypothesis was supported; I found that 67 women (4.2% of the total sample of 1581) were members of the *T-EPD* group, meaning that they met diagnostic criteria for MDD, had a trauma exposure, but did not meet criteria for PTSD. Only 30 women had MDD but no trauma exposure. The individuals in the *T-EPD* group did evidence subthreshold PTSD, as predicted, as did members of the *MDD-Only* group, despite the fact that they did not have a trauma exposure that met the A2 criterion.



*Figure 2.* Number of participants in each category of dependent variable *trauma-based diagnosis*.



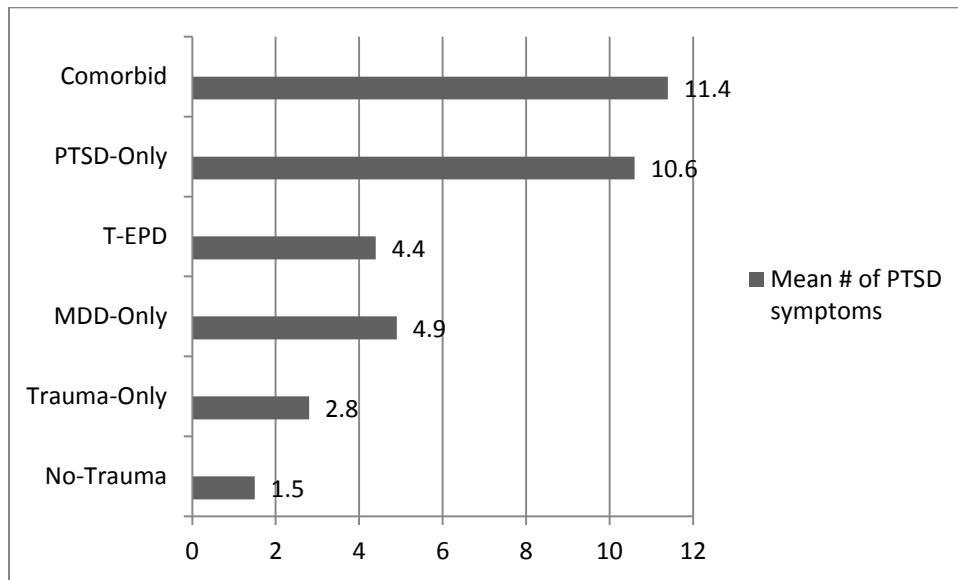


Figure 3. Mean number of PTSD symptoms in each category of the dependent variable *trauma-based diagnosis*.

I then conducted a one-way between subjects analysis of variance (ANOVA) in order to compare posttraumatic stress symptom scores across all six categories of the dependent variable *trauma-based diagnosis*. There was a significant effect of trauma-based diagnostic status on posttraumatic stress symptoms [ $F(5, 1575) = 561.17, p < .001$ ]. Post hoc comparisons using the Dunnett T3 test indicated that there were statistically significant mean score differences for posttraumatic stress symptoms for all comparisons *except* for that between the *MDD-Only* ( $M = 4.93, SD = 4.21$ ) and the *T-EPD* group ( $M = 4.45, SD = 2.72$ ), which was not statistically significantly ( $p = .96$ ).

I then proceeded to investigate the types of traumas endorsed across the categories of the dependent variable *trauma-based diagnosis*. Across all categories, either death of a loved one, or sudden death of a loved one, was the most highly endorsed exposure. Beyond that, the rank order of traumas differed quite a bit across the categories. Of particular note is that the *T-EPD* group had a much higher percentage of endorsement of having had a reproductive loss than the *MDD-*

*Only* group, and that the *MDD-Only* group had very low endorsement of abuse traumas (Table 6). Figures 4 through 9 provide a visual depiction of the types of index traumas in each group which were endorsed by participants at least 10% of the time. Note that the traumas endorsed in the *No-Trauma* and *MDD-Only* categories, although denoted as “worst” or “second worst” ever experienced, did not meet criteria A2 in order to be considered an index trauma.

Table 6

*Side by Side Comparison of Index Traumas Endorsed within each Trauma-Based Diagnostic Category*

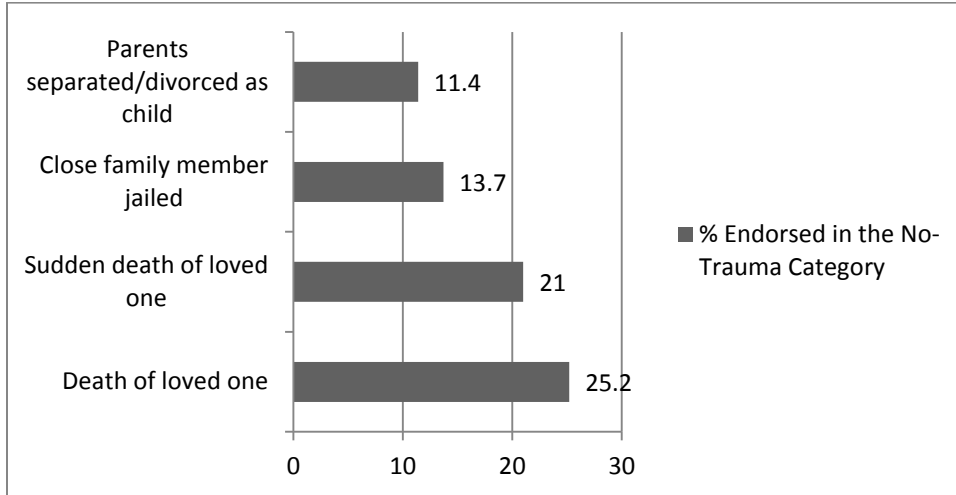
Trauma Type	Overall Sample n=1581	No- Trauma <sup>a</sup> n=453	Trauma- Only n=712	MDD- Only <sup>a</sup> n=30	T-EPD n=67	PTSD- Only n=222	Co-Morbid n=97
death of loved one (not sudden)	394(24.9)	114 (25.2)	198 (27.8)	10 (33.3)	23 (34.0)	42 (19.0)	7 (7.3)
sudden death of loved one	376(23.8)	95 (21.0)	182 (25.6)	6 (20.0)	16 (24.0)	52 (23.4)	25 (25.8)
parents separated/divorced	204(12.9)	52 (11.4)	114 (15.7)	3 (10.0)	7 (10.5)	24 (10.9)	4 (4.2)
witnessed domestic violence as child	152(9.6)	19 (4.2)	94 (13.2)	2 (6.7)	7 (10.5)	21 (9.5)	9 (9.4)
close family member jailed	149 (9.4)	62 (13.7)	50 (7.0)	6 (20.0)	6 (9.0)	14 (6.3)	11 (11.3)
hard time w EAB/SAB	138(8.7)	16 (3.6)	58 (8.2)	1 (3.3)	10 (15.0)	34 (15.3)	19 (19.6)
caregiver for someone w/ serious illness	118(7.4)	30 (6.6)	59 (8.3)	3 (10.0)	4 (6.0)	17 (7.7)	5 (5.1)
involved in serious accident	107 (6.7)	13 (2.9)	64 (9.0)	2 (6.7)	6 (9.0)	16 (7.3)	6 (6.2)
had serious money problems	96 (6.0)	14 (3.1)	48 (6.7)	1 (3.3)	6 (9.0)	13 (5.9)	14 (14.4)
child sexual abuse--contact	90 (5.7)		50 (7.1)		4 (6.0)	25 (11.3)	11 (11.3)
other trauma (not specified)	84 (5.4)	5 (1.1)	50 (7.1)		4 (6.0)	18 (8.1)	7 (7.3)
emotional abuse/neglect	83 (5.3)	4 (0.8)	29 (4.1)	1 (3.3)	4 (6.0)	30 (13.6)	15 (15.4)
witnessed serious accident	78 (4.9)	14 (3.0)	48 (6.7)	3 (10.0)	2 (3.0)	9 (4.1)	2 (2.1)
was robbed/attacked	69 (4.3)	1 (0.2)	48 (6.7)		3 (4.5)	11 (5.0)	6 (6.2)
adult physical abuse	56 (3.5)	7 (1.6)	27 (3.8)	1 (3.3)	2 (3.0)	14 (6.4)	5 (5.2)
child sexual abuse--penetration	44 (2.8)		23 (3.2)		1 (1.5)	13 (5.9)	7 (7.3)
childhood physical abuse	43 (2.8)		21 (2.9)		2 (3.0)	14 (6.3)	6 (6.2)
sexual harassment	41 (2.6)	7 (1.5)	23 (3.2)		2 (3.0)	4 (1.8)	5 (5.2)
saw robbery/attack	39 (2.5)	5 (1.1)	23 (3.3)		2 (3.0)	7 (3.2)	2 (2.1)
adult sexual abuse--penetration	31 (2.0)	1 (0.2)	12 (1.6)	1 (3.3)	4 (6.0)	10 (4.6)	3 (3.1)
was separated/divorced	29 (1.9)	2 (0.4)	16 (2.3)		2 (3.0)	7 (3.2)	2 (2.1)
was fostered/adopted	27 (1.7)	9 (2.0)	12 (1.7)			4 (1.8)	2 (2.1)
had a very serious illness	25 (3.6)	5 (1.1)	20 (2.8)	1 (3.3)	6 (9.0)	17 (7.7)	7 (7.3)

*(Table 6 continues)*

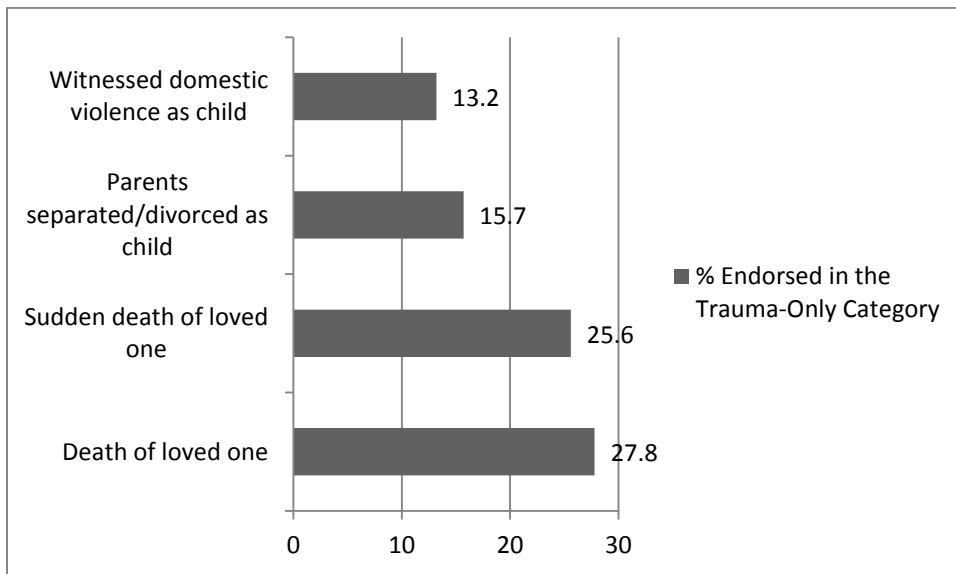
(Table 6 continued)

adult sexual abuse--contact	21 (1.3)		12 (1.7)			5 (2.3)	4 (4.1)
was in natural or man-made disaster	21(1.3)	1 (.2)	11 (1.5)		2 (3.0)	6 (2.7)	1 (1.0)
was jailed	20 (1.2)	4 (0.9)	9 (1.2)	1 (3.3)	1 (1.5)	3 (1.4)	2 (2.1)
had painful medical/ritual procedure	15 (0.9)		11 (1.5)			3 (1.4)	1 (1.0)
was physically neglected	5 (0.4)		2 (0.3)			2 (1.0)	1 (1.0)
was in war zone	4 (0.3)		4 (0.5)				

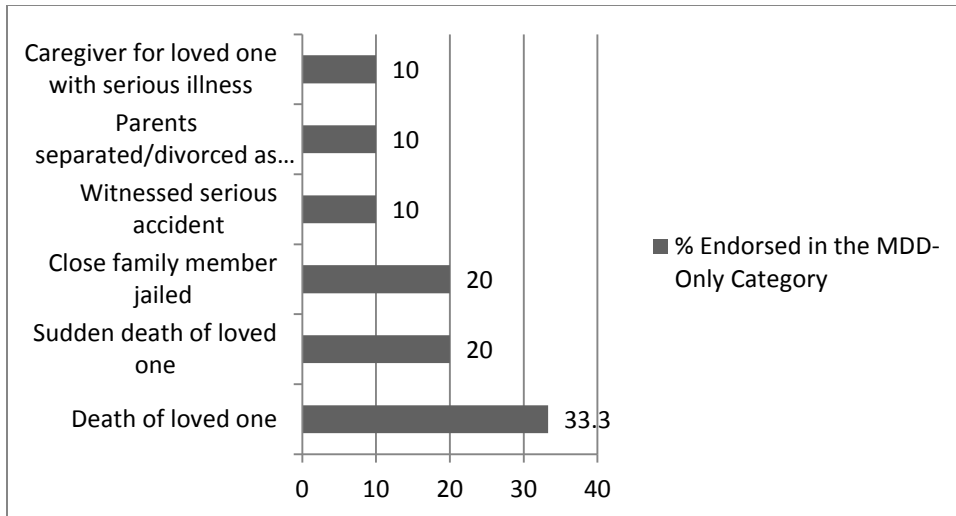
<sup>a</sup>Trauma exposures endorsed by those in the *No-Trauma* and *MDD-Only* categories were designated as “worst/second worst” but did not meet threshold for index trauma because they did not meet the DSM-IV PTSD diagnostic A2 “fear, helplessness, or horror” criterion.



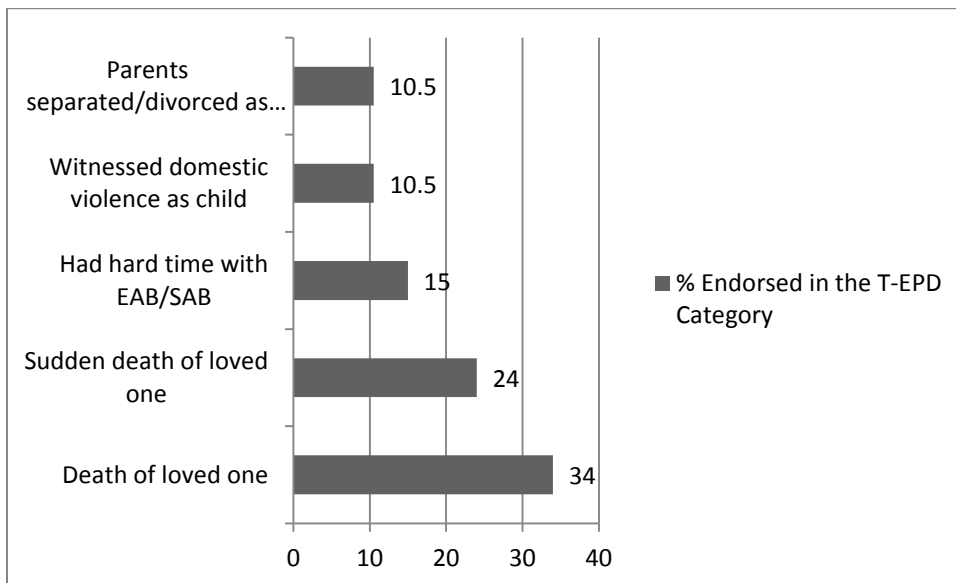
*Figure 4.* Type of endorsement of index trauma by women in the *No-Trauma* category greater than 10% of the time. Note that in this category the trauma endorsed did *not* meet the A2 criterion.



*Figure 5.* Type of endorsement of index trauma by women in the *Trauma-Only* category greater than 10% of the time.



*Figure 6:* Type of endorsement of trauma by women in the *MDD-Only* category greater than 10% of the time. Note that in this category the trauma endorsed did *not* meet the A2 criterion.



*Figure 7.* Type of endorsement of index trauma by women in the *T-EPD* category greater than 10% of the time.

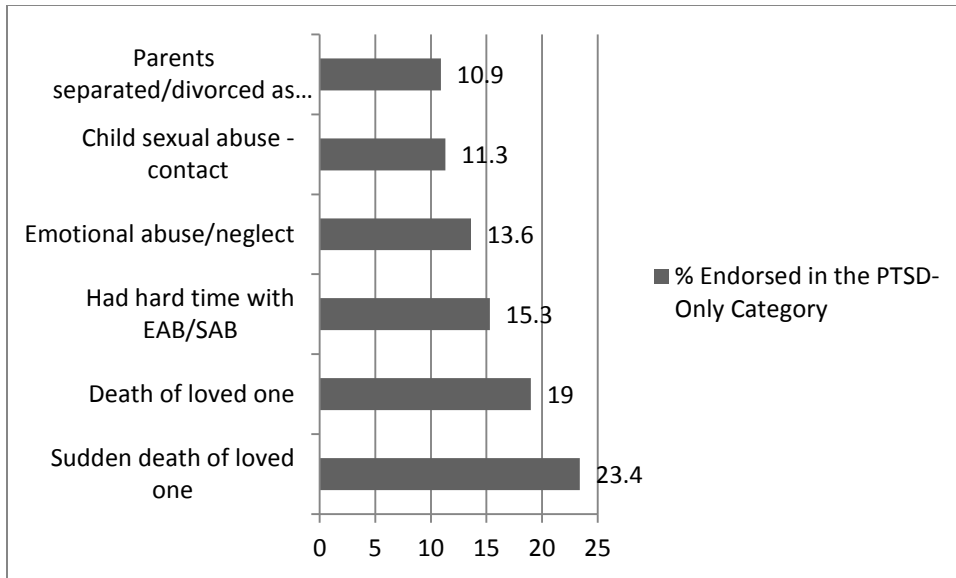


Figure 8. Type of endorsement of index trauma by women in the *PTSD-Only* category greater than 10% of the time.

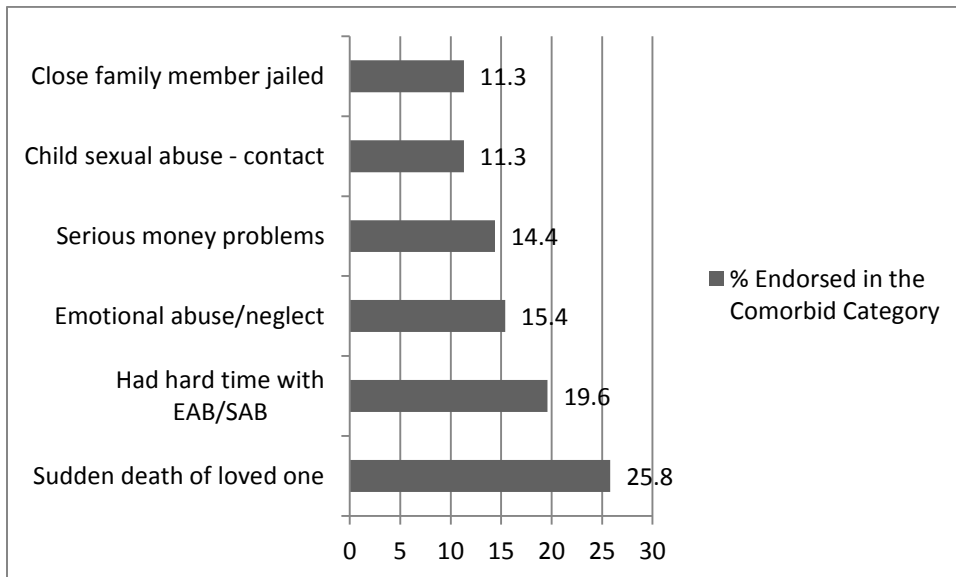


Figure 9. Type of endorsement of index trauma by women in the *Comorbid* category greater than 10% of the time.

*Hypothesis 1b: Antenatally depressed women with index trauma exposure (T-EPD) will differ from antenatally depressed women without index trauma exposure by type of traumas endorsed overall (index or not), sociodemographic risk factors, and other comorbid conditions.*

The relationship between the dependent variable *trauma-based diagnosis* and the combination of independent variables overall was ascertained through assessment of the model chi-square statistic. The relationship was found to be statistically significant (-2 LL 2631.292,  $X^2 = 1671.61$ , df 55,  $p < .001$ , Nagelkerke pseudo  $R^2 = .70$ ). The reference category for the trauma-based diagnosis dependent variable was the *No-Trauma* group.

Examination of overall significance of the relationship between specific independent predictors and the dependent variable revealed that all predictors were statistically significant at  $p < .01$ , except for *interpersonal sensitivity*, *dissociation* score, and *partner status*. Because these variables were part of the conceptual model and because removing them represented a significant change in the chi-square for the model as a whole, I chose to leave these variables in the final model.

Table 7 shows the results of the model fitting and overall comparisons between the independent variables considered and the dependent variable *trauma-based diagnosis* as a whole. Tables will follow (8 through 12) which will portray the comparisons between each overall statistically significant predictor for each individual *category* of the *trauma-based diagnosis* dependent variable.



Table 7

*Model Fitting Criteria and Likelihood Ratio Tests for Multinomial Logistic Regression*

	-2 Log Likelihood	X <sup>2</sup>	df	p
Intercept	2631.29	<.001	0	
Interpersonal Sensitivity score	2641.771	10.480	5	.063
Dissociation score	2637.392	6.100	5	.297
Somatization score	2647.643	16.351	5	.006
PTSD symptoms	3256.683	625.391	5	<.001
Cumulative sociodemographic risk	2682.176	50.884	5	<.001
Sum of other traumas	2764.682	133.390	5	<.001
Abuse trauma	2680.069	48.777	5	<.001
Reproductive trauma	2654.694	23.402	5	<.001
Loss/separation trauma	2670.127	38.835	5	<.001
Partnered	2633.945	2.654	5	.753
Generalized anxiety	2646.728	15.436	5	.009

Women in the *Trauma-Only* category (compared to the *No-Trauma* reference group and controlling for the confounding effects of the other categories) were less likely to have potential sociodemographic risk factors, and were more likely to have higher levels of all types of trauma exposures (Table 8).

Table 8

*Logistic Regression Analysis of Trauma-Based Diagnosis as a Function of SES, Trauma, PTSD, and other Comorbidities: Non-Exposed ('No-Trauma') Study Participants Versus those with Trauma-Exposure but no PTSD or Depression ('Trauma-Only')*

Variables	$\beta$	Wald $\chi^2$ -test	Odds Ratio	95% Confidence Interval for Odds Ratio	
				<i>Lower</i>	<i>Upper</i>
SES cumulative risk index	-0.42	42.87	0.66***	0.58	0.74
Has a life partner	-0.02	0.01	0.98	0.94	1.02
Endorsed abuse trauma	1.37	39.71	3.95***	2.58	6.05
Endorsed reproductive trauma	0.75	7.25	2.12**	1.23	3.68
Endorsed loss/separation trauma	1.04	33.79	2.83***	1.99	4.02
Endorsed other type of trauma	0.64	101.56	1.90***	1.67	2.15
Lifetime PTSD symptom count	0.05	2.37	1.08	0.98	1.13
Dissociation score	-0.12	0.13	0.98	0.89	1.09
Interpersonal Sensitivity score	0.03	2.30	0.98	0.94	1.02
Somatization score	-0.02	1.15	0.98	0.94	1.02
Meets Generalized Anxiety diagnosis	1.55	2.06	4.69	0.57	38.82

\* $p < .05$  \*\* $p < .01$  \*\*\* $p < .001$

Women in the *MDD-Only* category (compared to the *No-Trauma* reference group and controlling for the confounding effects of the other categories) were less likely to have symptoms of somatization, more likely to have higher levels of PTSD symptoms, more likely to have

‘other’ trauma (but had no statistically significant relationship with abuse, reproductive, or loss traumas), and were more likely to meet diagnosis for generalized anxiety (Table 9).

Table 9

*Logistic Regression Analysis of Trauma-Based Diagnosis as a Function of SES, Trauma, PTSD, and other Comorbidities: Non-Exposed (‘No-Trauma’) Study Participants Versus those with MDD-Only*

Variables	$\beta$	Wald $\chi^2$ -test	Odds Ratio	95% Confidence Interval for Odds Ratio	
				Lower	Upper
SES cumulative risk index	-0.52	0.09	0.95	0.68	1.33
Has a life partner	-0.57	0.79	0.56	0.16	1.99
Endorsed abuse trauma	0.93	3.69	2.53	0.98	6.52
Endorsed reproductive trauma	-1.26	1.36	0.28	0.03	2.36
Endorsed loss/separation trauma	-0.12	0.06	0.89	0.34	2.30
Endorsed other type of trauma	0.30	4.41	1.35*	1.02	1.79
Lifetime PTSD symptom count	0.33	18.58	1.39***	1.20	1.62
Dissociation score	0.11	2.34	1.12	0.97	1.28
Interpersonal Sensitivity score	0.09	4.68	1.10*	1.01	1.19
Somatization score	-0.16	8.13	0.85**	0.76	0.95
Meets Generalized Anxiety diagnosis	3.03	6.00	20.71*	1.83	234.06

\* $p < .05$  \*\* $p < .01$  \*\*\* $p < .001$

Women in the *T-EPD* category (compared to the *No-Trauma* reference group and controlling for the confounding effects of the other categories) were more likely to have higher levels of PTSD symptoms, less likely to have potential sociodemographic risk factors, less likely

to have symptoms of somatization, and were more likely to have abuse trauma, reproductive trauma (but had no statistically significant relationship with loss trauma), and were more likely to meet diagnosis for generalized anxiety (Table 10).

Table 10

*Logistic Regression Analysis of Trauma-Based Diagnosis as a Function of SES, Trauma, PTSD, and other Comorbidities: Non-Exposed ('No-Trauma') Study Participants Versus those with Trauma-Exposed Perinatal Depression ('T-EPD')*

Variables	$\beta$	Wald $\chi^2$ -test	Odds Ratio	95% Confidence Interval for Odds Ratio	
				Lower	Upper
SES cumulative risk index	-0.47	14.52	0.62***	0.49	0.79
Has a life partner	-0.31	0.52	0.73	0.31	1.71
Endorsed abuse trauma	0.92	6.96	2.52**	1.27	5.00
Endorsed reproductive trauma	1.21	9.05	3.35**	1.52	7.37
Endorsed loss/separation trauma	0.45	1.45	1.56	0.75	3.23
Endorsed other type of trauma	0.62	40.70	1.85***	1.53	2.24
Lifetime PTSD symptom count	0.25	18.90	1.29***	1.15	1.44
Dissociation score	-0.06	0.53	0.94	0.79	1.11
Interpersonal Sensitivity score	0.04	1.05	1.04	0.97	1.11
Somatization score	-0.00	0.02	1.00	0.94	1.06
Meets Generalized Anxiety diagnosis	2.95	6.80	19.08**	2.08	175.14

\* $p < .05$  \*\* $p < .01$  \*\*\* $p < .001$

Women in the *PTSD-Only* category (compared to the *No-Trauma* reference group and controlling for the confounding effects of the other categories) were more likely to have higher

levels of PTSD symptoms (understandably), less likely to have potential sociodemographic risk factors, less likely to have symptoms of somatization, and were more likely to have abuse trauma, reproductive trauma, and “other” trauma (but had no statistically significant relationship with loss trauma) (Table 11).

Table 11

*Logistic Regression Analysis of Trauma-Based Diagnosis as a Function of SES, Trauma, PTSD, and other Comorbidities: Non-Exposed (‘No-Trauma’) Study Participants Versus those with PTSD-Only*

Variables	$\beta$	Wald $\chi^2$ -test	Odds Ratio	95% Confidence Interval for Odds Ratio	
				<i>Lower</i>	<i>Upper</i>
SES cumulative risk index	-0.49	18.13	0.61****	0.49	0.77
Has a life partner	0.01	0.00	1.01	0.45	2.25
Endorsed abuse trauma	1.34	16.58	3.83****	2.01	7.32
Endorsed reproductive trauma	1.00	6.28	2.71*	1.24	5.90
Endorsed loss/separation trauma	6.48	1.29	1.62	0.70	3.75
Endorsed other type of trauma	0.65	56.27	1.92****	1.62	2.27
Lifetime PTSD symptom count	1.00	190.20	2.72****	2.36	3.14
Dissociation score	-0.04	0.39	0.96	0.88	1.10
Interpersonal Sensitivity score	0.04	1.97	1.05	0.98	1.11
Somatization score	-0.08	7.38	0.93**	0.88	0,98
Meets Generalized Anxiety diagnosis	1.93	2.84	6.92	0.73	65.63

\* $p < .05$  \*\* $p < .01$  \*\*\*\* $p < .001$

Women in the *Comorbid* category (compared to the *No-Trauma* reference group and controlling for the confounding effects of the other categories) were more likely to have higher levels of PTSD symptoms (understandably), less likely to have potential sociodemographic risk factors, less likely to have symptoms of somatization, more likely to have abuse trauma, reproductive trauma, and “other” trauma (but had no statistically significant relationship with loss trauma), and were more likely to meet diagnosis for generalized anxiety (Table 12).

Table 12

*Logistic Regression Analysis of Trauma-Based Diagnosis as a Function of SES, Trauma, PTSD, and other Comorbidities: Non-Exposed (‘No-Trauma’) Study Participants Versus those with PTSD Comorbid with MDD*

Variables	$\beta$	Wald $\chi^2$ -test	Odds Ratio	95% Confidence Interval for Odds Ratio	
				<i>Lower</i>	<i>Upper</i>
SES cumulative risk index	-0.43	10.19	0.65**	0.50	0.89
Has a life partner	0.34	0.52	1.41	0.56	3.54
Endorsed abuse trauma	1.68	17.18	5.36***	2.42	11.86
Endorsed reproductive trauma	1.57	13.04	4.83***	2.05	11.34
Endorsed loss/separation trauma	1.13	3.58	3.08	0.96	9.90
Endorsed other type of trauma	0.71	55.86	2.03***	1.69	2.45
Lifetime PTSD symptom count	1.01	144.80	2.75***	2.33	3.24
Dissociation score	-0.06	0.70	0.94	0.81	1.09
Interpersonal Sensitivity score	0.08	6.22	1.09*	1.02	1.16
Somatization score	-0.06	4.48	0.94*	0.88	0.99
Meets Generalized Anxiety diagnosis	2.35	4.02	10.52*	1.06	104.88

\* $p < .05$  \*\* $p < .01$  \*\*\* $p < .001$

Taken as a whole, the results of this multinomial logit analysis provide partial support for Hypothesis Ib. Analyses show that while women in the *T-EPD* category differ from women in the *MDD-Only* by type of trauma exposure, and sociodemographic factors, they look similar when taking other comorbid factors into consideration, including somatization symptoms and generalized anxiety diagnosis.

When considering the bivariate associations of cumulative sociodemographic risk with the trauma-based diagnosis dependent variable, we see that each category shows some level of risk, and that the *MDD-Only* group has the highest mean of risk factors. (Figure 10).

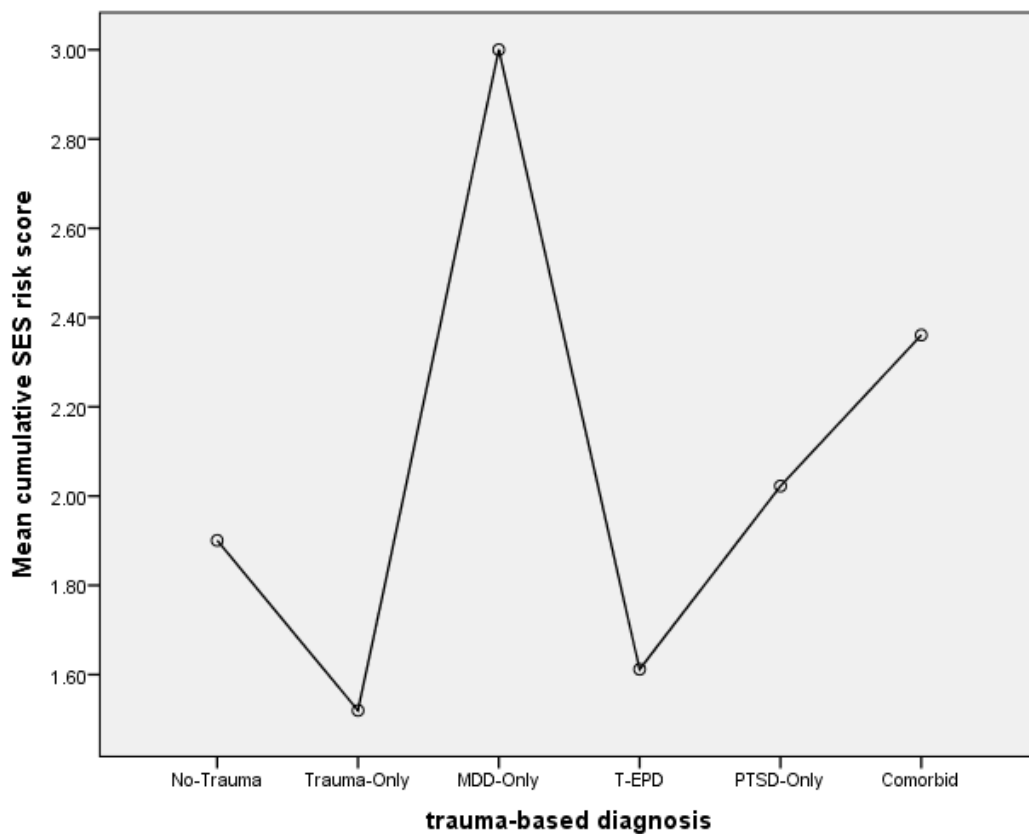


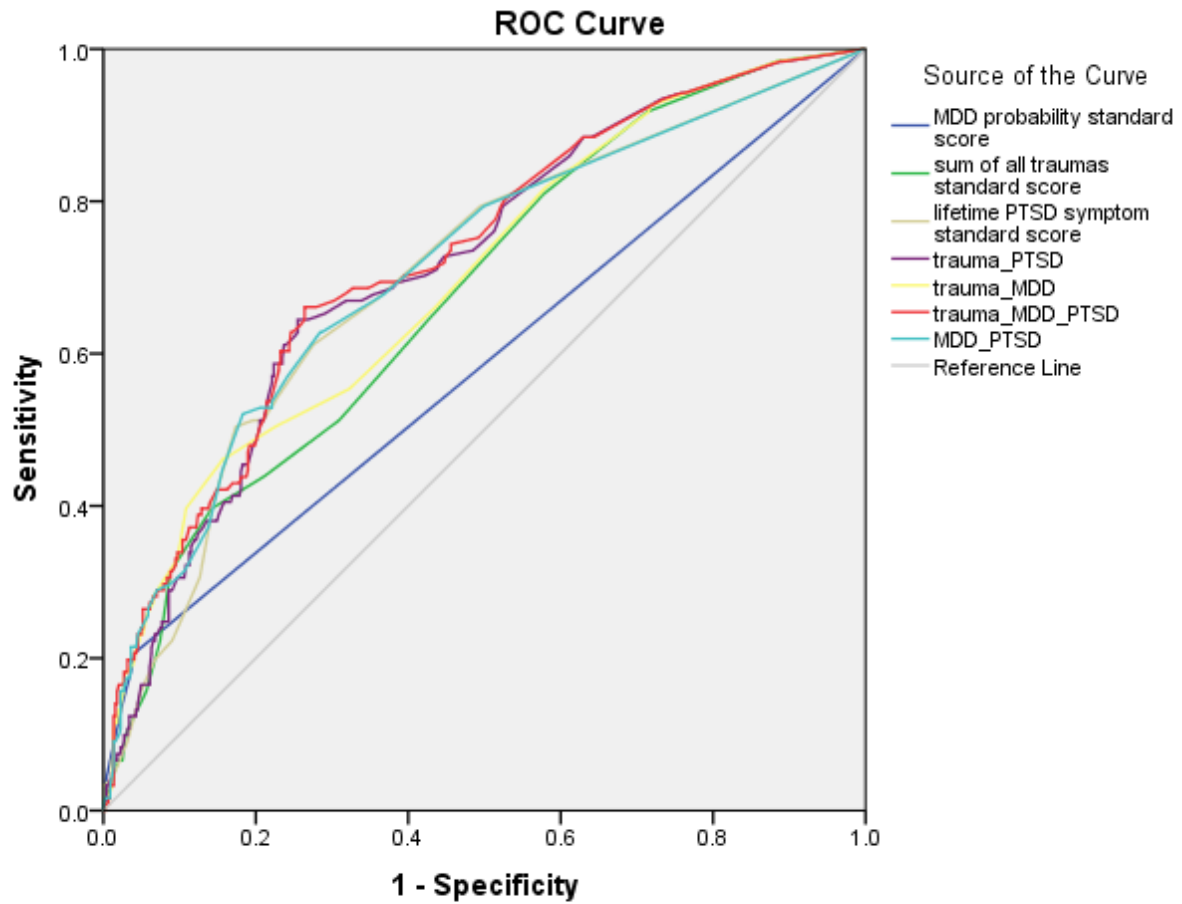
Figure 10. Mean of sociodemographic risk factors within each category of the dependent variable.

*Hypothesis 1c: Symptoms of PTSD and the number of traumas endorsed by women will be better predictors of postpartum depression than symptoms of depression in pregnancy.*

As explained in Chapter 3, I used ROC modeling to explore prediction of postpartum depression based on standard scores of continuous counts of trauma exposures, depression probability scores, and posttraumatic stress symptom count, measured in early pregnancy. ROC modeling provides us with a visual curve, and a calculated ‘area under the curve’ (AUC) statistic; the AUC ranges from 0.5 and 1.0, with larger values indicative of better fit.

Assessment of the ROC curves and AUC statistics (Figure 11 and Table 13) demonstrate that Hypothesis 1c is supported; prenatal depressive probability score is the *least* adequate predictor of postpartum depression. (Reminder: due to skip patterns, symptom counts for depression are not available; however probability scores are). Trauma exposure history alone (AUC = .679) is better than depressive probability score level (AUC = .584). Trauma history and posttraumatic stress symptom count are much better predictors, and adding depressive probability score improves upon that only very marginally (AUC = .726, as compared to AUC = .715).



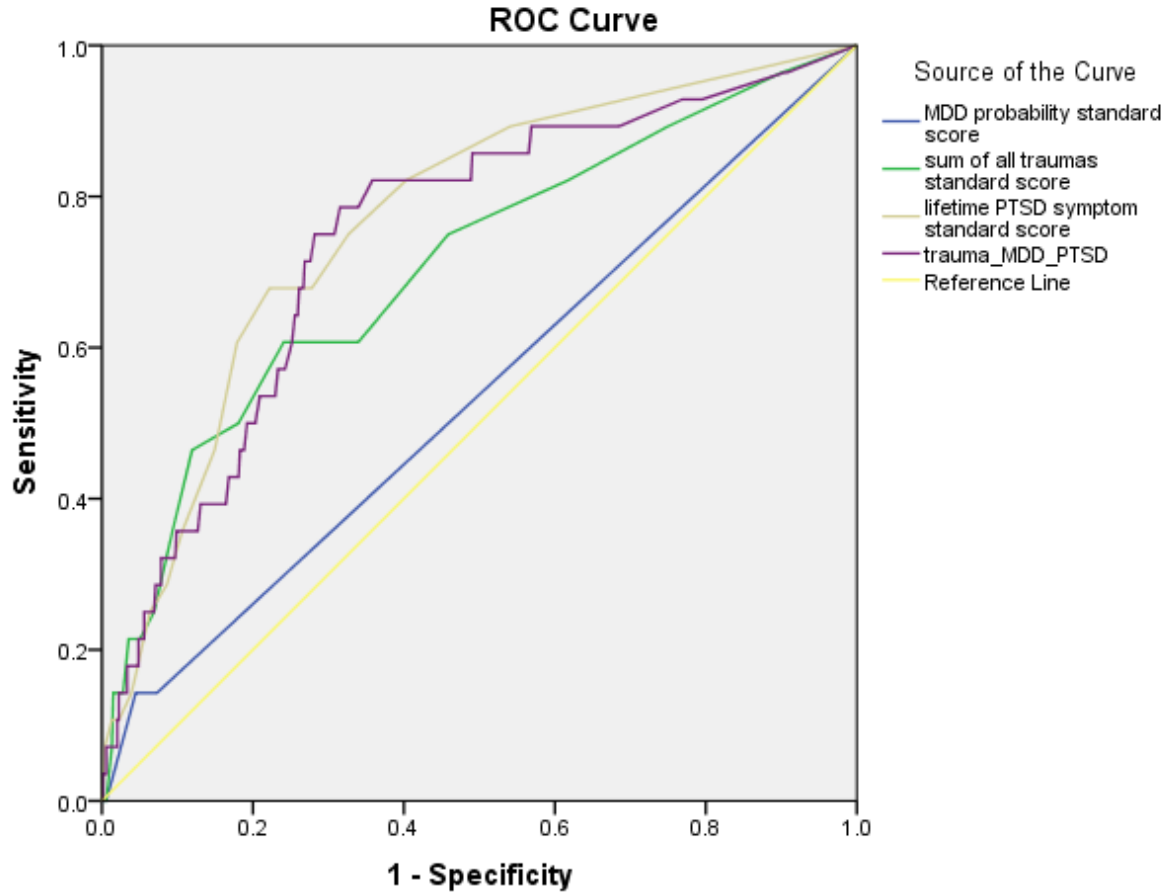


*Figure 11.* Receiver-operator characteristic growth curve model predicting postpartum depression based on standard scores of continuous counts of trauma exposures, depression probability scores, and posttraumatic stress symptom count, measured in early pregnancy.

Table 13  
*Area Under the Curve (AUC) Results for Prediction of Postpartum Depression*

Variable	AUC	SE	Asymptotic <i>p</i>	Asymptotic 95% CI	
				Lower	Upper
MDD probability sum (standard score)	.584	.031	.005	.522	.645
sum of all endorsed trauma exposures (standard score)	.679	.027	<.001	.626	.731
Lifetime PTSD symptom count (standard score)	.703	.027	<.001	.649	.756
trauma score X PTSD score	.715	.026	<.001	.665	.766
trauma score X MDD	.698	.027	<.001	.646	.751
trauma score X PTSD X MDD	.726	.026	<.001	.675	.777
MDD X PTSD	.711	.027	<.001	.657	.765

I also used ROC modeling to predict current PTSD diagnosis at six weeks postpartum. Not surprisingly, lifetime posttraumatic stress symptom load by itself is the best predictor of postnatal current PTSD diagnosis (AUC = .768), as compared to cumulative trauma exposure alone (AUC = .709) or the combination of trauma, PTSD, and depressive probability scores (AIC = .747). Depressive probability score was not a statistically significant predictor. (Figure 12 and Table 14).



*Figure 12.* Receiver-operator characteristic growth curve model predicting postpartum PTSD based on standard scores of continuous counts of trauma exposures, depression probability scores, and posttraumatic stress symptom count, measured in early pregnancy.

Table 14

*Area Under the Curve (AUC) Results for Prediction of Postpartum PTSD*

Variable	AUC	SE	Asymptotic p	Asymptotic 95% CI	
				Lower	Upper
MDD probability sum (standard score)	.537	.059	.513	.421	.652
sum of all endorsed trauma exposures (standard score)	.709	.055	<.001	.601	.816
Lifetime PTSD symptom count (standard score)	.768	.045	<.001	.680	.856
trauma score X PTSD X MDD	.747	.048	<.001	.652	.841

**Aim II Analyses**

**Aim II: To explore the implications of DSM-5 (APA, 2013) changes in qualifying trauma criteria in relation to perinatal mental health.**

*Hypothesis IIa: Constraining the trauma criteria per DSM-5 guidelines will reduce the rate of PTSD prevalence in the STACY sample, and will thereby reduce the rate of comorbidity between PTSD and MDD.*

Constraining the trauma criteria in line with *DMS-5* (APA, 2013) criteria results in a dramatic reduction in the rate of PTSD in the sample, from 319/1581 women (20.2%) to 162/1581 (10.2%). This also reduces the rate of comorbidity of PTSD and MDD from 50% to 33%, and the rate of comorbidity of PTSD and postpartum depression from 51.2% to 29.8%. Table 15 shows specifically which traumas were/were not removed from consideration as “allowable” index traumas per DSM-5 (2013) PTSD diagnostic formulation.

Table 15

*Index Trauma Exposures Endorsed by STACY Participants in Relationship to DSM-5 criteria for PTSD Diagnosis*

Index Trauma	<i>n</i>	Allowable per DSM-5 criteria?		Lifetime PTSD Symptoms	
		<i>yes</i>	<i>no</i>	<i>M</i>	<i>SD</i>
Child sexual abuse/penetration	44	X		8.4	5.1
Emotional Abuse/Neglect	81		X	7.9	4.4
Adult sexual abuse/penetration	30	X		7.2	5.0
Adult sexual abuse/contact	21	X		7.2	5.5
Child physical abuse	43	X		7.1	5.2
Adult physical abuse	52	X		6.8	4.9
Reproductive Trauma	124		X	6.7	4.3
Child sexual abuse/contact	90	X		6.6	4.7
Physical neglect	5		X	6.6	5.6
Had serious illness	53		X	6.4	4.6
Serious money problems	87		X	6.0	4.3
Was fostered/adopted	23		X	5.9	4.3
Was jailed	19		X	5.7	4.1
Was separated/divorced	29		X	5.6	4.7
Other trauma (unspecified)	82		X	5.5	4.2
Sexual harassment	39		X	4.9	4.4
Was in a disaster	21	X		4.9	3.6
Saw robbery/attack	36	X		4.8	4.4
Sudden death of loved one	309	X		4.7	4.1
Family member	122		X	4.7	4.2

*(Table 15 continues)*

jailed					
Had serious accident	101	X		4.6	4.2
Was robbed/attacked	68	X		4.5	4.3
Caregiver for seriously ill person	107		X	4.4	4.0
Painful medical/ritual procedure	15	X		4.4	3.2
Witnessed domestic violence	148		X	4.2	4.0
Saw serious accident	75	X		4.0	3.7
Death of loved one (not sudden)	321		X	3.7	3.8
Parents separated/divorced when child	177		X	3.7	3.9
Exposure to war	4	X		2.5	3.0

*Hypothesis IIb: Those individuals who no longer meet PTSD diagnostic criteria per DMS-5 will have similar levels of postpartum depression.*

A one-way between subjects analysis of variance (ANOVA) was conducted in order to compare postpartum depressive symptom scores among those who had never met the PTSD diagnosis, those who met diagnosis per *DSM-IV* (APA, 1994), and those who met PTSD diagnosis via *DSM-5* (APA, 2013). There was a significant effect of diagnostic status on postpartum depressive symptoms [ $F(2, 561) = 30.22, p < .001$ ]. Post hoc comparisons using the Dunnett T3 test indicated that the mean score for postpartum depressive symptoms for those who had never met the diagnosis ( $M = 61.06, SD = 18.29$ ) was significantly different ( $p < .001$ ) than the mean score for those who met *DSM-IV* PTSD diagnosis ( $M = 71.06, SD = 20.40$ ), and also significantly different ( $p < .001$ ) than the mean score for those who met *DSM-5* diagnosis ( $M = 77.83, SD = 20.39$ ). However, there was *no* statistically significant difference in postpartum

depressive symptom scores between those who met *DSM-IV* diagnosis, and those who met *DSM-5* diagnosis ( $p = .12$ ).

*Hypothesis IIc: Those individuals who no longer meet PTSD diagnostic criteria per DMS-5 will have similar levels of bonding impairment to those individuals who do meet DSM-5 criteria.*

I also conducted a one-way between subjects analysis of variance (ANOVA) in order to compare bonding impairment scores among those who had never met the PTSD diagnosis, those who met diagnosis per *DSM-IV* (APA, 1994), and those who met PTSD diagnosis via *DSM-5* (APA, 2013). There was a significant effect of diagnostic status on symptoms of postpartum bonding impairment [ $F(2, 561) = 7.70, p = .001$ ]. Post hoc comparisons using the Dunnett T3 test indicated that the mean score for symptoms of bonding impairment for those who had never met the diagnosis ( $M = 16.64, SD = 7.54$ ) was significantly different ( $p = .01$ ) than the mean score for those who met *DSM-IV* PTSD diagnosis ( $M = 19.55, SD = 8.92$ ), and also significantly different ( $p = .01$ ) than the mean score for those who met *DSM-5* diagnosis ( $M = 19.47, SD = 7.54$ ). However, there was *no* statistically significant difference ( $p = 1.0$ ) in symptoms of bonding impairment between those who met *DSM-IV* diagnosis, and those who met *DSM-5* diagnosis.

## Chapter 5 Discussion

In this chapter the implications of the findings of this secondary analysis will be discussed, as well as potential limitations of the study and suggestions for directions for future research as well as implications for the field of social work. I have endeavored to include as many elements of the Seng conceptual model (2002) in these analyses as possible within the constraints of a secondary analysis. I have considered the effects of trauma leading to PTSD and/or MDD; have taken into consideration non-modifiable and co-occurring risk factors including cumulative sociodemographic risk and other co-morbid conditions, such as interpersonal sensitivity, somatization, dissociation, and generalized anxiety. I have considered how PTSD and MDD contribute to adverse outcomes, including not only postpartum depression but also impairment in mother-baby bonding. The following discussion of study aims and hypotheses will highlight specific ways in which there appears to be interplay between trauma, PTSD, and MDD, and will also consider how this interplay is affected by changes in the diagnostic formulation of PTSD.

*Aim I: To determine the extent to which it is warranted to integrate perinatal mental health care to combine attention to depression, trauma, and posttraumatic stress.* Many women experience all manner of trauma and stress and yet do not develop PTSD or MDD or other psychiatric challenges. Their resiliency may be born of innate factors or may stem from the contextual factors of their lives; that in the face of trauma and stress there was help and nurturing available at critical junctures. Yet for those women who were not resilient, it is important to understand the extent to which trauma-informed help is needed. In these data the experience of trauma and sociodemographic stress appear to be “equal opportunity” experiences which are spread across the groups of interest, even in the *No-Trauma* group. This is similar to the Kim



study (et al., 2013) which also found moderate to high levels of psychosocial stress in both the sample as a whole and within the diagnostic subgroups considered; although they did find a strong prediction of PTSD based on indicators of unstable housing, which was not assessed in the STACY study. The Cook et al 2004 (et al., 2004) study also did not find significant differences based on sociodemographic characteristics for women in their study with/without PTSD. There may be unmeasured variables in both the STACY and the Cook study, such as personality differences, family or social support, or quality of partner relationships, which mediate the association of sociodemographic disadvantage and psychiatric morbidity and differentiate between those women who will be resilient in the face of trauma. Yet understanding who is *not* resilient to traumas and stressors is essential from a clinical perspective, and increases opportunity for identification and treatment engagement. As a whole these analyses show that integrating mental health care to combine attention to depression, trauma, and posttraumatic stress is warranted.

*Hypothesis Ia: Some women who meet diagnosis for major depression will meet the criterion for index trauma exposure (“Trauma-Exposed Perinatal Depression” [T-EPD]), and will be subthreshold for PTSD (will have at least three symptoms of PTSD but not meet the full diagnostic criteria).* As mentioned in the previous chapter, this hypothesis was supported; the 67 women in the *T-EPD* group did evidence subthreshold PTSD. However, the 30 women in the *MDD-Only* group also evidenced subthreshold PTSD; in fact there was no statistically significant difference in the rate of symptom endorsement, despite the fact that the *MDD-Only* individuals did not endorse an index trauma. There is a gradient response evident overall; with the *No-Trauma* controls experiencing an average of 1.5 symptoms, the *Trauma-Only* group experiencing an average of 2.8 symptoms, the depressed-only groups experiencing an average of between 4.4

(*T-EPD*) and 4.9 symptoms (*MDD-Only*), the *PTSD-Only* group experiencing 10.6, and the *Comorbid* group experiencing 11.4. This finding is consistent with a study of the latent structure of PTSD which posits that PTSD is a dimensional disorder (Ruscio, Ruscio, & Keane, 2002). Overall, it may be that a distinction between depression which is comorbid with PTSD and that which is not many depend more on whether a woman has an *enduring reaction* to trauma (subthreshold PTSD), rather than whether she endorsed any specific trauma exposure as an index trauma. That enduring reaction can change over time, as well - it may be that subthreshold PTSD symptoms at any point in time represent a delayed expression of reaction to trauma, and, as a meta-analytic review (Smid, Mooren, van der Mast, Gersons, & Kleber, 2009) suggests, may eventually result in full diagnosis. Further, emerging research shows that there may be a specificity of risk for “acquired capacity for suicide” based on specific PTSD symptom clusters (numbing and anxious arousal) (Zuromski, Davis, Witte, Weathers, & Blevins, 2014). Thus, identifying women with depression and PTSD symptomatology may help us to assess risk for delayed onset of PTSD diagnosis and acquired capacity for suicide, as well.

*Hypothesis 1b: Antenatally depressed women with index trauma exposure (T-EPD) will differ from antenatally depressed women without index trauma exposure by type of traumas endorsed overall (index or not), sociodemographic risk factors, and other comorbid conditions.* Differences did emerge across the diagnostic categories related to the types of trauma exposures endorsed overall (regardless of whether they were ‘index’ traumas – meaning they were “worst” or “second worst”).

Women in the *Trauma-Only* group were more likely than the *No-Trauma* reference category to endorse loss/separation traumas, yet interestingly loss/separation was not a statistically significant predictor of membership in any of the other categories. This may be

reflective of the ubiquity of loss and separation to the human experience and our ability to adapt, except in cases of complicated bereavement (Shear et al, 2011). Or it may reflect an effect of the presence of multiple trauma exposures in the diagnostic categories, which may have attenuated the effect of such losses – which in the context of abuse trauma may have even been welcome separations. Other perinatal studies reviewed, which also simultaneously collected data on trauma, PTSD, and MDD, did not report outcomes specifically related to loss/separation trauma, except in the case of the Cook (et al., 2004) study, which reported that multiple lifetime traumas and prolonged separation from mother in childhood were risk factors associated with PTSD in pregnancy.

Although the role of loss trauma in the form of sudden death of a loved one has been specifically examined for its contribution to the development of PTSD in samples of the general population (e.g., Breslau, et al., 1998), to my knowledge this is the first pregnancy study to specifically look at the role of loss/separations in this manner. There is a separate body of literature finding support for the development of PTSD specific to previous pregnancy loss (e.g., Turton, Hughes, Evans, & Fainman, 2001). However, it was an exclusion criterion for the STACY study that participants had not had previously carried a pregnancy past 20 weeks; had multiparous women been included it is reasonable to assume that women who had experienced a stillbirth or loss of a child might be experiencing complicated bereavement. A criticism of the PTSD diagnosis that has led to changes in the *DSM-5* (2013) was that death of a loved one was over-diagnosing individuals; yet it is interesting that loss/separation as a whole did not predict membership to any of the diagnostic categories.

Women in all categories except for those with *MDD-Only* were more likely to endorse abuse traumas than those in the *No-Trauma* reference group. This finding is in line with studies

which have considered the association of violence-related trauma and PTSD during pregnancy (e.g., Harris-Britt et al., 2004) but differs from those which found an association of child sexual abuse and antenatal depression (e.g., Rich-Edwards et al., 2011). However, the Rich-Edwards et al. (2011) study did not assess or control for PTSD; it is therefore reasonable to assume that some of the participants may also have experienced posttraumatic stress as well. The finding of association of abuse traumas in the *Comorbid* category is in line with other studies which simultaneously assessed for PTSD and depression. For instance, the Kim et al (2013) study found increased odds for depression among those women who either met PTSD diagnosis or had subthreshold PTSD, and who had experienced partner or non-partner violence and physical or sexual abuse.

Women in all categories except for those with *MDD-Only* were also more likely to endorse reproductive traumas than those in the *No-Trauma* reference group. As mentioned, although there have been investigations into the effect of stillbirth on subsequent pregnancies (Turton et al., 2001), the literature is much more limited related to the effect of previous elective abortion (EAB) or spontaneous abortion (SAB, “miscarriage”) on current pregnancy. A study which looked at PTSD among women with previous pregnancy complications (Forray, Mayes, Magriples, Epperson, 2009), of which 75% were miscarriages, found an increased risk for antenatal PTSD. Previous STACY analyses related to reproductive trauma (Hamama, Rauch, Sperlich, Defever, & Seng, 2010) found that a history of sexual trauma predicted appraisal of either EAB or SAB as traumatic, and that those with reproductive trauma as an index trauma were at a lowered risk for PTSD comparatively to those whose index trauma was due to some other trauma. What this suggests is that reproductive trauma may co-occur with abusive trauma

in conferring risk for PTSD and PTSD comorbid with depression (but not among those with *MDD-Only*).

Odds ratios for traumas considered “other,” meaning that they were not either loss, abuse, or reproductive in nature, were increased for all diagnostic categories as compared to the *No-Trauma* comparison group, and were highest for the *Comorbid* group. It is difficult to make any direct comparisons to the literature for this finding, owing to the fact that the other studies which simultaneously looked at trauma, PTSD and MDD were either focused directly on a specific types of traumas (such as interpersonal partner violence, as in the Rodriguez et al, 2008 study) or did not report findings for a comprehensive list of potential traumatic exposures in similar fashion to the STACY study.

While the finding that there was increased risk of having experienced trauma of an abusive and reproductive nature for individuals in the diagnostic categories is perhaps easy to predict, the odds ratios suggesting that there is less sociodemographic risk in the diagnostic categories is a completely non-intuitive finding. It appears that the explanation for this might center on the fact that sociodemographic disadvantage is spread across the entire dataset, and that in fact the members of the *MDD-Only* category have the highest level of sociodemographic disadvantage. I interpret this to mean that for those with depression but no index trauma the depression may be more related to sociodemographic/life event stressors rather than traumatic insult. However, because depression is actually spread across three categories, and because the *No-Trauma* reference category also has high sociodemographic risk/stress, the direction of the association when all categories and all 55 comparisons are conducted simultaneously remains a bit of a mystery. Similarly to other studies of the effects of weathering (Collins, David, Handler, Wall, & Andes, 2004; Mustillo et al., 2004), previous STACY analyses (Seng et al, 2011) found

an increased risk for prematurity and low birth weight among African American women; however, PTSD in pregnancy associated specifically with child maltreatment was found to be a stronger predictor of shorter gestation and a nearly equal predictor of birthweight. In this study, the finding of lowered sociodemographic risk factors among the diagnostic categories, although perhaps not intuitive in nature, is similar to the Kim et al. (2013) study, and the Cook et al. (2004) study, as previously mentioned, and overall suggests that individual factors of resiliency and enduring effects of trauma confer the greatest risk, above and beyond sociodemographic indicators.

In terms of other comorbidities considered, although all those entered into the model were significantly associated at the bivariate level with the dependent variable, only somatization and generalized anxiety emerged as statistically significant in the multinomial logit model. Members of all diagnostic categories (but not the *Trauma-Only* category) were less likely to have symptoms of somatization than the reference category when all categories are considered simultaneously. Although a connection has previously been established between PTSD diagnosis and somatization in young adults in southeast Michigan (Andreski, Chilcoat, & Breslau, 1998), I am aware of no other studies which directly assess the risk for symptoms of somatization in childbearing women, and no studies which assess this risk related to the comorbidity of PTSD and MDD. The finding of lowered risk for symptoms of somatization is therefore a novel finding. This may seem odd considering that factor analysis of depression has largely favored a two-factor solution including non-somatic (depressed mood, feeling worthless, suicide ideation) and somatic (problems with sleep, fatigue, appetite) (Tsai, Elhai, Pietrzak, Hoff, & Harpaz-Rotem, 2014); yet there is surprisingly little overlap between the somatic symptoms of depression and the symptoms queried by the SCL-90-R (Derogatis, 1977/2000), which include

stomach pain, back pain, headaches, and chest pain, for example. Similarly to sociodemographic risk, these somatic symptoms appear to be spread among the diagnostic categories, including in the *No-Trauma* reference category, and may indicate that symptoms exclusively physical in nature, although associated with the diagnostic categories at the bivariate level, are nonetheless less prominent as a risk factor when all comparisons are considered simultaneously. From a clinical perspective, then, it may be more important to focus on the symptoms of physical problems that are specifically associated with depression and/or PTSD, including sleep problems, fatigue, changes in appetite, and irritability.

A difference emerged in relation to generalized anxiety, with those in the depression groups (*MDD-Only*, *T-EPD*, and *Comorbid*) at higher risk, however there was no statistically significant relationship between those with *PTSD-Only* and generalized anxiety. Many studies of perinatal mental health to date have aggregated PTSD with generalized anxiety and other anxiety disorders (see Nast, Bolten, Meinschmidt, & Hellhammer, 2013 review); which makes it difficult to parse the relative contributions of PTSD and generalized anxiety. However, the finding of increased risk for comorbidity with generalized anxiety in all of the diagnostic categories excepting for those with *PTSD-Only* is in line with findings of a large, longitudinal case-control cohort study ( $n = 2402$ ) of individuals (both female and male) in the Netherlands which found evidence for a shared vulnerability for comorbid PTSD with both anxiety and depressive disorders (Spinhoven, Penninx, van Hemert, Rooij, & Elzinga, 2014). Their multivariable models suggest that being female and having a history of child sexual and physical abuse are factors underlying the comorbidity and conclude that there should be routine assessment for PTSD among persons identified with anxiety and depressive disorders.

The finding that those with *PTSD-Only*, in the absence of MDD, are not at increased risk for generalized anxiety also affirms the *DSM-5* (2013) removal of PTSD from being an anxiety-related disorder to placement instead as a trauma and stressor-related disorder. Overall, clearly anxiety should remain a concern for clinicians treating women who present with PTSD comorbid with depression during pregnancy.

Overall, the analyses for Hypothesis 1b allowed us to see that there are differences in the kinds of traumatic exposures and other comorbid disorders experienced by women in the depressed-only groups (either *MDD-Only* or *T-EPD*) as compared to the *No-Trauma* reference group and controlling for the other diagnostic categories. It is also clear that these differences may be accounted for by the level of subthreshold PTSD symptomatology, which is an important clinical finding, when we consider that higher number of subthreshold PTSD symptoms have been found to be associated with greater impairment, comorbidity and suicide ideation in a large sample of both men and women ( $n = 9,358$ ; Marshall et al., 2001). Similarly, Kim et al. (2013) found that pregnant women with subthreshold PTSD were clinically similar to those with full PTSD diagnosis.

As previously mentioned a recent publication of the National Center for PTSD (2014) which reviewed the literature on subthreshold PTSD diagnosis suggests that the designation has clinical relevance, and should continue to be a focus of research in general. The finding of a gradient pattern of PTSD symptoms irrespective of diagnosis in these analyses corroborate this, and suggest that it is indeed warranted to integrate perinatal mental health care to combine attention to depression, trauma, and posttraumatic stress. Because there is a dearth of studies which simultaneously assess for MDD *and* PTSD, we do not yet understand the extent to which the comorbidity may be driving the countless adverse outcomes evident for depression in



childbearing women. These analyses indicate that, in accordance with the proposed changes to the Seng model (2002), that exposure to trauma of an abusive nature predicts not only PTSD but also depression which is comorbid with subthreshold PTSD.

*Hypothesis 1c: Symptoms of PTSD and the number of traumas endorsed by women will be better predictors of postpartum depression than symptoms of depression in pregnancy.* Given that there is a gradient pattern of PTSD symptoms, it is important to look at what might predict postpartum depression. As shown in the preceding chapter, ROC analyses demonstrate that either trauma or PTSD predict postpartum depression better than antenatal MDD alone. The best prediction is the combination of trauma, PTSD, and MDD. This underscores how the comorbidity interplay ante- to post-partum are critically important. Depression during pregnancy was not, however, a statistically significant predictor of new-onset PTSD postpartum.

The finding that PTSD symptomatology in pregnancy and trauma were better predictors of postpartum depression is an important one, and suggests that we may need to modify the way in which we think about risk for postpartum psychiatric illness. The finding is similar to an Australian study, also of first-time parents, which found that maternal anxiety disorder seems to confer greater risk for postpartum mood disorder (either depression or anxiety) than does a history of depressive disorder (Matthey, Barnett, Howie, & Kavanaugh, 2003). It is also similar to the Records & Rice (2009) study which found that women with a history of physical and sexual abuse were between 3.6 to 8.4 times more likely to have postpartum depression (PTSD was not assessed in this particular study), and the Leeners et al (2006) study which also found increased risk for postpartum depression among women who had a history of childhood sexual abuse. These results differ from studies such as the Leigh & Milgrom 2008 study, which found that antenatal depression is a risk factor for postpartum depression. However, it is important to

note that PTSD was not assessed in this study, and so the role that comorbidity plays cannot be assessed. A study which did consider the comorbidity between history of trauma, PTSD symptoms and depression in pregnancy, found an association with postpartum stress symptoms in a large sample of childbearing women in Israel ( $n = 1071$ ) (Lev-Wiesel, Chen, Daphna-Tekoah, & Hod, 2009).

Rates of psychiatric symptomatology in pregnancy are higher than for general populations, yet we do not have a clear empirical understanding of why this may be. Historical writings from the psychoanalytic literature suggest that pregnancy is a sensitive time for women, and that the psychosexual processes of gestation, birth, and early parenting represent a “special” epoch in a woman’s life, when past trauma may be “triggered” (Bibring, 1959; Deutsch, 1945; Kestenberg, 1979; Pines, 1972). Pregnancy has been referred to as a “critical phase” (Benedek, 1970), a “liminal” phase in a rite of passage (Balin, 1988); and a time of “permeability,” whereby pregnancy “churns up primitive anxieties and revitalizes emotions that had been laid to rest” (Rafael-Leff, 1995/2001, p. 28). Such anxieties lead to mother/baby dyadic disruptions, that Fraiberg, Adelson, & Shapiro (1975) termed “ghosts in the nursery.” The extent to which a new mother was adequately attached to her own mother and the nature of her maternal representations of attachment in general may have implications for her attachment to her infant (Fonagy, Steele, & Steele, 1991; Huth-Bocks, Levendosky, Bogat, & von Eye, 2004; Stern, 1991).

An alternative explanation for the development of psychopathology in women centers on the increased risk for exposure to the “ghosts” in the first place. Because women are at such high risk for abusive traumas, we must question whether we are “medicalizing women’s misery” (Ussher, 2010) by persistently focusing on the psychopathology rather than the root causes of the misery. The theory of *response styles*, which describes the propensity toward rumination among

women (Nolen-Hoeksema & Morrow, 1991; Nolen-Hoeksema, Wisco, & Lyumomirsky, 2008), suggests that rumination has its roots in the ‘gender intensification’ that occurs at puberty, and which is characterized by societal expectations and restrictions to social roles women are allowed to play (Nolen-Hoeksema and Girgus, 1994).

More work is needed to examine this “sensitive period” of pregnancy with empirical data, and to expose the underlying reasons for the development of women’s misery. Yet these analyses do seem to suggest that pregnancy should be an important focus of research and clinical attention, especially for those women living with the challenges of PTSD and/or depression.

***Aim II: To explore the implications of DSM-5 (APA, 2013) changes in qualifying trauma criteria in relation to perinatal mental health.*** Given that there appears to be interplay between trauma, PTSD and depression in terms of predicting who will be at risk for psychiatric challenge in the postpartum period, it is also important to consider how the changes to the PTSD diagnosis per publication of the DSM-5 (2013) will impact these findings.

*Hypothesis IIa: Constraining the trauma criteria per DSM-5 guidelines will reduce the rate of PTSD prevalence in the STACY sample, and will thereby reduce the rate of comorbidity between PTSD and MDD.* In chapter 4, Table 15 showed that many of the STACY index traumas endorsed by participants would no longer be considered as “allowable” exposures for the PTSD diagnostic formulation. Note that many of what we might consider to be threats to the attachment system or abuses considered *early relational trauma* are removed from consideration as “allowable” traumas. To be specific, in the logit analyses for Aim I I used the variable *abuse trauma* to reflect interpersonal trauma of an abusive nature, including childhood physical abuse and sexual abuse contact/penetration, adult sexual abuse contact/penetration, and emotional abuse/neglect or physical neglect. Per *DSM-5* (2013), emotional abuse/neglect and physical

neglect would not be considered as allowable index traumas. For the variable *loss/separation trauma* used in the logit analyses, which included death of a loved one, sudden death of a loved one, separation/divorce of parents, separation/divorce of participant, and being fostered/adopted, only sudden death of a loved one would remain as “allowable.” We must consider the effect of removing such traumas from consideration, particularly those traumas which represent early threats to the attachment system. The framers of the new diagnostic formulation, the ‘working group’ for *DSM-5*, were mindful of “complex PTSD” (i.e., Cloitre et al, 2009; Herman, 1992) in the inclusion of a new *dissociative subtype* for PTSD. Given that it is commonly understood that it is the early relational traumas *in particular* which lead to the development of complex PTSD, it is quite ironic that the index trauma criterion has been constrained to exclude many of the traumas that may in fact be associated with the dissociative subtype. It is also important to note that in these data having your parents separate or divorce as a child ranks highly among the most endorsed index traumas, as does loss of loved one to death (either sudden or not), and emotional abuse/neglect, which are also traumas which would no longer be considered as “allowable” per *DSM-5*.

The variable *reproductive loss* would also be removed from consideration, as would other variables which affect women in large numbers, including sexual harassment, witnessing domestic violence, and being a caregiver for someone who is severely ill. PTSD is a relatively new diagnosis, and recognition of women’s experience of PTSD is even newer. Based on that, it seems reasonable to assume that we do not yet fully understand the development of PTSD in women, and certainly not in the perinatal period. The removal of these types of traumas from consideration begs the question of whether the *DSM-5* has unwittingly introduced a gender bias in the PTSD formulation. I have not found a feminist response to the *DSM-5* in the literature

specifically related to the PTSD diagnostic formulation. However, a special issue of *Feminism & Psychology* has already appeared which has articles critiquing the inclusion of premenstrual dysphoric disorder and the undue influence of drug industry ties to *DSM-5* development panel members (Cosgrove & Wheeler, 2013), critiquing the changes to the way sexual dysfunction is characterized (Duschinsky & Chachamu, 2013) and challenging the insistence on clinician rather than client perspective related to the lived experience of mental illness (Hornstein, 2013). More research is needed to understand the clinical impact of the changes to the PTSD trauma criterion for women in general, and for women in the perinatal period specifically.

This returns us also to the trouble with defining *trauma* and *stress*; certainly, clinicians will agree that all of the potentially traumatic situations about which STACY participants were queried can be experienced as stressful – but are they traumatic? Recall that the traumas we are discussing which were dismissed from the diagnosis per *DSM-5* (2013) criteria were identified *by the individual participant* as the “worst” traumas that had happened to them. Further, they went on to meet all remaining PTSD criteria in relation to these traumas. It is therefore important to consider what will happen to individuals who are symptomatic but who might go undetected, either because their appraisal of the trauma they suffered is not “allowable” or because they are not asked about the trauma they experienced in the first place.

*Hypothesis IIb: Those individuals who no longer meet PTSD diagnostic criteria per DMS-5 will have similar levels of postpartum depression.* These longitudinal analyses demonstrate that there is *no statistically significant difference* in prediction of postpartum depression between women who met *DSM-IV* (1994) but no longer meet diagnosis by *DSM-5* (2013) criteria and women who continue to meet diagnosis via *DSM-5*. This is also an important finding because it means that strict application of the *DSM-5* criteria will result in a

diminishment of our ability to predict postpartum depression, due to the fact that the women who evidenced PTSD per *DSM-IV* may no longer be brought to clinical attention. Given the poor outcomes for women and babies and children related to maternal (and paternal) postpartum depression, we must question whether we should constrain our ability to both detect and intervene on behalf of those at risk. These data suggest that the constraining of the trauma criterion per *DSM-5* will limit our ability to detect risk, and will exclude many individuals whose traumatic exposure was a threat to their early attachment system, and who may in fact have complicated and enduring symptoms.

*Hypothesis IIc: Those individuals who no longer meet PTSD diagnostic criteria per DMS-5 will have similar levels of bonding impairment to those individuals who do meet DSM-5 criteria.*

Identifying intergenerational risk of abuse and psychiatric vulnerability is paramount. Similarly to the previous analyses for postpartum depression, these analyses found that there was *no statistically significant difference* in prediction of bonding impairment scores whether a woman met PTSD diagnosis via *DSM-IV* (1994) or via *DSM-5* (2013). While bonding impairment via self-report measurement is an inadequate substitute for observational measures of dyadic functioning, this analysis should serve nonetheless as a red flag. Early impairment in a mother's ability to feel bonded to her baby is likely an early warning signal for future risk. Because those women who were dismissed from the diagnosis per *DSM-5* will potentially no longer be the focus of clinical concern, we will not be able to see this warning flag. Similarly to the prediction of postpartum depression, it is an important finding of these analyses that constraining the trauma criterion of the PTSD diagnosis will also constrain our ability to predict, detect, and treat women who experience bonding impairment with their infant.

## Limitations

Several limitations inherent to secondary analysis have already been identified in previous chapters. Additional limitations emerged during the analytic process. These included the limited ability to represent all features of the Seng conceptual model for cases of interest because many of those with subthreshold PTSD were not followed longitudinally. The longitudinal analyses which were undertaken were limited in scope to bivariate prediction of postpartum depression and bonding impairment as a result. I was also unable to consider stress hormone results for this reason, or other biological factors which might have been gleaned from chart data. Another limitation concerned the way in which depression was measured in the STACY data. Although a diagnostic gold-standard measure of MDD was utilized, no truly dimensional measure of depression was employed. Although probability scores were treated as dimensional measures for longitudinal bivariate analyses, I was unable to enter the probability scores into the multinomial logit model due to multicollinearity: due to skip patterns in the way the questions were asked the probability scores mapped on to the diagnosis so precisely that it created separation in the data. A truly dimensional measure would have allowed us to see the other side of the comorbidity coin – the symptom load of depression in the PTSD groups.

Another limitation of the analyses was the lack of multiparity in the data. Given the burgeoning literature related to trauma *as a result of birth itself* (e.g., Beck & Watson, 2008; Kitzinger & Kitzinger, 2007; Söderquist et al., 2004), and also due to the previously mentioned possibility of stillbirth or loss of a child, or having a child with severely compromised health, constraining the STACY dataset to primiparous mothers limits our ability to directly compare these results to a naturalistic clinic setting. A final, important limitation of these analyses is a lack of elements that might have gotten at *stress* rather than *trauma* and lent better prediction for

depression. These elements might include stress-specific measurements, detailed family histories of conferred risk for depression, measures of rumination, measures of housing instability and territorial incivilities, social support, etc.

### **Future Research**

More studies are needed which simultaneously assess for trauma, life stressors, PTSD, & MDD and other comorbid disorders and which use dimensional as well as diagnostic measures. Ideally such studies would follow unselected samples forward longitudinally and would include multiparous as well as primiparous mothers. Also needed are studies which connect bonding impairment to observational assessment of dyadic attachment and measures of parental reflective functioning.

We must critically examine the narrowing of the trauma criterion for PTSD per *DSM-5* (2013), particularly for women in the sensitive perinatal period, and invite clinician as well as research feedback to these changes; if the *DSM-5* is indeed a “living document,” capable of evolution, we must hasten its growth through critique. More studies are also needed which do a better job of codifying trauma/stress and determining whether there are meaningful distinctions to be made related to the prediction of psychiatric challenge.

Overall, these analyses suggest that we need trauma-informed and indeed trauma-explicit interventions to address symptoms of PTSD that may be contributing to the development of postpartum depression and which may help to improve the lives of mothers, babies, and families. However, development of such interventions will fall short if there is not concomitant training of clinicians—maternity care workers and those who work with women in the postpartum period, including obstetricians, midwives, social workers, psychologists, infant mental health professionals, case workers, pediatricians, etc. We also need to build more bridges between the



fields of mental health and infant mental health by focusing on their intersection in the childbearing year.

### **Social Work Implications**

Often it seems that psychosocial researchers spend a certain amount of time “re-inventing the wheel.” In that vein, I do not think that the notion that trauma and PTSD is associated with depression will surprise many clinicians. It is often the case, especially for research that addresses the needs of women specifically, that the evidence base lags behind clinical knowledge. PTSD, as an example, did not appear as an operational diagnosis until 1980 with the publication of the *DSM-III* (American Psychiatric Association, 1980), and it has taken much longer than that to recognize the increased risk for PTSD for women. Given that both depression and PTSD are experienced by women at increased rates, have demonstrated devastating outcomes, and are refractory to treatment, it is necessary to continue to strive to increase our understanding of these conditions in order to discover relevant components for the tailoring of interventions. These analyses could potentially lead to a paradigm shift in case conceptualization and treatment for childbearing women who do not meet PTSD diagnostic criteria but for whom unresolved trauma exposure and subthreshold PTSD may be a potentiating factor in the development of postpartum depression. It is solidly aligned with the advances in trauma-informed care structures and could lay groundwork for applying trauma-specific treatment for MDD when unresolved trauma and PTSD is also endorsed by the woman. It introduces the possibility that we should be focusing specifically on abuse and reproductive traumas in the childbearing year.

The field of social work has embraced the biopsychosocial model (Engel, 1977) in the training of social work clinicians and in clinical environments. These analyses are consonant

with the biopsychosocial perspective in that they suggest that contextual factors, such as a person's appraisal of the traumas they have endured, the specific type of traumas and life stressors experienced - together with symptomatology that might cut across diagnostic formulations, are all important components to be considered as resources are garnered to treat the whole individual in her unique circumstance. Social work scholars can help to advance the field in perinatal mental health research by agitating for broader adoption of the biopsychosocial perspective in prenatal and postpartum care; a move which would increase our awareness of trauma exposure and psychiatric symptomatology across the board.

Although the social work profession does not appear to be driving the discussion in perinatal mental health, despite urgings (Abrams & Curran, 2007; Sheppard, 1994), it is equally evident that many social workers are actively engaged on the ground in the implementation of perinatal interventions. There also appears to be a natural alliance between social work and the infant mental health perspective; owing to the relationship between maternal mental health and bonding portrayed in these analyses, this alliance should be continue to be fostered. And while it is important to add evidence to our collective clinical knowledge about the provision of mental health care in general and in the childbearing year specifically, there is also a role for social workers' leadership to continue to bring attention to the structural inequalities and gendered disparities that undergird the development of compromised perinatal mental health; these analyses may lend fuel to that fire, as well.

## **Conclusion**

It is my hope that the results of these dissertation analyses will further our understanding of the comorbidity of trauma, posttraumatic stress (PTSD) and depression in childbearing women. I further hope that these understandings will help us to find areas for intervention at both

the individual and societal levels, and ultimately help to improve the lives of mothers, babies, and families.

## APPENDIX A: DSM DIAGNOSTIC CRITERIA FOR PTSD

### *DSM Diagnostic Criteria for PTSD*

<i>DSM-IV</i>	<i>DSM-5</i>
<p>A: The person has been exposed to a traumatic event in which both of the following were present:</p> <ol style="list-style-type: none"> <li>1) The person experienced, witnessed, or was confronted with an event or events that involved actual or threatened death or serious injury, or a threat to the physical integrity of self or others.</li> <li>2) The person's response involved intense fear helplessness, or horror.</li> </ol>	<p>A: Exposure to actual or threatened death, serious injury, or sexual violence in one or more of the following ways:</p> <ol style="list-style-type: none"> <li>1) Directly experiencing the event(s) as it occurred to others</li> <li>2) Witnessing, in person, the event(s) as it occurred to others.</li> <li>3) Learning that the event(s) occurred to a close family member or close friend. In cases of actual threatened death of a family member or friend, the event(s) must have been violent or accidental</li> <li>4) Experiencing repeated or extreme exposure to aversive details of the traumatic event (e.g., first responders collecting human remains, police officers repeatedly exposed to details of child abuse)</li> </ol> <p>Note: Criterion A4 does not apply to exposure through electronic media, television, movies, or pictures, unless this exposure is work related.</p>
<p>B: The traumatic event is persistently re-experienced in one (or more) of the following ways:</p> <ol style="list-style-type: none"> <li>1) Recurrent and intrusive distressing recollections of the event, including images, thoughts, or perceptions.</li> <li>2) Recurrent distressing dreams of the event.</li> </ol>	<p>B: The traumatic event is persistently re-experienced in the following way(s): (one required)</p> <ol style="list-style-type: none"> <li>1. Recurrent, involuntary, and intrusive memories. Note: Children older than six may express this symptom in repetitive play.</li> <li>2. Traumatic nightmares. Note: Children</li> </ol>

- 3) Acting or feeling as if the traumatic event were recurring (includes a sense of reliving the experience; illusions, hallucinations, and dissociative flashback episodes, including those that occur on awakening or when intoxicated). Note: In young children, trauma-specific reenactment may occur.
- 4) Intense psychological distress at exposure to internal or external cues that symbolize or resemble an aspect of the traumatic event.
- 5) Physiological reactivity on exposure to internal or external cues that symbolize or resemble an aspect of the traumatic event.

C: Persistent avoidance of stimuli associated with the trauma and numbing of general responsiveness (not present before the trauma), as indicated by three (or more) of the following:

- 1) Efforts to avoid thoughts, feelings, or conversations associated with the trauma
- 2) Efforts to avoid activities, places, or people that arouse recollections of the trauma
- 3) Inability to recall an important aspect of the trauma
- 4) Markedly diminished interest or participation in significant activities
- 5) Feeling of detachment or estrangement from others
- 6) Restricted range of affect (e.g., unable to have loving feelings)
- 7) Sense of a foreshortened future (e.g., does not expect to have a career, marriage, children, or a normal

may have frightening dreams without content related to the trauma(s).

3. Dissociative reactions (e.g., flashbacks) which may occur on a continuum from brief episodes to complete loss of consciousness. Note: Children may reenact the event in play.
4. Intense or prolonged distress after exposure to traumatic reminders.
5. Marked physiologic reactivity after exposure to trauma-related stimuli.

C: Persistent effortful avoidance of distressing trauma-related stimuli after the event: (one required)

1. Trauma-related thoughts or feelings.
2. Trauma-related external reminders (e.g., people, places, conversations, activities, objects, or situations).

lifespan)

D. Persistent symptoms of increased arousal (not present before the trauma), as indicated by two (or more) of the following:

- (1) Difficulty falling or staying asleep
- (2) Irritability or outbursts of anger
- (3) Difficulty concentrating
- (4) Hypervigilance
- (5) Exaggerated startle response

D: Negative alterations in cognitions and mood that began or worsened after the traumatic event: (two required)

1. Inability to recall key features of the traumatic event (usually dissociative amnesia; not due to head injury, alcohol, or drugs).
2. Persistent (and often distorted) negative beliefs and expectations about oneself or the world (e.g., "I am bad," "The world is completely dangerous").
3. Persistent distorted blame of self or others for causing the traumatic event or for resulting consequences.
4. Persistent negative trauma-related emotions (e.g., fear, horror, anger, guilt, or shame).
5. Markedly diminished interest in (pre-traumatic) significant activities.
6. Feeling alienated from others (e.g., detachment or estrangement).
7. Constricted affect: persistent inability to experience positive emotions.

E: Duration of the disturbance (symptoms in Criteria B, C, and D) is more than 1 month.

E: Trauma-related alterations in arousal and reactivity that began or worsened after the traumatic event: (two required)

1. Irritable or aggressive behavior
2. Self-destructive or reckless behavior
3. Hypervigilance
4. Exaggerated startle response
5. Problems in concentration
6. Sleep disturbance

F: The disturbance causes clinically significant distress or impairment in social, occupational, or other important areas of functioning.

F: Persistence of symptoms (in Criteria B, C, D, and E) for more than one month.

G: Significant symptom-related distress or functional impairment (e.g., social, occupational).

H: Disturbance is not due to medication, substance use, or other illness

Specify if: With dissociative symptoms.

In addition to meeting criteria for diagnosis, an individual experiences high levels of either of the following in reaction to trauma-related stimuli:

1. Depersonalization: experience of being an outside observer of or detached from oneself (e.g., feeling as if "this is not happening to me" or one were in a dream).
2. Derealization: experience of unreality, distance, or distortion (e.g., "things are not real").

Specify if: With delayed expression.

Full diagnosis is not met until at least six months after the trauma(s), although onset of symptoms may occur immediately.

(APA, 1994)

(APA, 2013)

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## APPENDIX B: WAYNE STATE IRB CONCURRENCE OF EXEMPTION

**WAYNE STATE  
UNIVERSITY**

IRB Administration Office  
87 East Canfield, Second Floor  
Detroit, Michigan 48201  
Phone: (313) 577-1628  
FAX: (313) 993-7122  
<http://irb.wayne.edu>

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### CONCURRENCE OF EXEMPTION

**To:** Michelle Sperlich  
Social Work Instruction Un  
4756 Cass Avenue

**From:** Dr. Deborah Ellis *M. Kusch/RS*  
Chairperson, Behavioral Institutional Review Board (B3)

**Date:** November 08, 2013

**RE:** IRB #: 1010113B3X

Protocol Title: Trauma Exposure and Depression in a Community Sample of First-Time Mothers

Sponsor:

Protocol #: 1310012517

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The above-referenced protocol has been reviewed and found to qualify for **Exemption** according to paragraph #4 of the Department of Health and Human Services Code of Federal Regulations [45 CFR 46.101(b)].

- Social/Behavioral/Education Exempt Protocol Summary Form (received in the IRB Office 10/28/2013)
- Protocol (received in the IRB Office 10/28/2013)

This proposal has not been evaluated for scientific merit, except to weigh the risk to the human subjects in relation to the potential benefits.

- 
- Exempt protocols do not require annual review by the IRB.
  - All changes or amendments to the above-referenced protocol require review and approval by the IRB **BEFORE** implementation.
  - Adverse Reactions/Unexpected Events (AR/UE) must be submitted on the appropriate form within the timeframe specified in the IRB Administration Office Policy (<http://irb.wayne.edu/policies-human-research.php>).

**NOTE:** Forms should be downloaded from the IRB Administration Office website <http://irb.wayne.edu> at each use.



## APPENDIX C: CONTENT VALIDITY INDEX PANEL CHECKLIST

### *Content Validity Index Panel Checklist*

The 2013 *DSM-5* lists the following A Criteria for “allowable” trauma exposure:

A: Exposure to actual or threatened death, serious injury, or sexual violence in one or more of the following ways:

- 1) Directly experiencing the event(s) as it occurred to others
- 2) Witnessing, in person, the event(s) as it occurred to others.
- 3) Learning that the event(s) occurred to a close family member or close friend. In cases of actual threatened death of a family member or friend, the event(s) must have been violent or accidental
- 4) Experiencing repeated or extreme exposure to aversive details of the traumatic event (e.g., first responders collecting human remains, police officers repeatedly exposed to details of child abuse)

Note: Criterion A4 does not apply to exposure through electronic media, television, movies, or pictures, unless this exposure is work related.

INSTRUCTIONS: Following is a checklist of different types of trauma exposures per the STACY study (Life Stressor Checklist; Wolfe & Kimerling, 1997, and the Abuse Assessment Screen; McFarlane et al., 1992). Please read each item and indicate with a check mark whether you think that the question assesses for a <i>DSM-5</i> “allowable” trauma for the PTSD diagnosis.	Is this a <i>DSM-5</i> allowable trauma? (Please check YES or NO)	
	YES	NO
Have you ever <i>been in</i> a serious disaster, for example, a massive earthquake, hurricane, tornado, fire, or explosion?		
Have you ever <i>been in</i> a war zone or refugee camp, or been a political detainee?		
Have you ever <i>seen</i> a serious accident, for example, a bad car wreck or an on-the-job accident?		
Have you ever <i>had</i> a very serious accident or accident-related injury, for example, a bad car wreck or an on-the-job accident?		
Was a close family member ever sent to jail?		
Have <i>you</i> ever been sent to jail?		
Were you ever put in foster care or put up for adoption?		
Did your parents ever separate or divorce while you were living with them?		
Have <i>you</i> ever been separated or divorced?		
Have you ever had serious money problems, for example, not enough money for food or place to live?		
Have you ever had a very serious physical or mental illness, for example, cancer, heart attack, serious operation, felt like killing yourself, or being hospitalized because of nerve		

problems?		
Have you ever had a procedure that was very painful or dangerous for medical reasons or as part of a ritual, like circumcision, or as part of something like a gang initiation?		
Have you ever been emotionally abused or neglected, for example, being frequently shamed, embarrassed, ignored, or repeatedly told that you were 'no good'?		
Have you ever been physically neglected, for example, not fed, not properly clothed, or left to take care of yourself when you were too young or ill?		
Have you ever had a hard time because of an abortion or miscarriage?		
Have you ever been responsible for taking care of someone close to you who had a severe physical illness or mental handicap, for example, cancer, stroke, Alzheimer's disease, AIDS, felt like killing themselves, hospitalized because of nerve problems, can't hear, see, or walk?		
Has someone close to you died suddenly or unexpectedly, for example an accident, sudden heart attack, murder, or suicide?		
Has someone close to you died?		
When you were young, BEFORE AGE 16, did you ever see violence between family members, for example, hitting, kicking, slapping, punching?		
Have you ever SEEN a robbery, mugging, or attack taking place?		
Have you ever BEEN robbed, mugged, or physically attacked by someone you did not know? By physically attacked, we mean hit, slapped, choked, burned, or beat up.		
BEFORE AGE 16, were you ever abused or physically attacked by someone you knew, for example, a parent, boyfriend, or husband? By physically attacked, we mean hit, slapped, choked, burned, or beat up."		
AFTER AGE 16, were you ever abused or physically attacked by someone you knew, for example, a parent, boyfriend, or husband? By physically attacked, we mean hit, slapped, choked, burned, or beat up.		
Have you ever been bothered or harassed by sexual remarks, jokes, or demands for sexual favors by someone at work or school, for example, a co-worker, a boss, a customer, another student, or a teacher?		
BEFORE AGE 16, were you ever touched or made to touch someone else in a sexual way because they forced or manipulated you in some way or threatened to harm you if you didn't?		
AFTER AGE 16, were you ever touched or made to touch someone else in a sexual way because they forced or		

manipulated you in some way or threatened to harm you if you didn't?		
BEFORE AGE 16, did you ever have oral, anal, or genital sex when you didn't want to because someone forced or manipulated you in some way or threatened to harm you if you didn't?		
AFTER AGE 16, did you ever have oral, anal, or genital sex when you didn't want to because someone forced or manipulated you in some way or threatened to harm you if you didn't?		
Are there any other traumatic things that happened to you but are not on our list?		

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**ABSTRACT****TRAUMA EXPOSURE, POSTTRAUMATIC STRESS, AND DEPRESSION IN A  
COMMUNITY SAMPLE OF FIRST-TIME MOTHERS**

by

**MICHELLE IRENE SPERLICH****August 2014****Advisor:** Dr. Arlene Weisz**Major:** Social Work (Infant Mental Health)**Degree:** Doctor of Philosophy

The adverse effects of posttraumatic stress and depression have separately been well-documented in the perinatal mental health literature. However, few studies have considered the comorbidity between trauma, posttraumatic stress *and* depression. This dissertation study brings attention to this comorbidity and explores implications of recent changes to diagnostic criteria for posttraumatic stress disorder related to the ability to predict postpartum depression and impairments in mother/infant bonding. Following a conceptual framework which outlines the effects of violence and trauma on adverse childbearing outcomes, hypotheses were that many women with depression in pregnancy would endorse trauma and would be at risk for subthreshold posttraumatic stress, that the type of traumas would differ based on diagnosis, and that posttraumatic stress in pregnancy would emerge as an important predictor of postpartum depression. Additional hypotheses were that recent changes in diagnostic criteria would reduce the apparent rate of posttraumatic stress disorder diagnosis in these data and would have the effect of limiting the ability to predict postpartum depression and impairments in mother/baby bonding. A secondary analysis of a prospective study of a diverse community sample of 1581



first-time mothers was undertaken. Multinomial logistic regression and receiver-operator growth curve modeling were used to test the study hypotheses. Results from the analyses broadly supported the hypotheses, finding differences in the kinds of traumatic exposures among diagnostic categories, risk for subthreshold posttraumatic stress regardless of trauma endorsement, and risk for postpartum depression among women with posttraumatic stress in pregnancy. Analyses also showed that diagnostic changes which constrain the trauma criterion for posttraumatic stress disorder limits the ability to predict women at risk for postpartum depression and bonding impairment. As a whole these analyses show that integrating mental health care to combine attention to depression, trauma, and posttraumatic stress is warranted and that changes to posttraumatic stress disorder diagnostic criteria must be further examined for implications for perinatal mental health. These analyses show that trauma-informed interventions are needed to help to improve the lives of mothers, babies, and families by addressing symptoms of posttraumatic stress that contribute to the development of postpartum depression.

## AUTOBIOGRAPHICAL STATEMENT

“Mickey” earned her Bachelor of Science degree in Special Education in 1983 and Master of Arts in English Literature in 1994, both from Eastern Michigan University. After working as a vocational teacher for several years, she obtained apprenticeship and professional training in midwifery and practiced as a Certified Professional Midwife from 1987 to 2004. During this time she became interested in women’s mental health issues during pregnancy and early motherhood, particularly related to sequelae of childhood and/or adult sexual trauma. She embarked on an independent qualitative research project in 1998 which culminated in the publication of a book, co-authored with Dr. Julia Seng, entitled *Survivor Moms: Women’s Stories of Birthing, Mothering, and Healing after Sexual Abuse* (Motherbaby Press, 2008).

Mickey joined Dr. Seng’s research team at University of Michigan in 2005 and served as Research Assistant/Project Manager for a variety of projects related to perinatal mental health until 2011, including an NIH/NINR-funded, ROI prospective cohort study of posttraumatic stress in pregnancy known as “STACY.” During this time, Mickey and Julia co-authored several papers and were co-investigators on a pilot project of an international psychoeducational intervention for pregnant survivors of abuse, known as the “Survivor Moms’ Companion.”

Mickey’s experiences as part of Seng’s multidisciplinary team sparked her interest in social work and infant mental health and led her to pursue doctoral studies at Wayne State University in 2010. In addition to her doctoral studies in the School of Social Work, Mickey has pursued a dual title with the Infant Mental Health program, has received training and fellowship support at the Merrill Palmer Skillman Institute for Child and Family Development, and has taught research methodology and statistics as a part-time faculty member in the School of Social Work. This dissertation study represents the culmination of Mickey’s doctoral training.