EXAMINING THE RELATIONSHIP BETWEEN MATERNAL STRESSFUL LIFE EVENTS AND UROGENITAL INFECTION IN PRETERM BIRTH USING A BIOBEHAVIORAL MODEL

A Dissertation

by

JOY LAVONNE ANDERSON

Submitted to the Office of Graduate Studies of
Texas A&M University
in partial fulfillment of the requirements for the degree of

DOCTOR OF PHILOSOPHY

August 2008

Major Subject: Health Education

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Approved by:

Chair of Committee,	Ranjita Misra
Committee Members,	Bruce Thompson
	James Eddy
	E. Lisako McKyer
Head of Department,	James Eddy

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ABSTRACT

Examining the Relationship between Maternal Stressful Life Events and Urogenital Infection in Preterm Birth Using a Biobehavioral Model.

(August 2008)

Joy Lavonne Anderson, B.S., Florida A&M University;

M.S., Florida State University

Chair of Advisory Committee: Dr. Ranjita Misra

This dissertation examined the relationship between maternal stressful life events and urogenital infection in preterm birth. A systematic literature review revealed ambivalent findings concerning the relationship between maternal stress and infection during pregnancy; the effects of this relationship on pregnancy outcome were not examined in the reviewed studies. The current study employed a biobehavioral model consisting of maternal stressful life events (illness among family members, divorced, moved, became homeless, partner lost job, mom lost job, argued with partner more than usual, partner did not want the child, inability to pay bills, got in a physical fight, partner went to jail, close friend/relative had a bad problem with drinking or drug use, and close friend/relative died) and urogenital infection (genital warts, herpes, chlamydia, gonorrhea, pelvic inflammatory disease, syphilis, Group B streptococcus, bacterial vaginosis, trichomoniasis, yeast infection, urinary tract infection, and

other infection) to examine the relationship between these variables in preterm birth. Data from 1,647 respondents of the 2005 Florida Pregnancy Risk Assessment Monitoring System survey were analyzed using descriptive statistics, chi-square and student *t*- tests, analysis of variance, and structural equation modeling (SEM). Of the respondents, 42% were White, 37.8% had preterm deliveries, and the mean age was 27.1 years. White mothers who became homeless ($p = 0.021$) or had a partner in jail ($p = 0.041$) during the 12 months prior to delivery had more preterm deliveries as compared to full-term deliveries. Other non-White mothers who had an ill family member ($p = 0.010$) had fewer preterm deliveries. In general, mothers diagnosed with Group B streptococcus during pregnancy ($p = 0.031$) had fewer preterm deliveries. Black mothers diagnosed with herpes ($p = 0.006$) had fewer preterm deliveries. SEM revealed a significant relationship between maternal stress and infection, in general ($p < 0.001$), and among White ($p < 0.001$), Black ($p < 0.001$), and Hispanic ($p < 0.001$) mothers. The interaction between these variables was not significant, in general, or among racial/ethnic groups. Results of this study indicate that culturally tailored prevention programs designed to help women cope with multiple risk factors may prove beneficial in reducing preterm birth rates.

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CHAPTER I

INTRODUCTION

Preterm birth is defined as the delivery of an infant at less than 37 weeks of gestation (USDHHS, 2000). This phenomenon is "associated with multiple complex and poorly understood, but interrelated, biologic, psychologic, and social factors that appear to be expressed in the common pathway of preterm birth" (IOM, 2007, p. xi). One of the *Healthy People* 2010 objectives is to reduce the rate of preterm birth to 7.6 percent by year 2010 (USDHHS, 2000). However, current rates not only exceed the proposed objective but continue to rise. For example, in 2004, the national average for preterm birth was 12.5 percent, representing an eight percent increase since year 2000 (March of Dimes, 2007).

Previous epidemiological investigations have identified several factors that are associated with increased risk of preterm birth (Lu & Chen, 2004; Lu & Halfon, 2003; McGregor et al., 1995; Wadhwa et al., 2001). Maternal stress and urogenital infection are the two most common independent risk factors for preterm birth (Lu & Halfon, 2003; Wadhwa et al., 2001). In this context, maternal stress is defined as any physical or psychological challenge that threatens or is perceived to have the potential to threaten the expectant mother's homeostasis (Moutquin, 2003b). Empirical evidence suggests that women

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This dissertation follows the style of *The Health Educator*.

experiencing increased levels of stress during pregnancy are at increased risk for preterm birth, even after controlling for biomedical, sociodemographic and behavioral factors (Wadhwa et al., 2001). This relationship is one of the more "consistent and unambiguous" etiologies of preterm birth (Wadhwa et al., 2001, p. 19). However, additional research is encouraged to determine the nature of the combined effects of stress and other obstetric risk factors such as infection on preterm birth (IOM, 2006).

Preterm birth may be mediated or moderated through maternal urogenital infections (Fiscella, 1996). These infections ascend into the mother's upper reproductive tract during pregnancy resulting in intra-amniotic infection (Fiscella, 1996, 2004). Preterm labor, preterm premature rupture of membranes and clinical chorioamnionitis are associated with intra-amniotic infection and are leading precursors to preterm birth (Fiscella, 1996, 2004; McGregor et al., 1995). Approximately 30-50% of preterm births are associated with infection (Othman, Neilson, & Alfirevic, 2007). Hence, maternal infections and their related immune pathophysiology are leading factors for further investigation of biological processes causing preterm birth (Wadhwa et al., 2001).

Studies of childbirth suggest that neuroendocrine and immune processes play important roles in the physiology and pathophysiology of preterm birth (Hobel & Culhane, 2003; Wadhwa et al., 2001). Maternal stress may influence one or both of these processes (Hobel & Culhane, 2003; Wadhwa et al., 2001). Wadhwa's Biobehavioural Model of Stress, Infection, and Preterm Birth (2001)

highlights a neuroendocrine pathway by which maternal stress may produce increased risk for preterm birth. The interaction of maternal stress with the neuroendocrine system possibly results in a premature and/or greater degree of activation of the placental-fetal endocrine systems that promote childbirth. Additionally, maternal stress may modulate immune responses to increase susceptibility to maternal infection and intrauterine or fetal inflammatory processes, thereby increasing the risk of preterm birth. The model also suggests that the neuroendocrine and immune processes extensively cross-regulate each other; therefore, exposure to high levels of stress and infectious pathogens during pregnancy yields an interaction and multiplicative effect on preterm birth (Wadhwa et al., 2001).

Little research, however, has empirically examined the impact of the relationship between stress and infection on pregnancy and/or adverse pregnancy outcomes that may arise from this interaction. Hence, the purpose of this study is to examine the relationship between maternal stressful life events and urogenital infection in preterm birth using a biobehavioral model. Elucidating the relationship between these variables is essential for planning, designing, and implementing tailored intervention strategies and improving pregnancy outcomes.

This dissertation is organized into five chapters (chapters II-IV are self-supporting manuscripts intended for publication). Chapter I provides an overview of the content that follows. Chapter II is a systematic literature review

of current research examining the relationship between maternal stress and urogenital infection in pregnancy. Limitations in the current research are highlighted and directions for future research are provided. Chapter III examines the relationship between maternal stressful life events and urogenital infection in preterm birth using a biobehavioral model. Chapter IV explores racial and ethnic differences associated with the above mentioned relationship (i.e., maternal stressful life events and urogenital infection) in preterm birth. Chapter V provides a summary of the previous chapters and directions for future research.

CHAPTER II

MATERNAL STRESS AND UROGENITAL INFECTION DURING PREGNANCY: A SYSTEMATIC REVIEW OF THE LITERATURE

Pregnancy outcome can be influenced by many risk factors that are experienced during the gestation period (Bale, 2005). One of the most troubling risk factors for obstetricians and reproductive health specialists is maternal stress (Bale, 2005). Maternal stress during pregnancy may expose the unborn child to increased risk for preterm birth, low birthweight, birth defects, physical and behavioral anomalies, neurological and behavioral impairments, and developmental delays (Bale, 2005; Krabbendam et al., 2005).

Studies examining the relationship between maternal stress during pregnancy and its associated outcomes have focused on various types of stress, e.g., emotional, nutritional, and physical (Bale, 2005). These stressors may vary from life events to daily hassles (Mulder et al., 2002). Research indicates that an expectant mother's central nervous and endocrine systems are activated during her physiological response to stressors (Mulder et al., 2002), thereby influencing the development of the unborn child (de Weerth & Buitelaar, 2005). The degree of maternal stress response varies based on genetic factors, personality characteristics, previous experience, social support, and coping skills. Therefore, pregnant women may respond differently to identical stressors (Mulder et al., 2002).

Since maternal infections have been studied as potentially preventable risk factors for adverse pregnancy outcomes (O' Neill, Hertz-Picciotto, Pastore, & Weatherley, 2003), they are of great concern to obstetricians and reproductive health specialists. These infections may affect the mother and/or unborn child, either *in utero* or at the time of delivery (Majeroni & Ukkadam, 2007). Many of the infections have been linked with adverse pregnancy outcomes similar to those identified in maternal stress research. Urogenital infections, especially those of the cervix and those transmitted through sexual contact, may cause pregnancy complications (Darwish, Makarem, Alnashar, & Hamadeh, 2005; Mullick, Watson-Jones, Beksinska, & Mabey, 2005). Additionally, urinary tract infections affect the prognosis of pregnancy outcome (Yaris et al., 2004).

Recent calls to action for research exploring pregnancy and its related adverse outcomes suggest that risk factors such as maternal stress and urogenital infection should be examined simultaneously (IOM, 2006). Thus, the purpose of this literature review was to systematically examine empirical evidence concerning the relationship between maternal stress and urogenital infection during pregnancy. Additionally, the review was designed to answer the following questions: is there a relationship between maternal stress and urogenital infection in human pregnancy?, if related, is the association protective or destructive in nature?, and what are the common methodological characteristics in literature assessing this relationship?

Methods

Seven computerized databases (*Academic Search Complete, CINAHL Plus, ERIC, Health Source: Nursing/Academic Edition, MasterFILE premier, MEDLINE, and Psychology & Behavioral Sciences Collection*) were searched for studies from year 2001 to year 2008 using variations and Boolean connections of key terms *stress*, *infection*, and *preterm birth*. In order to identify as many studies as possible, no limit was placed on sample size or study location. The initial search produced a small number of eligible studies (n = 2). Although these studies investigated the relationship between stress and infection during pregnancy, they did not examine the relationship within the context of pregnancy outcome. Therefore, the term preterm birth was replaced by the term *pregnancy* in order to expand the search and include studies examining the relationship between stress and infection during pregnancy (n = 22).

Studies were selected for review if they met the following inclusion criteria: were published in a peer-reviewed journal in English, represented empirical studies, and assessed the relationship between maternal stress and urogenital infection during pregnancy (n = 6). Studies were excluded if they did not test this relationship during human pregnancy (n = 5). To supplement this search, reference lists of the eligible studies were reviewed and additional publications that met all the inclusionary and exclusionary criteria were located and added to the pool (n = 4). The eligible studies from the initial search were included; thus, the final sample was comprised of seven studies.

Each study was summarized according to Garrard's Matrix Method for conducting systematic literature reviews (Garrard, 2004). The matrix included author, year of publication, purpose of the study, number of study participants, sample description, design and methodology, major findings, and a methodological quality score. The summaries are listed in chronological order in Table 1. Each study's methodological characteristics were assessed using an abstraction form modified from Goodson, Buhi, and Dunsmore (2006). Individual characteristic scores were totaled to determine the overall methodological quality score (MQS) of each study (Lee, Schotland, Bacchetti & Bero, 2002). The highest possible MQS for eligible studies was 24. The MQSs and associated criteria are presented in Table 2.

For the purposes of this review, each study's assessment of a relationship between maternal stress and urogenital infection during pregnancy was examined and noted as a separate finding. Each finding was categorized and coded according to the type of stress and infection investigated in the study. Additionally, the nature of the relationship between the variables was considered.

Results

Characteristics of Investigated Studies

Of the seven eligible studies, approximately half (n = 3) were published in the last five years (Harville, Savitz, Dole, Thorp, & Herring, 2007; Nelson et al., 2008; Trabert & Misra, 2007). These studies were published in journals

Table 1

Description of Studies

Author	Purpose of Study	n	Sample Description	Design & Methodology	Major Findings	MQS Score
Ruiz, RJ, Fullerton, J, Brown, CEL, Schoolfield, J (2001)	To better delineate the role of stress and cortisol in patients having PTL and PTB; examine the relationships of maternal cortisol and perceived stress to PTL and PTB as predicted by fFN; examine the confounding role that genitourinary infections may play in this complex interrelationship	78	central Texas, 2 private practice OB/GYN offices, predominantly Medicaid clients, 40% Hispanic, 20% African American, 40% Anglo-American	Prospective, longitudinal	There was no relationship observed between genitourinary infection and PSS scores.	11
Culhane, JF, Rauh, V, McCollum, KF, Hogan, VK, Agnew, K, Wadhwa, PD (2001)	To examine whether chronic maternal stress predisposes pregnant women to infection, and whether the effects of chronic stress on susceptibility to infection are independent of the effects of other established sociodemographic and behavioral risk factors	454	Philadelphia, PA, consortium of public health centers, 62% African American	Cross-sectional	After controlling for the effects of all sociodemographic and behavioral risk factors, moderate to high levels of chronic stress remained significantly associated with BV status.	14

Table 1. Continued

Author	Purpose of Study	n	Sample Description	Design & Methodology	Major Findings	MQS Score
Ruiz, RJ, Fullerton, J, Brown, CEL, Dudley, D (2002)	To investigate the relationships and predictability of perceived stress, CRH levels, and PTL on gestational age at birth, to develop a predictive model of preterm birth using the variables and ethnicity, to examine differences in CRH levels by groups that did or did not have genitourinary infections, and to examine proportions of cigarette smoking in groups that had high or low stress scores	78	central Texas, 2 private practice OB/GYN offices, predominantly Medicaid clients, 20% Hispanic, 5% African American, 75% Anglo-American	Prospective, longitudinal	No relationship was observed between genitourinary infection and PSS scores.	11
Culhane, JF, Rauh, V, McCollum, KF, Elo, IT, Hogan, V (2002)	To explore the contribution of chronic social stressors to racial/ethnic differences in rates of BV among pregnant women	2304	Philadelphia, PA, public health centers, 67% Black	Cross-sectional	After adjustment for sociodemographic and behavioral risk, perceived stress was associated independently with BV.	11

Table 1. Continued

Author	Purpose of Study	n	Sample Description	Design & Methodology	Major Findings	MQS Score
Trabert, B, Misra, DP (2007)	To examine a number of social and behavioral factors that may relate to BV prevalence during pregnancy in a low-income African American population	438	Maryland, Johns Hopkins Medical Institution Clinics and Hospital, 100% African American	Cross-sectional	There were no statistically significant associations between BV and anxiety or hassles (stress).	13
Harville, WE, Savitz, DA, Dole, N, Thorp, JM, Herring, AH (2007)	To examine the association between stress and BV in a larger cohort study of North Carolina pregnant women that included several reported measures of stress as well as measurement of stress hormones and measurement of BV at two time points	897	North Carolina, University of North Carolina Hospitals, 22% African American, 68% White, 10% Asian/Native American	Longitudinal	After adjustment for confounders, BV odds ratios were modestly elevated only for perceived stress, total life events, and John Henryism. Risk of BV was slightly raised in those not in the lowest quartile of cortisol and CRH levels. Risk was modestly higher in the second lowest quartile of both cortisol and CRH.	14

Table 1. Continued

Author	Purpose of Study	n	Sample Description	Design & Methodology	Major Findings	MQS Score
Nelson, DB, Bellamy, S, Nachamkin, I, Ruffin, A, Allen-Taylor, L, Friedenberg, FK (2008)	To determine the clinical, behavioral and/or demographic factors contributing to asymptomatic BV among pregnant women and to examine if asymptomatic BV positive pregnant women had an increased risk of adverse pregnancy outcomes compared to symptomatic BV positive pregnant women	754	Philadelphia PA, 2 obstetrical practices, Hospital of the University of Pennsylvania, privately and publicly insured, 72% African American	Cross-sectional	BV positive pregnant women without symptoms reported significantly lower mean stress scores. Asymptomatic BV positive women reported lower stress scores.	12

Table 2

Methodological Quality Score: Criteria for Assessment and Frequency Distributions among Reviewed Studies

Methodology Criteria	Scores	Distribution of characteristics among the 7 studies Frequency (n)	Percent (%)
Definition:	Conceptual (Both) = 2	0	0.0
	Conceptual (One variable) = 1	4	57.1
	Not Defined = 0	3	42.9
Definition:	Operational (Both) = 2	7	100.0
	Operational (One variable)= 1	0	0.0
	Not Defined = 0	0	0.0
Validity:	Reported (Both) = 2	0	0.0
	Reported (One measure) = 1	0	0.0
	Not reported = 0	7	100.0
Reliability:	Reported (Both) = 2	1	14.3
	Reported (One measure) = 1	3	42.9
	Not reported = 0	3	42.9
Theoretical Framework:	Presented = 2	0	0.0
	Implied = 1	0	0.0
	Not presented = 0	7	100.0
Research Paradigm:	Mixed Methods = 2	0	0.0
	Quantitative/Qualitative = 1	7	100.0
Design:	Longitudinal = 2	3	42.9
	Cross-sectional = 1	4	57.1
Sample Size:	Large (>300) = 3	5	71.4
	Medium (>100 and <300) = 2	0	0.0
	Small (<100) = 1	2	28.6
Sample Design:	Random and nationally	0	0.0
	Random and not nationally	0	0.0
	Convenience = 0	7	100.0
Data Analysis:	Multivariate = 4	1	14.3
	Multiple/Logistic regression = 3	6	85.7
	Bivariate/ANOVA = 2	0	0.0
	Univariate/Descriptives = 1	0	0.0
Limitations:	Stated = 1	7	100.0
	Not stated = 0	0	0.0

representing the fields of maternal and child health, nursing, and obstetrics and gynecology.

All of the reviewed studies examined maternal stress as an independent variable (Culhane, Rauh, McCollum, Elo, & Hogan, 2002; Culhane et al., 2001; Harville et al., 2007; Nelson et al., 2008; Ruiz, Fullerton, Brown, & Dudley, 2002; Ruiz, Fullerton, Brown, & Schoolfield, 2001; Trabert & Misra, 2007). However, none presented a conceptual definition of maternal stress. Additionally, none of the studies used a theoretical framework to guide scientific inquiry.

Studies' Methodological Quality

Each study was assigned a MQS based on definitions (conceptual and operational), reliability and validity, use of research paradigm and theoretical framework, research design and sampling, and type of data analysis and limitations. The range of MQS scores varied between 11 and 14. The mean, median and mode for the MQS distribution were similar (mean = 12.28 ± 1.38, median = 12, and mode = 11), indicating that the studies were of similar methodological quality. The frequency distribution of each methodological characteristic ranged from 0 to 100%.

With regards to research paradigm, all the seven studies were quantitative in nature, although with varying research designs. Fifty-seven percent of these quantitative studies (n = 4) employed a cross-sectional design (Culhane et al., 2002; Culhane et al., 2001; Harville et al., 2007; Nelson et al., 2008), while the

remaining studies were longitudinal (n = 3; Ruiz et al., 2002; Ruiz et al., 2001; Trabert & Misra, 2007). None of the studies employed a qualitative or mixed methods (i.e., quantitative and qualitative) approach.

All seven studies employed convenience/non-probability (cohort) samples (Culhane et al., 2002; Culhane et al., 2001; Harville et al., 2007; Nelson et al., 2008; Ruiz et al., 2002; Ruiz et al., 2001; Trabert & Misra, 2007). Although the sample sizes ranged from 78 to 2304, most studies (71.4%, n = 5) reported sample sizes greater than 300 respondents (Culhane et al., 2002; Culhane et al., 2001; Harville et al., 2007; Nelson et al., 2008; Trabert & Misra, 2007). The remaining studies employed fewer than 100 respondents in their research project (Ruiz et al., 2002; Ruiz et al., 2001).

All of the studies were conducted in various regions of the United States, i.e., central Texas (Ruiz et al., 2002; Ruiz et al., 2001), Philadelphia, Pennsylvania (Culhane et al., 2002; Culhane et al., 2001; Nelson et al., 2008), North Carolina (Harville et al., 2007), and Maryland (Trabert & Misra, 2007). In regards to the racial/ethnic composition of the sample, Trabert & Misra (2007) employed an exclusively African American sample while three of the studies employed a predominantly African American sample (Culhane et al., 2002; Culhane et al., 2001; Nelson et al., 2008). Ruiz, Fullerton, Brown, & Dudley (2002) excluded African American respondents from data analysis due to small group size.

Data collection method also varied greatly within the eligible studies. Maternal stress data was primarily collected using in-person interviews (n = 3) (Culhane et al., 2001; Nelson et al., 2008; Trabert & Misra, 2007). However, one study utilized both telephone interviews and self-administered questionnaires (Harville et al., 2007). The remaining three studies did not disclose information regarding their method of data collection (Culhane et al., 2002; Ruiz et al., 2002; Ruiz et al., 2001).

Maternal stress measures differed among the studies. Operationally, the majority of reviewed studies (57.1%, n = 4) measured maternal stress using only the *Perceived Stress Scale* (PSS; Culhane et al., 2001; Nelson et al., 2008; Ruiz et al., 2002; Ruiz et al., 2001). Two studies employed additional measures along with the PSS (Culhane et al., 2002; Harville et al., 2007). One study utilized measures of anxiety and hassles to operationalize maternal stress (Trabert & Misra, 2007).

Data on infections were obtained from test results concerning respondent's biological specimens, i.e., vaginal smears, genital tract swabs, perineal/rectal swabs, and urine samples. These samples were collected either by clinicians or patients themselves. The collection protocols also varied greatly. Three of the studies employed clinician-based collection (Culhane et al., 2001; Ruiz et al., 2002; Ruiz et al., 2001), while two studies employed both clinician and patient self-collection methods (Nelson et al., 2008; Trabert & Misra, 2007). One study employed exclusively self-collection of biological specimens (Harville

et al., 2007). The data collection method was not disclosed for the study conducted by Culhane et al. (2002).

Urogenital infection measures also differed among the studies. In 71.4% of reviewed studies (n = 5), bacterial vaginosis (BV) was the only urogenital infection of interest (Culhane et al., 2002; Culhane et al., 2001; Harville et al., 2007; Nelson et al., 2008; Trabert & Misra, 2007). In each of these studies, BV was diagnosed according to the Nugent Method, a scoring system which minimizes clinical subjectivity and is based on proportions of distinguishable bacterial morphological types (i.e., 0-3 = positive, 4-6 = intermediate, 7-10 = positive; Culhane et al., 2002; Culhane et al., 2001). The remaining studies (n = 2) employed a different method of BV diagnosis involving pH, clue cell, and whiff test analyses (Ruiz et al., 2002; Ruiz et al., 2001). These studies assessed the presence of additional urogenital infections. Urinary tract infection and chlamydia were diagnosed by urine culture and the Genoprobe method, respectively. Group B streptococcus was diagnosed using chart review.

In terms of psychometric measures, none of the studies reported testing of data for validity. Although some of the studies employed reliability testing (n = 4), the reporting was infrequent. Regarding the maternal stress variable, four of the studies reported that reliability testing was conducted on the study's data (Culhane et al., 2001; Ruiz et al., 2002; Ruiz et al., 2001; Trabert & Misra, 2007). Additionally, one study reported assessing inter-rater reliability for evaluation of biological samples for urogenital infection (Culhane et al., 2001).

Data analysis techniques ranged from descriptive to multivariate analyses among the studies. Multivariate techniques were the highest level of data analysis conducted in all studies. Multiple/logistic regression represented 85.7% (n = 6) of these methods (Culhane et al., 2002; Culhane et al., 2001; Nelson et al., 2008; Ruiz et al., 2002; Ruiz et al., 2001; Trabert & Misra, 2007), while 14.3% (n = 1) were generalized estimating equations (Harville et al., 2007).

The final methodological characteristic assessed whether the authors addressed the limitations of their studies. Additionally, authors' consideration of nomothetic causal effects was noted. All studies reported limitations associated with their findings (Culhane et al., 2002; Culhane et al., 2001, Harville et al., 2007; Nelson et al., 2008; Ruiz et al., 2002; Ruiz et al., 2001; Trabert & Misra, 2007); however, one study suggested that maternal stress caused urogenital infection (BV; Culhane et al., 2001).

Description of Study Findings

Three studies assessed the relationship between maternal stress and urogenital infection using multiple stress measures, thereby producing more than one finding (Table 3; Culhane et al., 2001; Nelson et al., 2008; Ruiz et al., 2002; Ruiz et al., 2001; Trabert & Misra, 2007). The remaining studies employed one stress measure (Culhane et al., 2002; Harville et al., 2007).

Table 3

Association between Maternal Stress and Urogenital Infection by Stress in Empirical Studies

	Nature of Finding/Relationship				
Stress Measure	Positive	Negative	No relationship	Total	Percentage
Perceived Stress Scale	4	0	2	6	50
Objective Stress	1	0	0	1	8.33
Neighborhood Stress	1	0	0	1	8.33
Anxiety	0	0	1	1	8.33
Hassles	0	0	1	1	8.33
Spielberger State-Trait Anxiety Inventory	0	1	0	1	8.33
Sarason Life Experiences Survey	1	0	0	1	8.33
Total	7	1	4	12	100
Percentage	58.33	8.33	33.33	100	

Consequently, the seven studies produced a total of 12 findings (average = 1.71/study, range 1-3). Maternal stress was evaluated as an independent (predictor) variable for urogenital infection in all reviewed studies.

Most findings (83.3%, n = 10) consisted of tests for maternal stress and bacterial vaginosis. Nearly seventeen percent (n = 2) of findings consisted of tests of maternal stress and a collective urogenital infection variable comprised of BV, Group B strep, urinary tract infection, and chlamydia. The nature of the relationship between maternal stress and the various urogenital infection variables was ambivalent – 58.3% of the findings rendered a positive relationship, 33.3% yielded no statistically significant relationship, and 8.3% indicated an inverse relationship.

Discussion

This systematic review contributes to the body of literature in a number of ways. First, this review identified a gap in the literature, as evidenced by the paucity of publications simultaneously examining the relationship between maternal stress and urogenital infection during pregnancy. Second, this review is the first to systematically examine empirical evidence of this relationship. Lastly, this review offers an analytical dimension not found in nonsystematic reviews, as it provides a critical assessment of the methodological quality of the reviewed studies.

As with any investigation, this review possesses limitations. The search protocol may have excluded applicable studies not indexed in the selected

databases or cited in the reviewed studies. Also, the methodological quality instrument was neither tested for validity nor reliability. Since the MQS favors longitudinal and mixed-method designs, random and nationally representative samples, and theory-driven inquiries, these criteria may be inappropriate for research assessing maternal stress and urogenital infection during pregnancy. Additionally, the criteria may have attenuated the findings and conclusions presented in this review.

The average MQS for the reviewed studies was 12.28. This value is only slightly above the MQS scale's mid-point (12), indicating that studies assessing the relationship between maternal stress and urogenital infection during pregnancy can be greatly improved. One area for improvement may involve the reporting of theory-guided research. It was surprising to note that studies assessing maternal stress and urogenital infection during pregnancy were not grounded in theory, since the field of psychoneuroimmunology, which studies the influence of stress on immune function and its effect on the onset and progression of disease, now includes models for pregnancy (Coussons-Read, Okun, & Simms, 2003). Theory, research, and practice lie along a continuum along which researchers should move with ease (Glanz, Rimer & Lewis 2002). Therefore, it is difficult to explain the lack of theory reported in research studies. Possible explanations include scholarly journal limitations, researcher's replication and enhancement of prior research, and the researcher's background.

Nevertheless, researchers should re-evaluate the importance of theory usage in studies assessing maternal stress and urogenital infection during pregnancy.

Application of theory in this research will ease the task of conceptualizing and operationalizing maternal stress. While all studies provided operational definitions of maternal stress, the operational measures varied across the studies; none provided a conceptual definition. Researchers have cautioned about the consequences associated with conceptual and methodological inconsistencies in research designs which make it difficult to determine what types of stress cause poor pregnancy outcomes (Gennaro & Hennessy, 2003). Additionally, findings regarding the relationship between maternal stress and preterm birth differ based on how stress is conceptualized (Gennaro & Hennessy, 2003).

Some researchers conceptualize maternal stress as daily hassles, psychological distress, and/or perceived stress (Gennaro & Hennessy, 2003). Other researchers have focused on stressful life events, anxiety, nervousness, depression, and psychic functioning (Moutquin, 2003b). Although anxiety, depression, and anger are byproducts of stress, a number of researchers assert that these terms should not be used synonymously with stress (Gennaro & Hennessy, 2003; Moutquin, 2003b).

Another related issue is that stress measures differ according to the conceptual definitions of stress. Different measures have yielded different, even contradictory results (Hobel & Culhane, 2003). These findings suggest that

future research should develop a universal protocol for conceptualizing and operationalizing maternal stress during pregnancy. Development of this protocol should include the combination and enhancement of various stress measures that have proven valid and reliable in prior research. This approach will result in a comprehensive protocol to assist researchers in identifying stressors and developing interventions that provide coping skills for maternal stress during pregnancy; thereby improving pregnancy outcomes.

Lastly, the use of psychometric measures is an area for improvement. In the reviewed studies, tests of validity are absent while tests of reliability are infrequent. Although researchers should be concerned about both measures, validity is of greater importance (Windsor, Clark, Boyd & Goodman, 2004). Windsor et al. (2004) advises that if the data does not measure what it is intended to measure, reliability is irrelevant. Increased use of psychometric measures can strengthen research conclusions and enhance generalizability.

In conclusion, the paucity of studies on maternal stress and infection indicates a need for further investigation. Future research should theoretically examine the relationship between maternal stress and urogenital infection during pregnancy. Application of theory will ease the task of conceptualizing and operationalizing maternal stress. Researchers should also use rigorous psychometric techniques to establish validity and reliability of the data employed in their investigations. Although research has identified both maternal stress and urogenital infection as independent predictors of pregnancy outcome, this

literature review was unable to provide conclusive evidence regarding the nature of the relationship between these variables during pregnancy. However, positive findings identified through this review can serve as impetus for future research examining the nature of the relationship between maternal stress and urogenital infection and adverse pregnancy outcomes (e.g., preterm birth, low birthweight, and infant mortality).

CHAPTER III

MATERNAL STRESSFUL LIFE EVENTS AND UROGENITAL INFECTION IN PRETERM BIRTH: EXPLORING A BIOBEHAVIORAL APPROACH

Preterm birth, defined as the delivery of an infant before 37 weeks of gestation (USDHHS, 2000), is increasingly conceptualized as a common but complex disorder (IOM, 2006; Green et al., 2005). Currently in the United States (U.S.), preterm birth is the primary determinant of very low birth weight and the leading cause of neonatal mortality (Fiscella, 2004; Green et al., 2005). It is also the leading cause of infant mortality in many developed nations (Kramer et al., 2001). Additionally, preterm birth is the primary determinant of early childhood mortality and morbidity in the U.S. (Green et al., 2005).

In 2000, the U.S. preterm birth rate was 11.6 percent (March of Dimes, 2007). The Healthy People initiative recognized this problem as a public health priority and set an objective to reduce the preterm birth rate to 7.6 percent by the year 2010 (objective 16-11a ; USDHHS, 2000). However, subsequent prevention efforts have proven unsuccessful in reducing the U.S. preterm birth rate. In fact, the most recent data available from the March of Dimes' Peristats System indicates that annually 12.5 percent of women give birth prematurely, representing an eight percent increase since the year 2000 (March of Dimes, 2007).

In response to the Healthy People 2010 objective and research findings concerning near term infants i.e., those born between 35 to 37 weeks of gestation (Lothian, 2006), the March of Dimes' perinatal research program made significant changes to its preterm birth research agenda. In 2001, based on findings suggesting that near term infants incurred increased risks or consequences associated with preterm birth, even at normal birth weight, (Buekens & Klebanoff, 2001), the research focus was shifted from birthweight to gestational age. Recognizing the need for development of targeted interventions for individuals at increased risk for preterm birth, the program also shifted in the emphasis from behavioral to social and biological mechanisms and interactions (Buekens & Klebanoff, 2001).

Five years later in 2006, with the rates of preterm birth still rising, the Institute of Medicine (IOM) *Committee on Understanding Premature Birth and Assuring Healthy Outcomes* made recommendations for improved research efforts while recognizing prevention as the key to significant gains in the study of preterm birth (IOM, 2006). Previous research focused on studying individual risk factors in isolation which may explain why prevention programs have not yielded expected results. Hence, current IOM recommendations emphasize the need to study multiple risk factors for preterm birth simultaneously (IOM, 2006).

Maternal stress and urogenital infection have been identified as independent risk factors for preterm birth (Lu & Halfon, 2003; Wadhwa et al.,

2001). Maternal stress is defined as any physical or psychological challenge that threatens or is perceived to have the potential to threaten homeostasis within an expectant mother (Moutquin, 2003b). The belief that maternal psychological factors can affect birth outcomes can be traced to Biblical times (Kramer et al., 2001). However, several investigations on the relationship between stress, immune status, and overall health (Gennaro & Hennessy, 2003), sparked researchers' interest in the relationship between stress and preterm birth (Kramer et al., 2001). The earliest empirical studies analyzing this relationship were published in the late 1970s (Moutquin, 2003b).

In the past two decades, urogenital infection emerged as an important contributing factor to preterm labor (Othman, Neilson, & Alfirevic, 2007) thereby impacting preterm birth rates (Fiscella, 1996). Globally, these infections affect nearly one billion women per year (Othman et al., 2007). Studies suggest that urogenital infections ascend into the mother's upper reproductive tract during pregnancy resulting in intra-amniotic infection (Fiscella, 1996). Known effects of intra-amniotic infection include preterm labor, preterm premature rupture of membranes, and clinical chorioamnionitis and are leading precursors of preterm birth (Fiscella, 1996; McGregor et al., 1995). As many as 30-50% of preterm births are associated with various markers of intra-amniotic infection (Othman et al., 2007). The relationship between urogenital infection and preterm birth has been recognized by some physicians for more than 50 years (Goldenberg, Culhane, & Johnson, 2005).

Although the relationship between stress and preterm birth is well accepted, additional research is necessary to determine the combined effects of stress and other obstetric risk factors, such as infection, on pregnancy outcomes (IOM, 2006). Furthermore, biological and/or psychosocial links of maternal stress and their association with urogenital infection during pregnancy provide differing and conflicting conclusions (Culhane, Rauh, McCollum, Elo, & Hogan, 2002; Culhane et al., 2001; Harville, Savitz, Dole, Thorp, & Herring, 2007; Nelson et al., 2008; Ruiz, Fullerton, Brown, & Dudley, 2002; Ruiz, Fullerton, Brown, & Schoolfield, 2001; Trabert & Misra, 2007). For example, while Nelson et al. (2008) indicate that there is a significant positive relationship between perceived stress and urogenital infection (bacterial vaginosis), Ruiz et al. (2002) indicate that there is no relationship, highlighting the need for further research examining this relationship in pregnancy outcomes.

Thus, the purpose of this study was to examine the relationship between maternal stressful life events and urogenital infection in preterm birth using a biobehavioral model. The model was based on Wadhwa's Biobehavioural Model of Stress, Infection, and Preterm Birth (2001) and was developed to answer the following research questions: is there a relationship between stress and infection in preterm birth after controlling for possible confounders in the model?, and does an interaction between stress and infection yield a multiplicative effect on preterm birth?

Methods

Study Sample

The 2005 Florida Pregnancy Risk Assessment Monitoring System (PRAMS) data was utilized to address the aforementioned research questions. PRAMS is a surveillance project of the Centers for Disease Control and Prevention (CDC) and state health agencies (USDHHS, 2007). PRAMS was initiated in 1987 with the goal of improving the health of mothers and infants by reducing adverse birth outcomes such as low birthweight, infant mortality and morbidity, and maternal morbidity. The state of Florida is one of 37 PRAMS participating states (USDHHS, 2007). The state's Department of Health (DOH) Office of Vital statistics, DOH Bureau of Epidemiology, and county health departments assist with this joint effort (Florida DOH, 2007). Annually, PRAMS collects state-specific, population-based data on maternal attitudes and experiences before, during, and shortly after pregnancy. PRAMS data is used for planning and assessing health programs and for describing maternal experiences that may influence maternal and infant health (USDHHS, 2007).

Participants for the 2005 Florida PRAMS survey included 2,785 mothers who gave birth to a live infant in 2005 (Florida DOH, 2007) and were selected from all births recorded by the Florida Office of Vital Statistics, using a stratified random sampling strategy based on maternal race, age, and infant birth weight:

Stratum 1: White/low birth weight/age greater than or equal to 20 years old

Stratum 2: Black/low birth weight/age greater than or equal to 20 years old

Stratum 3: Low birth weight/age less than 20 years old

Stratum 4: White/normal birth weight/age greater than or equal to 20 years old

Stratum 5: Black/normal birth weight/age greater than or equal to 20 years old

Stratum 6: Normal birth weight/age less than 20 years old

The 2005 Florida PRAMS data was primarily collected via mail. Secondary data collection was conducted via telephone interview with non-responders approximately one month after the initial survey mail-out. Participants responded to the survey two to five months post-delivery (Florida DOH, 2007).

Measures

Dependent variable

Preterm birth. Preterm birth was assessed based on PRAMS-linked birth certificate data. Reports of gestation periods lasting fewer than 37 weeks were treated as positive responses. The variable was dichotomized (*yes* vs. *no*).

Independent variables

Maternal stress. Maternal stress was assessed based on the mother's report of stressful life events experienced during the 12 months before giving birth. Thirteen life events served as indicators for *maternal stress – illness among family members, divorced, moved, became homeless, partner lost job,*

mom lost job, argued with partner more than usual, partner did not want the child, inability to pay bills, got in a physical fight, partner went to jail, close friend/relative had a bad problem with drinking or drug use, and *close friend/relative died*. For analyses involving interaction effects, a maternal stress construct was created by summing the 13 stressful life events. The range for this construct was 0-13, with a higher score indicating greater stress during pregnancy. The reliability of the construct was 0.664.

Infection. Infection was assessed based on the mother's self-report of being informed by a doctor, nurse, or health care worker during her pregnancy that she had a urinary tract infection, a sexually transmitted disease, or any vaginal infection. The mother's specification of diagnosis with any of 12 urogenital infections (i.e., genital warts [warts], herpes, chlamydia, gonorrhea, pelvic inflammatory disease [PID], syphilis, Group B streptococcus (Group B strep), bacterial vaginosis, trichomoniasis [trich], yeast infection, urinary tract infection [UTI], and other infection) was also considered. An affirmative answer to either question was treated as a positive response. The 12 infections served as indicators for *urogenital infection*. For analyses involving interaction effects, an infection construct was created by summing the 12 urogenital infections. The range for this construct was 0-12, with a higher score indicating greater exposure to infection during pregnancy. The reliability of the construct was 0.360.

Covariates

Maternal age. Age was obtained from PRAMS-linked birth certificate data.

Plurality. Plurality was obtained from PRAMS-linked birth certificate data.

Pre-pregnancy body mass index (BMI). BMI was calculated based on the mother's self-reported height, without shoes, and weight, prior to pregnancy.

Smoking during pregnancy. Smoking status of the respondent was obtained from PRAMS-linked birth certificate data. This variable was dichotomized (yes vs. no).

Maternal race. Race was obtained from PRAMS-linked birth certificate data and reported as White, Black, Chinese, Japanese, Filipino, Other Asian, Other non-White, Hawaiian, American Indian, or Alaskan Native. This variable was dichotomized to create comparison groups with similar sample sizes (*White* vs. *non-White*).

Maternal education. Educational attainment was obtained from PRAMS-linked birth certificate data and was reported as 0-8 years, 9-11 years, 12 years, 13-15 years, or ≥ 16 years. This variable was dichotomized for uniform distribution (*below median* vs. *at median and above*).

Previous low birth weight (PLBW). PLBW were assessed based on the mother's self-report of delivery of an infant weighing less than 5 pounds, 8

ounces just prior to the current delivery. This variable was dichotomized (yes vs. no).

Previous preterm birth (PPB). PPB was assessed based on mother's self-report of delivery of an infant more than 3 weeks before its due date just prior to the current delivery. This variable was dichotomized (yes vs. no).

Assisted reproduction. Assisted reproduction was assessed based on the mother's report of receiving treatment from a doctor, nurse, or other health care worker to aid with conception. Maternal indication of receiving any of the surveyed treatments during the month prior to pregnancy (fertility-enhancing drugs prescribed by a doctor, artificial insemination, assisted reproductive technology such as in vitro fertilization, or other medical treatment as specified by the mother) was also considered. An affirmative answer to either question was treated as a positive response. This variable was dichotomized (yes vs. no).

Inadequate prenatal care. Inadequate prenatal care was assessed by maternal self-report of timing of first visit for prenatal care. An answer greater than 12 weeks or 3 months was treated as a positive response. This variable was dichotomized (yes vs. no).

Medical complications. Medical complications was based on mother's report of having medical problems during the pregnancy. An affirmative answer to any of the 12 surveyed problems (diabetes prior to pregnancy, diabetes during pregnancy, vaginal bleeding, kidney infection, severe nausea, incompetent cervix, hypertension, problems with placenta, preterm labor pains, premature

rupture of membranes, blood transfusion, or an injury sustained from a car accident) was treated as a positive response. This variable was dichotomized (yes vs. no).

Income. Income was obtained from the PRAMS questionnaire and was reported as ≤ $10,000, $10,000 - $14,999, $15,000 - $19,999, $20,000 - $24,999, $25,000 - $34,999, $35,000 - $49,999, or ≥ $50,000, representing total household income before taxes during the 12 months prior to giving birth. This variable was dichotomized for uniform distribution (below median vs. at median and above).

Data Analysis

Prior to analysis, data were cleaned and missing records were handled by listwise deletion using statistical software. Factor analysis was conducted to address questions of construct validity for maternal stress and infection (Thompson, 2004), while reliability was assessed by evaluating internal consistency of the data (Huck, 2003).

Descriptive statistics were utilized to describe the sample. Chi-square tests, student *t*-test, and analysis of variance (ANOVA) were employed to test for significant differences among categorical and interval variables. Structural equation modeling (SEM) was utilized to explore the relationship or independent effects of maternal stressful life events and urogenital infection on preterm birth. SEM was also utilized to determine interaction effects of maternal stressful life events and urogenital infection on preterm birth. Data

were analyzed using SPSS 15.0 and AMOS 16.0 structural equation modeling software.

Results

The total number of mothers who were contacted for participation in the 2005 Florida PRAMS was 2785; 73.5% (n=2047) responded to the survey. Since, not all mothers responded to the survey, the data were analyzed for response bias in demographic variables of interest. This analysis revealed significant differences in the maternal age, marital status, maternal race, and maternal education of respondents versus non-respondents (Table 4). Respondents were older, married, White, and had higher educational attainment as compared to non-respondents. However, there were no significant differences between the two groups in the study's outcome variable (preterm birth). Listwise deletion was used to identify respondents with complete data for variables of interest in this study. The final sample was comprised of 1,647 mothers.

The mean age of the mothers within the sample was 27.1 ± 6.44 years. The study sample was 42.0% White and 58.0% non-White. The median educational attainment and income levels were 12 years and $20,000-$24,999, respectively. Approximately, nine percent of the mothers reported smoking during pregnancy. Overall, 37.8% of the mothers within this sample gave birth to preterm infants.

Table 4

Respondent vs. Non-Respondent Differences (n=2785)

	Mean (SD)			Test Statistic			
Variable	Resp.	Nonresp.	df	t	χ^2	p	Cohen's d
Maternal race	0.62 (0.485)	0.78 (0.414)	1		61.054	<0.001	0.351
Maternal education	0.77 (0.418)	0.64 (0.480)	1		48.557	<0.001	0.294
Marital status	0.51 (0.500)	0.42 (0.494)	1		16.037	<0.001	0.173
Maternal age	26.77 (6.622)	25.71 (6.603)	2783	-3.729		<0.001	0.160
Smoking during pregnancy	0.09 (0.285)	0.11 (0.310)	1		2.107	0.147	0.061
Plurality	1.07 (0.267)	1.06 (0.254)	2783	-0.861		0.389	0.037
Preterm birth	0.38 (0.485)	0.40 (0.490)	1		0.652	0.419	0.035

Significant differences between mothers delivering preterm as compared to full-term emerged in plurality, pre-pregnancy BMI, previous low birth weight (PLBW), previous preterm birth (PPB), assisted reproduction, and medical complications (Table 5). Mothers delivering preterm had more multiple birth pregnancies, greater pre-pregnancy BMI, and more PBLW, PPB, assisted reproduction, and medical complications.

Maternal Stressful Life Events

The percentage of respondents experiencing stressful events during pregnancy were as follows: illness among family members - 24.0%, divorced - 11.8%, moved – 39.1%, became homeless – 4.0%, partner lost job – 11.1%, mom lost job – 11.4%, argued with partner more than usual – 30.6%, partner did not want the child – 9.8%, inability to pay bills – 27.1%, got in a physical fight – 4.7%, partner went to jail – 5.0%, close friend/relative had a bad problem with drinking or drug use – 11.9%, and close friend/relative died – 17.7%. There were no significant differences in the percentage of preterm infants as compared to full-term infants born to mothers experiencing individual stressors (Table 6). However, when compared to full-term births, the percentage of preterm births was greater among mothers experiencing the majority of the stressful life events; only mothers experiencing a move, job loss by partner, or more frequent arguments with partner during pregnancy gave birth to fewer preterm infants.

Table 5

Preterm vs. Full-Term Differences (n=1647)

	Mean (SD)			Test Statistic			
Variable	Preterm	Full-term	df	t	χ^2	p	Cohen's d
Medical complications	0.86 (0.345)	0.61 (0.488)	1		118.026	<0.001	0.595
Plurality	1.17 (0.394)	1.02 (0.139)	1645	-11.054		<0.001	0.505
Previous preterm birth	0.20 (0.401)	0.08 (0.264)	1		56.648	<0.001	0.370
Previous low birthweight	0.16 (0.369)	0.07 (0.254)	1		35.653	<0.001	0.293
Assisted reproduction	0.07 (0.248)	0.03 (0.160)	1		15.224	<0.001	0.189
Pre-pregnancy BMI	25.80 (6.442)	25.18 (6.248)	1645	-1.943		0.052	0.098
Maternal age	27.48 (6.577)	26.91 (6.361)	1645	-1.746		0.081	0.088
Maternal education	0.84 (0.369)	0.82 (0.388)	1		1.346	0.246	0.059
Income	0.54 (0.499)	0.56 (0.497)	1		0.723	0.395	0.043
Smoking during pregnancy	0.09 (0.289)	0.08 (0.277)	1		0.275	0.600	0.027
Inadequate prenatal care	0.29 (0.455)	0.30 (0.460)	1		0.247	0.619	0.025
Maternal race	0.58 (0.494)	0.58 (0.494)	1		0.004	0.949	0.003

Table 6

Differences in Preterm vs. Full-Term Delivery by Maternal Stressful Life Events (n=1647)

Variable	Mean (SD) Preterm	Full-term	df	χ^2	p	Cohen's d
Partner lost job	0.098 (0.297)	0.119 (0.324)	1	1.767	0.184	0.068
Divorced	0.130 (0.337)	0.111 (0.315)	1	1.296	0.255	0.057
Illness among family members	0.254 (0.435)	0.232 (0.423)	1	0.952	0.329	0.049
Mom lost job	0.124 (0.329)	0.108 (0.311)	1	0.885	0.347	0.047
Inability to pay bills	0.284 (0.451)	0.264 (0.441)	1	0.818	0.366	0.046
Friend/relative had problem w/ drinking	0.127 (0.333)	0.115 (0.319)	1	0.493	0.483	0.035
Became homeless	0.045 (0.207)	0.038 (0.191)	1	0.467	0.494	0.034
Partner did not want child	0.104 (0.306)	0.095 (0.293)	1	0.403	0.525	0.032
Partner went to jail	0.055 (0.227)	0.048 (0.214)	1	0.366	0.545	0.030
Close friend/relative died	0.183 (0.387)	0.174 (0.379)	1	0.223	0.637	0.024
Argued w/ partner more than usual	0.302 (0.459)	0.310 (0.463)	1	0.111	0.739	0.017
Moved	0.387 (0.487)	0.394 (0.489)	1	0.073	0.786	0.014
Got in a physical fight	0.048 (0.214)	0.047 (0.211)	1	0.014	0.906	0.006

Urogenital Infection

The frequency of infection diagnosed among the respondents during pregnancy were as follows: warts - 0.6%, herpes – 1.6%, chlamydia – 3.1%, gonorrhea - 0.7%, PID – 0.3%, syphilis – 0.1%, Group B strep – 6.5%, vaginosis – 3.6%, trich - 0.9%, yeast infection – 9.4%, UTI – 19.9%, and other infection – 1.6%. Significant differences were noted in gestation periods of mothers with Group B strep during pregnancy (Table 7). The percentage of preterm births was significantly lower than that of full-term births among mothers diagnosed with Group B strep during pregnancy (p = 0.031, d = 0.113). No significant differences were noted with the other urogenital infections.

Maternal Stressful Life Events and Urogenital Infection

The relationship between maternal stressful life events and urogenital infection in preterm birth was analyzed using maximum likelihood estimation theory of structural equation modeling. The model fit was assessed using the chi-square statistic (χ^2), root mean square error of approximation (RMSEA), as well as the goodness-of-fit (GFI), comparative fit (CFI), and normed fit (NFI) indices, as model fit should be simultaneously evaluated from the perspective of several fit statistics (Stapleton, 1997). Smaller χ^2 and RMSEA values are indicative of good model fit (Stevens, 2002; Thompson, 2004). *Acceptable* fit for RMSEA is less than 0.08 (Sun, 2005), while *reasonable* fit is less than 0.06 (Sun, 2005; Thompson, 2004). Alternatively, larger values are indicative of good fit for GFI, CFI, and NFI; the closer the value to 1.00, the better the fit of

Table 7

Differences in Preterm vs. Full-Term Delivery by Urogenital Infection (n=1647)

Variable	Mean (SD) Preterm	Full-term	df	χ^2	p	Cohen's d
Group B streptococcus	0.048 (0.214)	0.075 (0.264)	1	4.663	0.031	0.113
Urinary tract infection	0.213 (0.410)	0.191 (0.394)	1	1.181	0.277	0.055
Herpes	0.013 (0.113)	0.019 (0.135)	1	0.784	0.376	0.046
Other infection	0.013 (0.113)	0.019 (0.135)	1	0.784	0.376	0.046
Pelvic inflammatory disease	0.002 (0.040)	0.004 (0.062)	1	0.678	0.410	0.044
Chlamydia	0.035 (0.185)	0.029 (0.169)	1	0.459	0.498	0.034
Bacterial vaginosis	0.039 (0.193)	0.035 (0.184)	1	0.125	0.724	0.018
Gonorrhea	0.008 (0.089)	0.007 (0.082)	1	0.076	0.783	0.014
Syphilis	0.002 (0.040)	0.002 (0.044)	1	0.026	0.872	0.008
Genital warts	0.006 (0.080)	0.006 (0.076)	1	0.020	0.887	0.007
Trichomoniasis	0.010 (0.098)	0.010 (0.098)	1	0.001	0.978	0.001
Yeast infection	0.095 (0.293)	0.095 (0.293)	1	<0.001	0.999	<0.001

the model to the data (Stapleton, 1997; Stevens, 2002; Thompson, 2004). Regarding GFI, an acceptable fit is a value that exceeds 0.90 (Roberts, 1999); however, 0.95 indicates a *good* fit (Sun, 2005). Similarly with CFI and NFI, values of 0.95 or greater indicate reasonable fit (Thompson, 2004).

Latent constructs

Modeling of the maternal stress latent construct revealed that all stressful life events were significant predictors of maternal stress in this sample (Table 8). Getting into a physical fight ($\beta = 0.490$, $p < 0.001$) and arguing with partner more than usual ($\beta = 0.482$, $p < 0.001$) were the strongest predictors, while illness among family members ($\beta = 0.186$, $p < 0.001$) and death of close friends/relatives ($\beta = 0.180$, $p < 0.001$) were the weakest predictors of maternal stress. The fit of this construct to the data was less than acceptable-to-good: $\chi^2(65, N = 1647) = 434.71$, $p < 0.001$; GFI = 0.960, CFI = 0.812, NFI = 0.787; RMSEA = 0.059. Consultation of the modification indexes indicated that correlation of the error variances for illness among family members and death of close friends/relatives, as well as divorce and partner did not want child, could improve the overall fit. Refitting the construct with these correlations resulted in an improved overall fit: $\chi^2(63, N = 1647) = 236.39$, $p < 0.001$; GFI = 0.978, CFI = 0.912, NFI = 0.884; RMSEA = 0.041.

Modeling of the infection latent construct revealed that all urogenital infections except those classified as "other" were significant predictors of infection (Table 9). Gonorrhea ($\beta = 0.463$, $p < 0.001$) and syphilis ($\beta = 0.401$,

Table 8

Predictors of Maternal Stress

Stressful Life Event	B	S.E.	C.R.	β	p
Got in a physical fight	0.104	0.006	17.703	0.49	<0.001
Argued w/ partner more than usual	0.222	0.013	17.389	0.482	<0.001
Divorced	0.151	0.009	16.775	0.467	<0.001
Inability to pay bills	0.208	0.012	16.789	0.467	<0.001
Partner did not want child	0.124	0.008	14.85	0.417	<0.001
Partner lost job	0.129	0.009	14.617	0.411	<0.001
Friend/relative had problem w/ drinking	0.132	0.009	14.491	0.408	<0.001
Became homeless	0.078	0.006	14.096	0.397	<0.001
Partner went to jail	0.083	0.006	13.467	0.381	<0.001
Mom lost job	0.110	0.009	12.178	0.346	<0.001
Moved	0.155	0.014	11.115	0.317	<0.001
Illness among family members	0.079	0.012	6.403	0.186	<0.001
Close friend/relative died	0.069	0.011	6.221	0.180	<0.001

Table 9

Predictors of Urogenital Infection

Urogenital Infection	B	S.E.	C.R.	β	p
Gonorrhea	0.039	0.003	13.096	0.463	<0.001
Syphilis	0.017	0.001	11.537	0.401	<0.001
Chlamydia	0.061	0.006	10.128	0.349	<0.001
Pelvic inflammatory disease	0.019	0.002	9.906	0.341	<0.001
Yeast infection	0.086	0.010	8.531	0.293	<0.001
Genital warts	0.022	0.003	8.365	0.287	<0.001
Bacterial vaginosis	0.042	0.006	6.549	0.224	<0.001
Herpes	0.026	0.004	6.006	0.205	<0.001
Urinary tract infection	0.070	0.014	5.129	0.175	<0.001
Group B streptococcus	0.039	0.008	4.604	0.157	<0.001
Trichomoniasis	0.008	0.003	2.482	0.085	0.017
Other infection	-0.001	0.004	-0.165	-0.006	0.869

$p < 0.001$) were the strongest predictors of urogenital infection. While trichomoniasis ($\beta = 0.085$, $p = 0.017$) and "other" infection ($\beta = -0.006$, $p = 0.869$) were the weakest predictors of urogenital infection. The fit of this construct to the data was less than acceptable-to-good: $\chi^2(54, N = 1647) = 302.302$, $p < 0.001$; GFI = 0.969, CFI = 0.637, NFI = 0.597; RMSEA = 0.053. Consultation of the modification indexes indicated that correlation of the error variances for chlamydia and gonorrhea, as well as yeast infection and urinary tract infection, could improve the overall fit. Refitting the construct with these correlations resulted in an improved overall fit: $\chi^2(52, N = 1647) = 140.845$, $p < 0.01$; GFI = 0.986, CFI = 0.870, NFI = 0.812; RMSEA = 0.032.

Measurement model

The measurement model consisted of: maternal stress, a latent construct measured by 13 stressful life events, and infection, a latent construct measured by 12 self-reported urogenital infections. This measurement model revealed that the indicators were associated with the appropriate constructs (Table 10). The fit of this model to the data was less than acceptable-to-good: $\chi^2(270, N = 1647) = 701.35$, $p < 0.01$; GFI = 0.966, CFI = 0.848, NFI = 0.776; RMSEA = 0.031.

Structural equation models involving preterm birth

Following satisfactory fitting of the measurement model, preterm birth was introduced as the dependent variable in the model. This structural model tested the direct and indirect relationships between maternal stress, infection, and preterm birth. The fit of this model was less than acceptable-to-good:

Table 10

Exploratory Factor Analysis for Measurement Model

Variable	Maternal Stress Pattern	r_s	Urogenital Infection Pattern	r_s
Got in a physical fight	0.495	0.495	0	0.076
Argued w/ partner more than usual	0.483	0.483	0	0.074
Inability to pay bills	0.471	0.471	0	0.072
Divorced	0.441	0.441	0	0.067
Partner lost job	0.419	0.419	0	0.064
Became homeless	0.407	0.407	0	0.062
Friend/relative had problem w/ drinking	0.406	0.406	0	0.062
Partner did not want child	0.382	0.382	0	0.058
Partner went to jail	0.381	0.381	0	0.058
Mom lost job	0.353	0.353	0	0.054
Moved	0.325	0.325	0	0.05
Illness among family members	0.165	0.165	0	0.025
Close friend/relative died	0.157	0.157	0	0.024
Syphilis	0	0.072	0.469	0.469
Pelvic inflammatory disease	0	0.063	0.415	0.415
Gonorrhea	0	0.051	0.334	0.334
Genital warts	0	0.05	0.325	0.325
Yeast infection	0	0.038	0.249	0.249
Herpes	0	0.037	0.242	0.242
Bacterial vaginosis	0	0.035	0.23	0.23
Chlamydia	0	0.031	0.205	0.205
Group B streptococcus	0	0.022	0.146	0.146
Urinary tract infection	0	0.02	0.133	0.133
Trichomoniasis	0	0.007	0.043	0.043
Other infection	0	0.001	0.009	0.009

$\chi^2(293, N = 1647) = 716.968$, $p < 0.001$; GFI = 0.967, CFI = 0.850, NFI = 0.772; RMSEA = 0.030. The path from maternal stress to infection was significant (Table 11; $\beta = 0.153$, $p < 0.001$); however, neither the path from maternal stress to preterm birth, nor the path from urogenital infection to preterm birth was significant.

In the next step, all aforementioned covariates were introduced to the model with direct effects to preterm birth. The fit of this model was less than acceptable: $\chi^2(623, N = 1647) = 3224.213$, $p < 0.001$; GFI = 0.894, CFI = 0.507, NFI = 0.457; RMSEA = 0.050. The path from maternal stress to infection remained significant ($p < 0.001$), while neither the path from maternal stress to preterm birth nor the path from infection to preterm birth became significant. Examination of the standardized residuals indicated that removal of four covariates, maternal age, smoking, maternal education, and income, from the model may improve the overall fit. Refitting the structural model without these covariates resulted in an improved overall model fit: $\chi^2(489, N = 1647) = 1928.388$, $p < 0.001$; GFI = 0.935, CFI = 0.650, NFI = 0.585; RMSEA = 0.042.

Consultation of the modification indexes indicated that correlation of the plurality and assisted reproduction covariates, as well as the previous preterm birth and previous low birth weight covariates, could also improve the overall fit. Refitting the model with these correlations (Figure 1) resulted in an improved overall fit: $\chi^2(487, N = 1647) = 1192.686$, $p < 0.001$; GFI = 0.956, CFI = 0.829,

Table 11

Stress and Infection on Preterm Birth

Path	B	S.E.	C.R.	B	P
Maternal stress → Preterm birth	0.011	0.015	0.780	0.024	0.435
Infection → Preterm birth	-0.010	0.017	-0.571	-0.020	0.568
Maternal stress ↔ Infection	0.153	0.042	3.677	0.153	<0.001
Maternal stress → Got in a physical fight	0.105	0.006	17.741	0.495	<0.001
Maternal stress → Argued w/ partner more than usual	0.223	0.013	17.281	0.483	<0.001
Maternal stress → Inability to pay bills	0.209	0.012	16.807	0.471	<0.001
Maternal stress → Divorced	0.143	0.009	15.517	0.441	<0.001
Maternal stress → Partner lost job	0.132	0.009	14.809	0.419	<0.001
Maternal stress → Became homeless	0.080	0.006	14.371	0.407	<0.001
Maternal stress → Friend/relative had problem w/ drinking	0.132	0.009	14.335	0.406	<0.001
Maternal stress → Partner did not want child	0.114	0.009	13.233	0.382	<0.001
Maternal stress → Partner went to jail	0.083	0.006	13.404	0.382	<0.001
Maternal stress → Mom lost job	0.112	0.009	12.368	0.354	<0.001
Maternal stress → Moved	0.158	0.014	11.298	0.324	<0.001
Maternal stress → Illness among family members	0.071	0.012	5.646	0.165	<0.001
Maternal stress → Close friend/relative died	0.060	0.011	5.369	0.157	<0.001
Infection → Syphilis	0.020	0.002	12.571	0.469	<0.001
Infection → Pelvic inflammatory disease	0.023	0.002	11.400	0.415	<0.001
Infection → Gonorrhea	0.028	0.003	9.325	0.334	<0.001
Infection → Genital warts	0.025	0.003	9.139	0.325	<0.001
Infection → Yeast Infection	0.073	0.010	7.060	0.249	<0.001
Infection → Herpes	0.031	0.004	6.875	0.242	<0.001
Infection → Bacterial vaginosis	0.043	0.007	6.517	0.229	<0.001
Infection → Chlamydia	0.036	0.006	5.649	0.205	<0.001

Table 11. Continued

Path	B	S.E.	C.R.	B	P
Infection → Group B streptococcus	0.036	0.009	4.184	0.147	<0.001
Infection → Urinary tract infection	0.053	0.014	3.718	0.132	<0.001
Infection → Trichomoniasis	0.004	0.003	1.216	0.043	0.224
Infection → Other infection	0.001	0.004	0.262	0.009	0.793

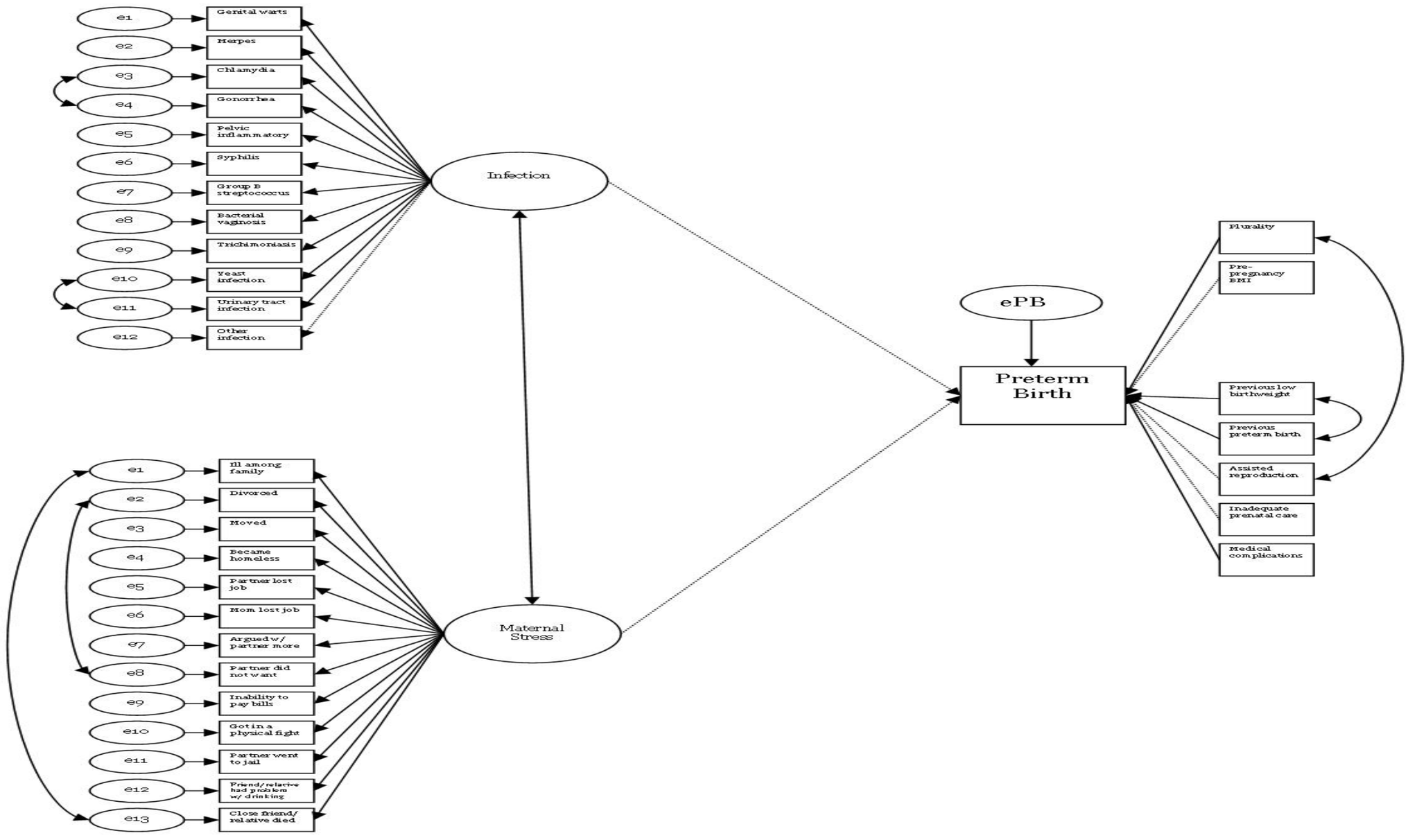

Figure 1. Maternal Stress and Infection Model. Significant predictors are depicted by solid arrows. Non-significant predictors are depicted by dashed arrows.

NFI = 0.743; RMSEA = 0.030. In this final model, the path from maternal stress to infection remained significant (Table 12; β = 0.153, $p < 0.001$), while neither the path from maternal stress to preterm birth nor the path from urogenital infection to preterm birth was significant.

In order to examine the interaction effects of maternal stress and infection on preterm birth, the interaction variable (constructed by multiplying the two constructs) was introduced to a model derived from the aforementioned final model (Figure 2). In this interaction model, the maternal stress and infection constructs were summations of the respective stressful life events and urogenital infections. The fit of this model to the data was less than acceptable-to-good: $\chi^2(40, N = 1647) = 221.93$, $p < 0.001$; GFI = 0.976, CFI = 0.866, NFI = 0.842; RMSEA = 0.053. The interaction of maternal stress and infection did not significantly predict preterm birth (Table 13; β = 0.022, $p = 0.357$). However, the interaction effect was significantly associated with maternal stress (β = 0.155, $p < 0.001$) and infection (β = 0.21, $p < 0.001$). Additionally, the paths from maternal stress to infection (β = 0.192, $p < 0.001$) and infection to preterm birth (β = -0.043, $p = 0.07$) were significant.

Discussion

The purpose of this study was to explore maternal stressful life events and urogenital infection in preterm birth. Specifically, the relationship between maternal stress and infection and the multiplicative effect of the interaction

Table 12

Stress, Infection, and Covariates on Preterm Birth

Path	B	S.E.	C.R.	B	p
Maternal stress → Preterm birth	0.001	0.013	0.112	0.003	0.911
Infection → Preterm birth	-0.006	0.016	-0.365	-0.012	0.715
Maternal stress ↔ Infection	0.153	0.042	3.687	0.153	<0.001
Maternal stress → Got in a physical fight	0.105	0.006	17.742	0.495	<0.001
Maternal stress → Argued w/ partner more than usual	0.223	0.013	17.289	0.483	<0.001
Maternal stress → Inability to pay bills	0.209	0.012	16.801	0.471	<0.001
Maternal stress → Divorced	0.143	0.009	15.509	0.441	<0.001
Maternal stress → Partner lost job	0.132	0.009	14.824	0.419	<0.001
Maternal stress → Became homeless	0.080	0.006	14.368	0.407	<0.001
Maternal stress → Friend/relative had problem w/ drinking	0.132	0.009	14.331	0.406	<0.001
Maternal stress → Partner did not want child	0.114	0.009	13.229	0.382	<0.001
Maternal stress → Partner went to jail	0.083	0.006	13.400	0.381	<0.001
Maternal stress → Mom lost job	0.112	0.009	12.362	0.353	<0.001
Maternal stress → Moved	0.158	0.014	11.303	0.325	<0.001
Maternal stress → Illness among family members	0.070	0.012	5.639	0.165	<0.001
Maternal stress → Close friend/relative died	0.060	0.011	5.366	0.157	<0.001
Infection → Syphilis	0.020	0.002	12.559	0.469	<0.001
Infection → Pelvic inflammatory disease	0.023	0.002	11.390	0.415	<0.001
Infection → Gonorrhea	0.028	0.003	9.328	0.334	<0.001
Infection → Genital warts	0.025	0.003	9.136	0.325	<0.001
Infection → Yeast Infection	0.073	0.010	7.072	0.250	<0.001
Infection → Herpes	0.031	0.004	6.872	0.242	<0.001
Infection → Bacterial vaginosis	0.043	0.007	6.526	0.230	<0.001
Infection → Chlamydia	0.036	0.006	5.654	0.205	<0.001

Table 12. Continued

Path	B	S.E.	C.R.	B	P
Infection → Group B streptococcus	0.036	0.009	4.171	0.146	<0.001
Infection → Urinary tract infection	0.053	0.014	3.753	0.134	<0.001
Infection → Trichomoniasis	0.004	0.003	1.217	0.043	0.223
Infection → Other infection	0.001	0.004	0.258	0.009	0.797
Pluralilty → Preterm birth	0.417	0.041	10.146	0.239	<0.001
Previous preterm birth → Preterm birth	0.193	0.040	4.781	0.132	<0.001
Medical complications → Preterm birth	0.240	0.024	9.979	0.227	<0.001
Previous low birthweight → Preterm birth	0.085	0.043	1.964	0.054	0.050
Assisted reproduction → Preterm birth	0.100	0.057	1.762	0.042	0.078
Pre-pregnancy BMI → Preterm birth	0.002	0.002	1.407	0.032	0.159
Inadequate prenatal care → Preterm birth	<0.001	0.024	0.004	<0.001	0.997
Previous low birthweight ↔ Previous preterm birth	0.056	0.003	19.885	0.562	<0.001
Assisted reproduction ↔ Pluralilty	0.014	0.001	10.003	0.254	<0.001

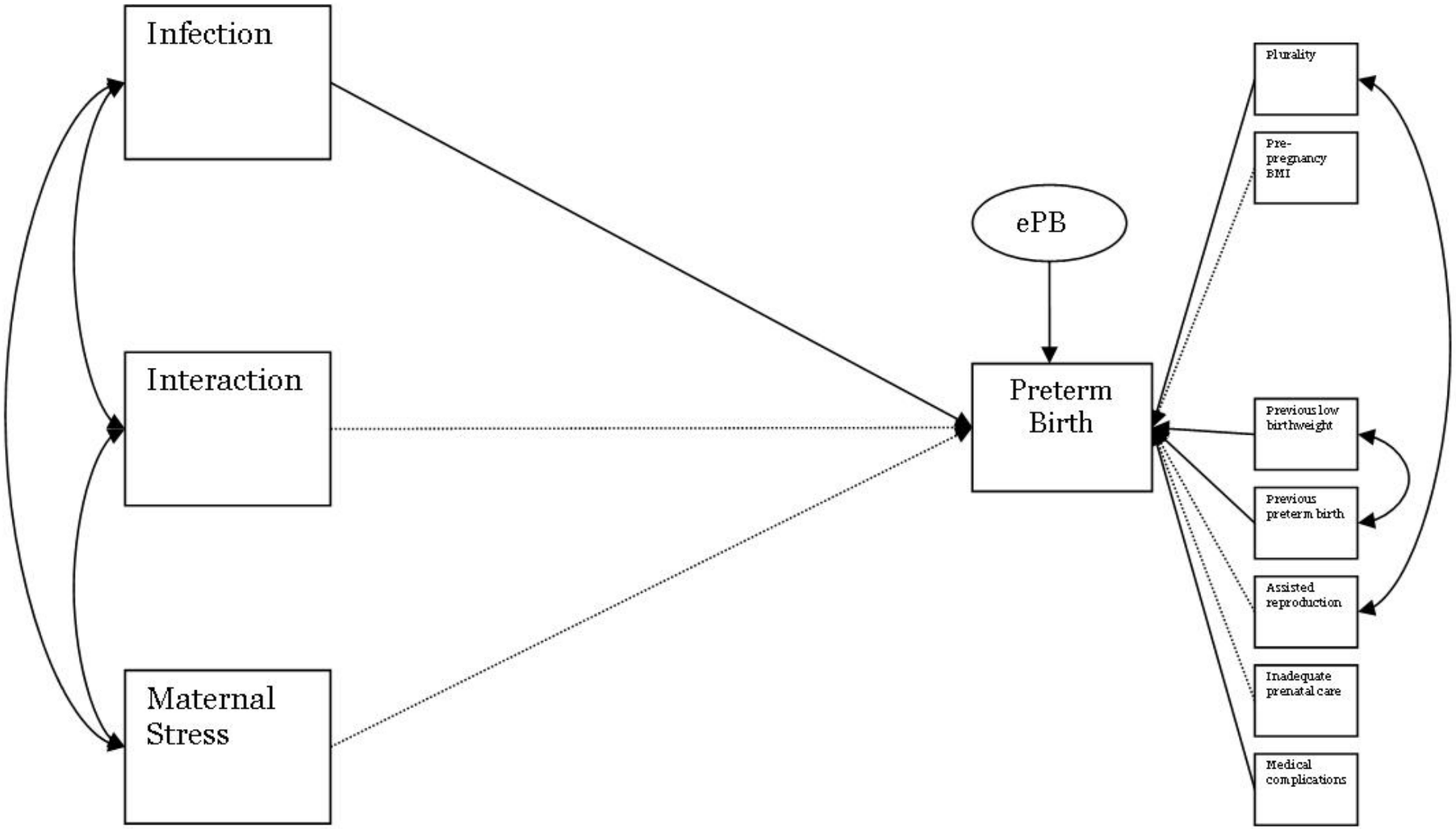

Figure 2. Interaction Model. Significant predictors are depicted by solid arrows. Non-significant predictors are depicted by dashed arrows.

Table 13

Direct and Interaction Effects on Preterm Birth

Path	B	S.E.	C.R.	β	p
Interaction → Preterm birth	0.005	0.006	0.922	0.022	0.357
Maternal stress → Preterm birth	0.002	0.006	0.286	0.007	0.775
Infection → Preterm birth	-0.026	0.014	-1.812	-0.043	0.070
Interaction ↔ Maternal stress	0.599	0.096	6.209	0.155	<0.001
Interaction ↔ Infection	0.320	0.038	8.349	0.210	<0.001
Maternal stress ↔ Infection	0.314	0.041	7.648	0.192	<0.001
Pre-pregnancy BMI → Preterm birth	0.003	0.002	1.505	0.034	0.132
Previous low birthweight → Preterm birth	0.083	0.043	1.920	0.053	0.055
Previous preterm birth → Preterm birth	0.193	0.040	4.792	0.132	<0.001
Assisted reproduction → Preterm birth	0.100	0.057	1.753	0.041	0.080
Inadequate prenatal care → Preterm birth	0.002	0.024	0.098	0.002	0.922
Medical complications → Preterm birth	0.246	0.024	10.25	0.233	<0.001
Plurality → Preterm birth	0.416	0.041	10.117	0.238	<0.001
Previous preterm birth ↔ Previous low birthweight	0.056	0.003	19.885	0.562	<0.001
Plurality ↔ Assisted reproduction	0.014	0.001	10.003	0.254	<0.001

between the two in preterm birth was investigated in order to gain a better understanding of the causes and effects of multiple risk factors on preterm birth. To the author's knowledge, this is the first study to examine maternal stress, infection, and their interactions using a biobehavioral model.

Stressful life events are prevalent in nearly all pregnancies; however, some are more common than others. In the current study, moving, arguing with partner more than usual, and inability to pay bills were the three most commonly reported events. These findings concur with a 2003 assessment of stressful life events in preterm birth, in which Moutquin revealed partner abuse or family disruption, a partner who is regularly absent, and financial insecurity due to job loss, transfer or no money at all as the three most commonly reported events (Moutquin, 2003b). The current study's findings appear to suggest that prevention efforts should be focused on mothers who move, argue with their partner, or are unable to pay their bills; however, there were no significant differences in the number of preterm versus full-term infants born to mothers experiencing these life events. Actually, it is surprising to note that mothers experiencing these events during pregnancy gave birth to fewer preterm infants than full-term infants. When the maternal stress construct was consulted for further insight, arguing with partner more than usual and inability to pay bills were two of the strongest predictors of maternal stress; however, moving was not. These findings suggest that prevention programs should be based on the predictive power rather than the prevalence of risk factors.

Similar to prior research, this study revealed that women experiencing increased levels of stress during pregnancy were at increased risk for preterm birth, even after controlling for sociodemographic and behavioral factors (Bennett & Botti, 1989; Dole et al., 2003; Hobel & Culhane, 2003; Lu & Chen, 2004; Ruiz, Fullerton, & Dudley, 2003). Dole and colleagues (2003) found that women with the highest number of negative life events had the highest risk for preterm birth (Dole et al., 2003). In that same year, Hobel and Culhane reported that expectant mothers experiencing increased levels of psychological or social stress were at a one-and-a-half to two-fold increased risk for preterm birth (Hobel & Culhane, 2003). Additionally, Ruiz and colleagues (2003) reported that a decrease in stress level during pregnancy had a strong positive correlation with gestational age at birth (Ruiz et al., 2003). In the current study, preterm birth had a positive and linear association with the number of stressful life events experienced during the mother's pregnancy. This finding suggests that mothers experiencing multiple stressors are at greater need for prevention programs that include counseling and coping resources.

With regards to urogenital infection, this study revealed that urinary tract and yeast infections were the most common infections in the sample; however, there was no increased risk for preterm birth among mothers diagnosed with these infections during pregnancy. Furthermore, there was a lack of strong predictive power between these infections and their respective construct. Although these findings should be interpreted with caution since the reliability

of this construct was low, they may indicate that mothers are unwilling to be tested for urogenital infection during pregnancy or they are unwillingly to disclose diagnosis, even after pregnancy. Research findings regarding the treatment of urogenital infections during pregnancy in preventing preterm birth are inconclusive (Goldenberg, Culhane, & Johnson, 2005; King & Flenady, 2002; Pararas, Skevaki, & Kafetzis, 2006), indicating that prevention efforts may need to highlight the importance of diagnosis and treatment as a protective factor against birth defects and infant mortality, which may indirectly improve preterm birth rates.

Previous efforts to simultaneously examine maternal stress and infection as risk factors for preterm birth have only examined the relationship in pregnancy, not pregnancy outcome. However, the findings of these studies are noteworthy because they revealed positive relationships between maternal stress and urogenital infection (Culhane et al., 2002; Culhane et al., 2001; Harville et al., 2007; Nelson et al., 2008). Specifically, Harville et al. (2007) found a positive relationship between maternal life experiences and bacterial vaginosis. Findings of the current study revealed a positive association between maternal stress and infection thus increasing the reliability of this finding. Hence, simultaneous stress assessments and infection screenings during pregnancy might improve preterm birth and pregnancy outcomes among expectant mothers. According to Wadhwa's biobehavioral model, the interaction between stress and infection yields a multiplicative effect on preterm birth. The small, yet

multiplicative effect on preterm birth observed in the current study concurs with Wadhwa's theory and highlights the need for prevention efforts that help women cope with multiple risk factors simultaneously.

It is important to note that maternal stress was strongly associated with infection even after controlling for covariates. Maternal age, maternal education, smoking, and income have often assumed significant roles in single risk factor research on preterm birth; however, in this sample, the overall fit of the model was improved upon removal of these covariates. This finding suggests that prevention efforts incorporating multiple risk factors must move beyond sociodemographic variables and investigate variables that are biomedical in nature. This suggestion is further strengthened by several findings from the current study. First, mothers who received assisted reproduction were at increased risk for preterm birth. Secondly, mothers who had previously given birth to low birthweight and/or preterm infants, as well as mothers with higher than normal pre-pregnancy BMIs, were at greater risk for preterm birth. Lastly, mothers who experienced medical complications during pregnancy and mothers who had multiple birth pregnancies were at increased risk for preterm birth. These findings confirm the importance of plurality, pre-pregnancy body mass index, previous low birthweight, previous preterm birth, assisted reproduction, and medical complications in the final biobehavioral model.

Lack of statistical significance of the effect of the two independent variables, maternal stress and infection, on preterm birth should not attenuate

the importance of the study's findings. Given that preterm birth is still poorly understood and prevention efforts have not yielded noteworthy results (Buekens & Klebanoff, 2001; IOM, 2006), the constraints related to statistical significance may not be appropriate for studies that simultaneously investigate risk factors of preterm birth. Prevention specialists may need to look beyond statistical significance in order to design effective programs, especially those that will focus on multiple risk factors. Considering that more than 400,000 infants were born preterm in the United States in year 2004, the practical significance of preventing preterm birth should be of concern, as prevention of this public health problem also provides a vehicle to reduce low birthweight, birth defects, infant mortality, and childhood morbidity and disability rates.

This study is not without limitations. Although the sample size is large, it is restricted to mothers from Florida. Also, the stressful life events and urogenital infections represented in the study are not exhaustive. As with most survey research, a degree of response bias is present in this study; non-responders were young, single, non-White, and less educated. Additionally, the study is based on self-reported data that is subject to role selection and recall biases and the findings represent analyses of unweighted data collected via stratified random sampling. Lastly, this study did not explore racial and ethnic differences in individual stressors or infections nor did the study explore the appropriateness of the tested models by racial and ethnic groups. However, the current findings provide insight for future studies analyzing the relationship

between maternal stressful life events and urogenital infection. Future studies should employ appropriate sample weights in order to improve generalizablity and examine racial and ethnic disparities.

In conclusion, the study's findings have implications for targeted prevention programs to improve preterm birth rates. These programs must recruit individuals who will benefit most from its efforts and engage those less likely to take advantage of those efforts. Prevention programs must address multiple risk factors and consider biomedical confounders. Additionally, the programs must advocate simultaneous stress assessments and infection screenings during pregnancy and provide counseling and coping resources. These prevention efforts must also increase awareness of infection treatment as a protective factor against adverse birth outcomes.

CHAPTER IV

UNCOVERING RACIAL AND ETHNIC DIFFERENCES IN PRETERM BIRTH: A BIOBEHAVIORAL APPROACH

Preterm birth refers to the delivery of an infant before 37 weeks of gestation (USDHHS, 2000). In the United States, preterm birth rates have increased by 30 percent in the last three decades (IOM, 2006). Despite the Healthy People 2010 recommendations that the rate of preterm birth be reduced to 7.6 percent by the end of the 2010 calendar year (USDHHS, 2000), an increasing number of American women are delivering preterm infants due to a myriad of factors (Wadhwa et al., 2001).

Previous epidemiological studies identified the following as risk factors for preterm birth: previous low birthweight or preterm delivery, in-vitro fertilization, multiple birth, medical complications, urogenital infection, low socioeconomic status, smoking, low pre-pregnancy body mass index, maternal age, drug use/abuse, psychosocial stress or stressful life events, and inadequate or no prenatal care (Moutquin, 2003b). Additionally, African American ethnicity has been the explored as a risk factor for preterm birth in a number of research studies (Fiscella, 1996, 2004; Lu & Chen, 2004; Lu & Halfon, 2003; Moutquin, 2003a, 2003b; Patel, Steer, Doyle, Little, & Elliott, 2004; Steer, 2005). Patel and colleagues (2004) identified a median gestational age of 39 completed weeks at delivery in Black babies, as compared with 40 completed

weeks in Whites. However, their finding does not explain excessive preterm birth rates among Black women (Moutquin, 2003b). Even after controlling for the effects of the sociodemographic, access-to-care, and behavioral factors, Black women display a nearly two-fold increased risk for preterm birth over White women (Lu & Halfon, 2003). Furthermore, Black women have higher rates of various types of very preterm birth (Fiscella, 2004). Despite significant research in this area, the unyielding problem of racial/ethnic disparities in preterm birth still exists and the cause of these disparities is yet to be identified.

It is highly implausible that a single risk factor can explain these disparities (Fiscella, 1996). Lu and Halfon (2003) identified stress and infection as promising explanations. To date, very few studies have examined the nature of the stress-infection-immune relationship in pregnancy and associated racial/ethnic disparities (Hobel & Culhane, 2003). However, Culhane and colleagues (2002) investigated racial/ethnic disparities in psychosocial links of maternal stress and bacterial vaginosis (Culhane, Rauh, McCollum, Elo, & Hogan, 2002). Although the study established racial/ethnic disparities in the relationship between maternal stress and urogenital infection during pregnancy, the implications for pregnancy outcome were not assessed.

The phenomenon of preterm birth and its associated racial/ethnic disparities is of interest to numerous governmental agencies and organizations. Current recommendations from the Institute of Medicine (2006) *Committee on Understanding Premature Birth and Assuring Healthy Outcomes* highlight the

need for improved research including investigations of the etiologies of preterm birth, studies of the multiple factors associated with preterm birth, and investigations of racial/ethnic and socioeconomic disparities in preterm birth (IOM, 2006).

Thus, the purpose of this study is to examine the racial/ethnic disparities in the relationship between maternal stressful life events and urogenital infection in preterm birth. Wadhwa's Biobehavioural Model of Stress, Infection, and Preterm Birth (2001) provided theoretical guidance for this investigation. The primary research question was: does maternal stress, infection, and associated interactions predict racial/ethnic disparities in preterm birth?

Methods

Study Sample

The 2005 Florida Pregnancy Risk Assessment Monitoring System (PRAMS) data was utilized to address the aforementioned research questions. PRAMS is a surveillance project of the Centers for Disease Control and Prevention (CDC) and state health agencies (USDHHS, 2007). PRAMS was initiated in 1987 with the goal of improving the health of mothers and infants by reducing adverse birth outcomes such as low birthweight, infant mortality and morbidity, and maternal morbidity. The state of Florida is one of 37 PRAMS participating states (USDHHS, 2007). The state's Department of Health (DOH) Office of Vital statistics, DOH Bureau of Epidemiology, and county health departments assist with this joint effort (Florida DOH, 2007). Annually, PRAMS

collects state-specific, population-based data on maternal attitudes and experiences before, during, and shortly after pregnancy. PRAMS data is used for planning and assessing health programs and for describing maternal experiences that may influence maternal and infant health (USDHHS, 2007).

Participants for the 2005 Florida PRAMS survey included 2,785 mothers who gave birth to a live infant in 2005 and were selected from all births recorded by the Florida Office of Vital Statistics, using a stratified random sampling strategy based on maternal race, age, and infant birth weight (Florida DOH, 2007):

Stratum 1: White/low birth weight/age greater than or equal to 20 years old

Stratum 2: Black/low birth weight/age greater than or equal to 20 years old

Stratum 3: Low birth weight/age less than 20 years old

Stratum 4: White/normal birth weight/age greater than or equal to 20 years old

Stratum 5: Black/normal birth weight/age greater than or equal to 20 years old

Stratum 6: Normal birth weight/age less than 20 years old

The 2005 Florida PRAMS data was primarily collected via mail. Secondary data collection was conducted via telephone interview with non-responders,

approximately one month after the initial survey mail-out (Florida DOH, 2007). Participants responded to the survey two to five months post-delivery.

Measures

Dependent variable

Preterm birth. Preterm birth was assessed based on PRAMS-linked birth certificate data. A response less than 37 weeks of gestation was treated as preterm delivery. The variable was dichotomized as *yes* versus *no*.

Independent variables

Maternal stress. Self-reported maternal stress was assessed based on 13 stressful life events experienced during the 12 months prior to delivery. The 13 life events that served as indicators for maternal stress were as follows: *illness among family members, divorced, moved, became homeless, partner lost job, mom lost job, argued with partner more than usual, partner did not want the child, inability to pay bills, got in a physical fight, partner went to jail, close friend/relative had a bad problem with drinking or drug use,* and *close friend/relative died*. These indicators were summed to create the *maternal stress* construct (range 0-13), with a higher score indicating greater stress during pregnancy. Cronbach's alpha indicated most of the stressors were dependent upon each other (reliability coefficient was 0.664).

Infection. Infection was assessed based on the mother's self-report of being informed by a doctor, nurse, or health care worker during her pregnancy that she had a urinary tract infection, a sexually transmitted disease, or any

vaginal infection. The mother's specification of diagnosis with any of the 12 urogenital infections (i.e., genital warts [warts], herpes, chlamydia, gonorrhea, pelvic inflammatory disease [PID], syphilis, Group B streptococcus [Group B strep], bacterial vaginosis, trichomoniasis [trich], yeast infection, urinary tract infection [UTI], and other infection) was also considered. An affirmative answer to either question was noted as infection during pregnancy. The 12 indicators were summed to create the *urogenital infection* construct. The range for this construct was 0-12, with a higher score indicating greater exposure to infection during pregnancy. Cronbach's alpha indicated most of the infections were independent of each other (reliability coefficient was 0.360).

Covariates

Maternal age. Age was obtained from PRAMS-linked birth certificate data.

Plurality. Plurality was obtained from PRAMS-linked birth certificate data.

Pre-pregnancy body mass index (BMI). BMI was calculated based on the mother's self-reported pre-pregnancy height (without shoes) and weight.

Smoking during pregnancy. Smoking status of the mother was obtained from PRAMS-linked birth certificate data. This variable was reported as yes versus no.

Maternal race/ethnicity. Race/ethnicity was obtained from PRAMS-linked birth certificate data and reported as White, Black, Chinese, Japanese,

Filipino, Other Asian, Other non-White, Hawaiian, American Indian, or Alaskan Native. Reports of Hispanic ethnicity were also considered. All mothers who indicated they were of Hispanic ethnicity were categorized as Hispanics, while the Chinese, Japanese, Filipino, and Other Asian groups were collapsed into the Asian group. Hawaiian, American Indian, and Alaskan Natives were added to the Other non-White group. Hence, the final analysis was comprised of five racial/ethnic groups (*White, Black, Asian, Other non-White,* and *Hispanic*).

Maternal education. Educational attainment was reported as an ordinal level variable with the following attributes: 0-8 years, 9-11 years, 12 years, 13-15 years, or ≥ 16 years. However, this variable was dichotomized as *below median* versus *at median and above* for uniform distribution.

Previous low birth weight (PLBW). PLBW was assessed based on the mother's self-report of delivery of an infant weighing less than 5 pounds, 8 ounces just prior to the current delivery. This variable was reported as yes versus no.

Previous preterm birth (PPB). PPB was assessed based on mother's self-report of delivery of an infant more than 3 weeks before its due date just prior to the current delivery. This variable was reported as yes versus no.

Assisted reproduction. Assisted reproduction was measured based on the mother's report of receiving treatment from a doctor, nurse, or other health care worker to aid with conception. Maternal indication of receiving any of the surveyed treatments during the month prior to pregnancy (fertility-enhancing

drugs prescribed by a doctor, artificial insemination, assisted reproductive technology such as in vitro fertilization, or other medical treatment as specified by the mother) was also considered. An affirmative answer to either question was treated as a positive response. This variable was dichotomized as yes versus no.

Inadequate prenatal care. Inadequate prenatal care was assessed by maternal self-report of timing of first visit for prenatal care. A response greater than or equal to 12 weeks (or 3 months) was treated as inadequate care. This variable was dichotomized as yes versus no.

Medical complications. Medical complications was based on mother's report of having medical problems during the pregnancy. Indication of any of the following 12 problems was considered as medical complications: diabetes prior to pregnancy, diabetes during pregnancy, vaginal bleeding, kidney infection, severe nausea, incompetent cervix, hypertension, problems with placenta, preterm labor pains, premature rupture of membranes, blood transfusion, or an injury sustained from a car accident. This variable was dichotomized as yes versus no.

Income. Total household income (before taxes) during the 12 months prior to delivery was reported in categories of ≤ $10,000, $10,000 - $14,999, $15,000 - $19,999, $20,000 - $24,999, $25,000 - $34,999, $35,000 - $49,999, or ≥ $50,000. However, this variable was dichotomized as below median versus at median and above to normalize the distribution of the variable.

Data Analysis

Prior to analysis, data were cleaned and missing records were handled by listwise deletion using SPSS statistical software. Construct validity for maternal stress and infection was established for this data in prior research. Reliability analysis was conducted to evaluate internal consistency of the data (Huck, 2003).

Descriptive statistics were utilized to describe the sample. Chi-square tests, student *t*-tests, and analysis of variance (ANOVA) were employed to test for significant differences among categorical and interval variables. Structural equation modeling (SEM) was utilized to investigate the relationship, as well as independent and interaction effects of maternal stressful life events and urogenital infection, on preterm birth by racial/ethnic group. Data were analyzed using SPSS 15.0 and AMOS 16.0 structural equation modeling software.

Results

The total number of mothers who were contacted for participation in the 2005 Florida PRAMS was 2785; 73.5% (n = 2047) responded to the survey. Since, not all mothers responded to the survey, the data were analyzed for response bias in demographic variables of interest. This analysis revealed significant differences in the maternal age (p <0.001, d = 0.160), marital status (p <0.001, d = 0.173), maternal race/ethnicity (p < 0.001, η_p^2 = 0.023), and maternal education (p <0.001, d = 0.294) of respondents versus non-

respondents. Respondents were older, married, and had higher educational attainment as compared to non-respondents. Post-hoc analysis revealed that White mothers were significantly more likely to respond as compared to Black and Hispanic mothers. No significant differences were noted between responders and non-responders in the study's outcome variable (preterm birth). Listwise deletion was used to identify respondents with complete data for variables of interest in this study. The final sample was comprised of 1,647 mothers.

The mean age of the mothers within the sample was 27.1 ± 6.44 years. The study sample was 42.0% White, 31.5% Black, 2.2% Asian, 2.5% Other non-White, and 21.9% Hispanic. The median educational attainment and income levels were 12 years and $20,000-$24,999, respectively. Approximately, nine percent of the mothers reported smoking during pregnancy.

Preterm Birth

Overall, 37.8% of the mothers gave birth to preterm infants. The percentage of preterm infants as compared to full-term infants by race/ethnicity were as follows: White – 37.9% vs. 62.1%, Black – 42.7% vs. 57.3%, Asian – 47.2% vs. 52.8%, Other Non-White – 36.6% vs. 63.4%, and Hispanic – 29.9% vs. 70.1%. Significant differences were noted in gestation term by race/ethnicity ($p = 0.003$, $\eta_p^2 = 0.010$). Post-hoc analysis revealed that Black mothers gave birth to significantly more preterm infants than full-term infants as compared to

Hispanic mothers. No other significant racial/ethnic differences were noted by gestation term.

However, significant differences in maternal age, plurality, and BMI in gestation term were noted by racial/ethnic group (Table 14). Black and Asian mothers delivering preterm were older than mothers in their racial group delivering full-term. For the majority of the racial/ethnic groups (White, Black, Other non-White, and Hispanic), mothers giving birth to preterm infants had more multiple births as compared to those giving birth to full-term infants. Among Hispanics, significantly higher BMIs were noted among mothers delivering preterm as compared to mothers delivering full-term.

Additionally, significant differences in previous low birth weight (PLBW), previous preterm birth (PPB), assisted reproduction, and medical complications in gestation term were noted by racial/ethnic group (Table 15). Among White, Black, and Other non-Whites, more preterm infants as compared full-term infants were born to mothers who had previous low birthweight deliveries. White and Black mothers giving birth to preterm infants had more previous preterm deliveries as compared to those giving birth to full-term infants. Whites, Other non-Whites, and Hispanic mothers who used assisted reproductive procedures were at greater risk of delivering preterm as compared to delivering full-term. Among Whites, Blacks, and Hispanics, more preterm

Table 14

Covariates and Preterm Birth by Racial/Ethnic Group

Covariate	Race/Ethnicity	Mean (SD) Preterm	Mean (SD) Full-term	df	t	p	Cohen's d
Maternal age	White	28.187 (6.470)	27.823 (6.635)	689	-0.707	0.480	0.056
	Black	26.575 (6.332)	25.532 (6.019)	516	-1.907	0.057	0.169
	Asian	32.765 (5.772)	29.053 (5.071)	34	-2.054	0.048	0.683
	Other non-White	30.600 (6.653)	30.077 (6.431)	39	-0.248	0.806	0.080
	Hispanic	26.380 (6.796)	26.506 (5.984)	359	0.176	0.860	0.020
Plurality	White	1.198 (0.418)	1.033 (0.191)	689	-7.095	0.000	0.510
	Black	1.122 (0.355)	1.007 (0.082)	516	-5.417	0.000	0.448
	Asian	1.118 (0.332)	1.000 (0.000)	34	-1.547	0.131	0.501
	Other non-White	1.267 (0.458)	1.000 (0.000)	39	-2.999	0.005	0.824
	Hispanic	1.176 (0.406)	1.008 (0.089)	359	-6.249	0.000	0.571

Table 14. Continued

Covariate	Race/Ethnicity	Mean (SD) Preterm	Full-term	df	t	p	Cohen's d
BMI	White	25.230 (6.434)	24.739 (6.312)	689	-0.985	0.325	0.077
	Black	26.825 (6.681)	26.607 (6.790)	516	-0.363	0.717	0.032
	Asian	21.994 (4.174)	23.250 (4.915)	34	0.821	0.418	0.275
	Other non-White	25.488 (6.392)	24.813 (5.309)	39	-0.364	0.718	0.115
	Hispanic	25.740 (5.938)	24.427 (5.342)	359	-2.067	0.039	0.233

Table 15

Dichotomous Covariates and Preterm Birth by Race/Ethnic Group

Covariate	Race/Ethnicity	Mean (SD) Preterm	Full-term	df	χ^2	p	Cohen's d
Previous low birthweight	White	0.149 (0.357)	0.047 (0.211)	1	21.770	<0.001	0.349
	Black	0.195 (0.397)	0.101 (0.302)	1	9.162	0.002	0.265
	Asian	0.176 (0.393)	0.053 (0.229)	1	1.393	0.250	0.385
	Other non-White	0.267 (0.458)	0.038 (0.196)	1	4.626	0.032	0.648
	Hispanic	0.111 (0.316)	0.075 (0.264)	1	1.250	0.265	0.124
Previous preterm birth	White	0.195 (0.397)	0.058 (0.235)	1	30.907	<0.001	0.419
	Black	0.235 (0.425)	0.091 (0.288)	1	20.438	<0.001	0.398
	Asian	0.235 (0.437)	0.053 (0.229)	1	2.503	0.114	0.523
	Other non-White	0.133 (0.352)	0.038 (0.196)	1	1.262	0.261	0.333
	Hispanic	0.148 (0.357)	0.091 (0.288)	1	2.573	0.109	0.176

Table 15. Continued

Covariate	Race/Ethnicity	Mean (SD) Preterm	Full-term	df	χ^2	p	Cohen's d
Assisted reproduction	White	0.099 (0.300)	0.040 (0.195)	1	9.904	0.002	0.236
	Black	0.018 (0.134)	0.010 (0.100)	1	0.608	0.436	0.068
	Asian	0.059 (0.243)	0.105 (0.315)	1	0.253	0.615	0.165
	Other non-White	0.200 (0.414)	0.000 (0.000)	1	5.611	0.018	0.683
	Hispanic	0.065 (0.247)	0.020 (0.139)	1	4.780	0.029	0.224
Medical complications	White	0.874 (0.332)	0.585 (0.493)	1	64.033	<0.001	0.687
	Black	0.896 (0.306)	0.633 (0.483)	1	46.132	<0.001	0.650
	Asian	0.706 (0.470)	0.474 (0.513)	1	1.990	0.158	0.472
	Other non-White	0.800 (0.414)	0.615 (0.496)	1	1.497	0.221	0.404
	Hispanic	0.796 (0.405)	0.636 (0.482)	1	8.960	0.003	0.359

Table 15. Continued

Covariate	Race/Ethnicity	Mean (SD) Preterm	Full-term	df	χ^2	p	Cohen's d
Income	White	0.679 (0.468)	0.716 (0.452)	1	1.020	0.312	0.079
	Black	0.389 (0.489)	0.374 (0.485)	1	0.128	0.721	0.032
	Asian	0.882 (0.332)	0.789 (0.419)	1	0.557	0.455	0.246
	Other non-White	0.733 (0.458)	0.538 (0.508)	1	1.518	0.218	0.403
	Hispanic	0.407 (0.494)	0.490 (0.501)	1	2.081	0.149	0.166
Smoking during pregnancy	White	0.164 (0.371)	0.133 (0.340)	1	1.284	0.257	0.088
	Black	0.050 (0.218)	0.051 (0.219)	1	0.001	0.970	0.003
	Asian	0.059 (0.243)	0.000 (0.000)	1	1.150	0.284	0.343
	Other non-White	0.000 (0.000)	0.192 (0.402)	1	3.285	0.070	0.677
	Hispanic	0.019 (0.135)	0.036 (0.186)	1	0.745	0.388	0.105

Table 15. Continued

Covariate	Race/Ethnicity	Mean (SD) Preterm	Full-term	df	χ^2	p	Cohen's d
Maternal education	White	0.851 (0.357)	0.874 (0.332)	1	0.738	0.390	0.067
	Black	0.842 (0.366)	0.815 (0.389)	1	0.635	0.426	0.071
	Asian	0.941 (0.243)	0.947 (0.229)	1	0.007	0.935	0.026
	Other non-White	0.867 (0.352)	0.769 (0.430)	1	0.575	0.448	0.248
	Hispanic	0.778 (0.418)	0.711 (0.454)	1	1.694	0.193	0.152
Inadequate prenatal care	White	0.214 (0.411)	0.224 (0.417)	1	0.095	0.757	0.024
	Black	0.376 (0.485)	0.397 (0.490)	1	0.252	0.616	0.045
	Asian	0.294 (0.470)	0.368 (0.496)	1	0.223	0.637	0.154
	Other non-White	0.200 (0.414)	0.385 (0.496)	1	1.497	0.221	0.404
	Hispanic	0.324 (0.470)	0.316 (0.466)	1	0.022	0.884	0.017

infants as compared to full-term infants were delivered by mothers whose pregnancies were complicated by medical problems.

Maternal Stressful Life Events

In general, no significant differences in gestation term were noted between mothers who experienced individual stressors during the 12 months prior to delivery and those who did not. However, when the sample was separated by individual stressors and racial/ethnic group, significant differences were noted in the gestation term of mothers experiencing the illness among family members, became homeless, and partner in jail stressful life events (Table 16). Among Other non-Whites, fewer preterm infants as compared to full-term infants were born to mothers who had an ill family member prior to delivery. White mothers faced with homelessness had a higher percentage of preterm deliveries as compared to full-term deliveries. Additionally, White mothers whose partner served jail time had a higher risk for preterm delivery as compared to full-term delivery.

Urogenital Infection

Although significant differences were noted in the gestation term of mothers diagnosed with Group B streptococcus during pregnancy in the overall sample (p = 0.031, d = 0.113), no differences were noted by racial/ethnic group. However, significant racial/ethnic group differences existed in the gestation term of mothers diagnosed with herpes infection during their pregnancy (Table 17). Among Black mothers, fewer preterm infants as compared to full-term

Table 16

Maternal Stress and Preterm Birth by Racial/Ethnic Group

Stressful Life Event	Race/Ethnicity	Mean (SD) Preterm	Full-term	df	χ^2	p	Cohen's d
Illness among family members	White	0.286 (0.453)	0.261 (0.440)	1	0.523	0.470	0.056
	Black	0.244 (0.431)	0.209 (0.407)	1	0.924	0.337	0.085
	Asian	0.118 (0.332)	0.158 (0.375)	1	0.122	0.727	0.114
	Other non-White	0.000 (0.000)	0.346 (0.485)	1	6.653	0.010	1.009
	Hispanic	0.250 (0.435)	0.206 (0.405)	1	0.875	0.349	0.106
Became homeless	White	0.042 (0.201)	0.014 (0.118)	1	5.314	0.021	0.170
	Black	0.059 (0.236)	0.047 (0.212)	1	0.350	0.554	0.052
	Asian	0.000 (0.000)	0.000 (0.000)	1	-	-	-
	Other non-White	0.000 (0.000)	0.000 (0.000)	1	-	-	-
	Hispanic	0.037 (0.190)	0.075 (0.264)	1	1.838	0.175	0.166

Table 16. Continued

Stressful Life Event	Race/Ethnicity	Mean (SD) Preterm	Full-term	df	χ^2	p	Cohen's d
Partner went to jail	White	0.073 (0.260)	0.037 (0.190)	1	4.197	0.041	0.155
	Black	0.041 (0.198)	0.077 (0.268)	1	2.947	0.086	0.156
	Asian	0.000 0.000	0.000 0.000	1	-	-	-
	Other non-White	0.000 (0.000)	0.077 (0.272)	1	1.213	0.271	0.400
	Hispanic	0.056 (0.230)	0.032 (0.175)	1	1.163	0.281	0.117
Divorced	White	0.115 (0.319)	0.075 (0.263)	1	3.172	0.075	0.137
	Black	0.158 (0.366)	0.152 (0.359)	1	0.046	0.831	0.019
	Asian	0.176 (0.393)	0.053 (0.229)	1	1.393	0.238	0.385
	Other non-White	0.000 (0.000)	0.038 (0.196)	1	0.591	0.442	0.277
	Hispanic	0.120 (0.327)	0.138 (0.346)	1	0.212	0.645	0.053

Table 16. Continued

Stressful Life Event	Race/Ethnicity	Mean (SD) Preterm	Full-term	df	χ^2	p	Cohen's d
Moved	White	0.424 (0.495)	0.364 (0.482)	1	2.472	0.116	0.123
	Black	0.385 (0.488)	0.407 (0.492)	1	0.275	0.600	0.047
	Asian	0.353 (0.493)	0.316 (0.478)	1	0.056	0.813	0.077
	Other non-White	0.267 (0.458)	0.538 (0.508)	1	2.853	0.091	0.562
	Hispanic	0.324 (0.470)	0.419 (0.494)	1	2.864	0.091	0.197
Partner lost job	White	0.107 (0.310)	0.110 (0.313)	1	0.012	0.912	0.009
	Black	0.095 (0.294)	0.104 (0.306)	1	0.123	0.726	0.031
	Asian	0.000 (0.000)	0.000 (0.000)	1	-	-	-
	Other non-White	0.000 (0.000)	0.154 (0.368)	1	2.557	0.110	0.591
	Hispanic	0.111 (0.316)	0.158 (0.366)	1	1.356	0.244	0.138

Table 16. Continued

Stressful Life Event	Race/Ethnicity	Mean (SD) Preterm	Full-term	df	χ^2	p	Cohen's d
Mom lost job	White	0.099 (0.300)	0.084 (0.278)	1	0.467	0.494	0.053
	Black	0.154 (0.362)	0.131 (0.338)	1	0.531	0.466	0.064
	Asian	0.000 (0.000)	0.053 (0.229)	1	0.920	0.337	0.324
	Other non-White	0.067 (0.258)	0.115 (0.326)	1	0.256	0.613	0.166
	Hispanic	0.148 (0.357)	0.126 (0.333)	1	0.308	0.579	0.063
Argued w/ partner more than usual	White	0.244 (0.430)	0.238 (0.426)	1	0.038	0.846	0.015
	Black	0.389 (0.489)	0.421 (0.495)	1	0.529	0.467	0.065
	Asian	0.294 (0.470)	0.105 (0.315)	1	2.043	0.153	0.472
	Other non-White	0.133 (0.352)	0.269 (0.452)	1	1.025	0.311	0.335
	Hispanic	0.287 (0.454)	0.320 (0.467)	1	0.388	0.533	0.072

Table 16. Continued

Stressful Life Event	Race/Ethnicity	Mean (SD) Preterm	Full-term	df	χ^2	p	Cohen's d
Partner did not want child	White	0.103 (0.305)	0.065 (0.247)	1	3.170	0.075	0.136
	Black	0.131 (0.338)	0.152 (0.359)	1	0.426	0.514	0.058
	Asian	0.000 (0.000)	0.000 (0.000)	1	-	-	-
	Other non-White	0.000 (0.000)	0.038 (0.196)	1	0.591	0.442	0.277
	Hispanic	0.083 (0.278)	0.091 (0.288)	1	0.054	0.817	0.027
Inability to pay bills	White	0.256 (0.437)	0.226 (0.419)	1	0.788	0.375	0.069
	Black	0.312 (0.464)	0.283 (0.451)	1	0.526	0.468	0.064
	Asian	0.059 (0.243)	0.000 (0.000)	1	1.150	0.284	0.343
	Other non-White	0.200 (0.414)	0.462 (0.508)	1	2.804	0.094	0.564
	Hispanic	0.343 (0.477)	0.304 (0.461)	1	0.512	0.474	0.082

Table 16. Continued

Stressful Life Event	Race/Ethnicity	Mean (SD) Preterm	Full-term	df	χ^2	p	Cohen's d
Got in a physical fight	White	0.053 (0.225)	0.030 (0.172)	1	2.318	0.128	0.115
	Black	0.045 (0.208)	0.074 (0.262)	1	1.817	0.178	0.122
	Asian	0.118 (0.332)	0.000 (0.000)	1	2.367	0.124	0.501
	Other non-White	0.133 (0.352)	0.038 (0.196)	1	1.262	0.261	0.333
	Hispanic	0.019 (0.135)	0.047 (0.213)	1	1.697	0.193	0.162
Friend/relative had problem w/ drinking	White	0.145 (0.353)	0.147 (0.354)	1	0.004	0.948	0.005
	Black	0.109 (0.312)	0.098 (0.297)	1	0.166	0.684	0.036
	Asian	0.059 (0.243)	0.000 (0.000)	1	1.150	0.284	0.343
	Other non-White	0.133 (0.352)	0.154 (0.368)	1	0.032	0.858	0.057
	Hispanic	0.130 (0.337)	0.087 (0.282)	1	1.535	0.215	0.137

Table 16. Continued

Stressful Life Event	Race/Ethnicity	Mean (SD) Preterm	Full-term	df	χ^2	p	Cohen's d
Close friend/relative died	White	0.179 (0.384)	0.175 (0.380)	1	0.023	0.879	0.012
	Black	0.222 (0.416)	0.209 (0.407)	1	0.127	0.722	0.031
	Asian	0.059 (0.243)	0.158 (0.375)	1	0.892	0.345	0.314
	Other non-White	0.133 (0.352)	0.115 (0.326)	1	0.029	0.866	0.053
	Hispanic	0.139 (0.347)	0.138 (0.346)	1	<0.001	0.989	0.002

Table 17

Infection and Preterm Birth by Racial/Ethnic Group

Urogenital Infection	Race/Ethnicity	Mean (SD) Preterm	Full-term	df	χ^2	p	Cohen's d
Herpes	White	0.027 (0.162)	0.019 (0.135)	1	0.499	0.480	0.054
	Black	0.000 (0.000)	0.034 (0.181)	1	7.588	0.006	0.264
	Asian	0.000 (0.000)	0.000 (0.000)	1	-	-	-
	Other non-White	0.000 (0.000)	0.000 (0.000)	1	-	-	-
	Hispanic	0.009 (0.096)	0.004 (0.063)	1	0.387	0.534	0.065
Genital warts	White	0.008 (0.087)	0.005 (0.068)	1	0.250	0.617	0.038
	Black	0.000 (0.000)	0.010 (0.100)	1	2.245	0.134	0.143
	Asian	0.000 (0.000)	0.000 (0.000)	1	-	-	-
	Other non-White	0.000 (0.000)	0.000 (0.000)	1	-	-	-
	Hispanic	0.019 (0.135)	0.004 (0.063)	1	1.949	0.163	0.138

Table 17. Continued

Urogenital Infection	Race/Ethnicity	Mean (SD) Preterm	Full-term	df	χ^2	p	Cohen's d
Chlamydia	White	0.015 (0.123)	0.014 (0.118)	1	0.019	0.891	0.011
	Black	0.068 (0.252)	0.047 (0.212)	1	1.031	0.310	0.089
	Asian	0.000 (0.000)	0.000 (0.000)	1	-	-	-
	Other non-White	0.000 (0.000)	0.038 (0.196)	1	0.591	0.442	0.277
	Hispanic	0.028 (0.165)	0.036 (0.186)	1	0.143	0.705	0.044
Gonorrhea	White	0.004 (0.062)	0.002 (0.048)	1	0.124	0.724	0.027
	Black	0.018 (0.134)	0.020 (0.141)	1	0.030	0.863	0.015
	Asian	0.000 (0.000)	0.000 (0.000)	1	-	-	-
	Other non-White	0.000 (0.000)	0.000 (0.000)	1	-	-	-
	Hispanic	0.000 (0.000)	0.000 (0.000)	1	-	-	-

Table 17. Continued

Urogenital Infection	Race/Ethnicity	Mean (SD) Preterm	Full-term	df	χ^2	p	Cohen's d
Pelvic inflammatory disease	White	0.000 (0.000)	0.000 (0.000)	1	-	-	-
	Black	0.005 (0.067)	0.007 (0.082)	1	0.107	0.743	0.029
	Asian	0.000 (0.000)	0.000 (0.000)	1	-	-	-
	Other non-White	0.000 (0.000)	0.000 (0.000)	1	-	-	-
	Hispanic	0.000 (0.000)	0.008 (0.089)	1	0.859	0.354	0.126
Syphilis	White	0.000 (0.000)	0.002 (0.048)	1	0.610	0.434	0.068
	Black	0.000 (0.000)	0.003 (0.058)	1	0.746	0.388	0.082
	Asian	0.000 (0.000)	0.000 (0.000)	1	-	-	-
	Other non-White	0.067 (0.2580	0.000 (0.000)	1	1.777	0.183	0.365
	Hispanic	0.000 (0.000)	0.000 (0.0000	1	-	-	-

Table 17. Continued

Urogenital Infection	Race/Ethnicity	Mean (SD) Preterm	Full-term	df	χ^2	p	Cohen's d
Group B streptococcus	White	0.073 (0.260)	0.096 (0.294)	1	1.090	0.296	0.083
	Black	0.041 (0.198)	0.071 (0.257)	1	2.088	0.148	0.131
	Asian	0.000 (0.000)	0.158 (0.375)	1	2.928	0.087	0.596
	Other non-White	0.000 (0.000)	0.077 (0.272)	1	1.213	0.271	0.400
	Hispanic	0.019 (0.135)	0.040 (0.195)	1	1.039	0.308	0.125
Bacterial vaginosis	White	0.027 (0.162)	0.033 (0.178)	1	0.193	0.660	0.035
	Black	0.068 (0.252)	0.054 (0.226)	1	0.441	0.506	0.058
	Asian	0.000 (0.000)	0.053 (0.229)	1	0.920	0.337	0.324
	Other non-White	0.000 (0.000)	0.000 (0.000)	1			
	Hispanic	0.019 (0.135)	0.020 (0.139)	1	0.006	0.937	0.009

Table 17. Continued

Urogenital Infection	Race/Ethnicity	Mean (SD) Preterm	Full-term	df	χ^2	p	Cohen's d
Trichomoniasis	White	0.004 (0.062)	0.005 (0.068)	1	0.027	0.870	0.013
	Black	0.023 (0.149)	0.020 (0.141)	1	0.036	0.850	0.017
	Asian	0.000 (0.000)	0.000 (0.000)	1	-	-	-
	Other non-White	0.000 (0.000)	0.077 (0.272)	1	1.213	0.271	0.400
	Hispanic	0.000 (0.000)	0.000 (0.000)	1	-	-	-
Yeast infection	White	0.084 (0.278)	0.082 (0.274)	1	0.012	0.912	0.009
	Black	0.109 (0.312)	0.125 (0.331)	1	0.312	0.577	0.050
	Asian	0.118 (0.332)	0.053 (0.229)	1	0.496	0.481	0.228
	Other non-White	0.200 (0.414)	0.192 (0.402)	1	0.004	0.952	0.019
	Hispanic	0.074 (0.263)	0.075 (0.264)	1	0.001	0.973	0.004

Table 17. Continued

Urogenital Infection	Race/Ethnicity	Mean (SD) Preterm	Full-term	df	χ^2	p	Cohen's d
Urinary tract infection	White	0.214 (0.411)	0.179 (0.384)	1	1.228	0.268	0.086
	Black	0.240 (0.428)	0.222 (0.416)	1	0.222	0.638	0.042
	Asian	0.118 (0.332)	0.158 (0.375)	1	0.122	0.727	0.114
	Other non-White	0.133 (0.352)	0.154 (0.368)	1	0.032	0.858	0.057
	Hispanic	0.185 (0.390)	0.182 (0.386)	1	0.006	0.940	0.009
Other infection	White	0.011 (0.107)	0.016 (0.127)	1	0.270	0.603	0.042
	Black	0.014 (0.116)	0.017 (0.129)	1	0.089	0.766	0.027
	Asian	0.000 (0.000)	0.053 (0.229)	1	0.920	0.337	0.324
	Other non-White	0.000 (0.000)	0.000 (0.000)	1	-	-	-
	Hispanic	0.019 (0.135)	0.024 (0.152)	1	0.094	0.759	0.036

infants were born to mothers diagnosed with herpes during their pregnancy. No differences in gestation term and infection were noted by any other racial/ethnic groups.

Maternal Stressful Life Events and Urogenital Infection

A biobehavioral model based on Wadhwa's Biobehavioural Model of Stress, Infection, and Preterm Birth (2001) was developed to test the direct, indirect, and interaction effects of maternal stress and infection on preterm birth (Figure 3). The model consisted of: maternal stress, (measured by 13 stressful life events), infection (measured by 12 self-reported urogenital infections, and the interaction effect of maternal stress and infection (constructed by multiplying the maternal stress and infection constructs). The outcome variable in the model was preterm birth. The fit of this model to the entire sample was less than acceptable-to-good: $\chi^2(40, N = 1647) = 221.93$, $p < 0.001$; GFI = 0.976, CFI = 0.866, NFI = 0.842; RMSEA = 0.053. SEM analysis indicated the interaction of maternal stress and infection was not a significant predictor of preterm birth (Table 18; $\beta = 0.022$, $p = 0.357$). Further, maternal stress was not a significant predictor of preterm birth ($\beta = 0.007$, $p = 0.775$). However, infection was a significant predictor of preterm birth ($\beta = -0.043$, $p = 0.07$) at $p < 0.10$, while maternal stress and infection had significant correlational effects on each other ($\beta = 0.192$, $p < 0.001$). Additionally, these direct and indirect effects along with the covariates accounted for 15% of the variance in preterm birth in this model.

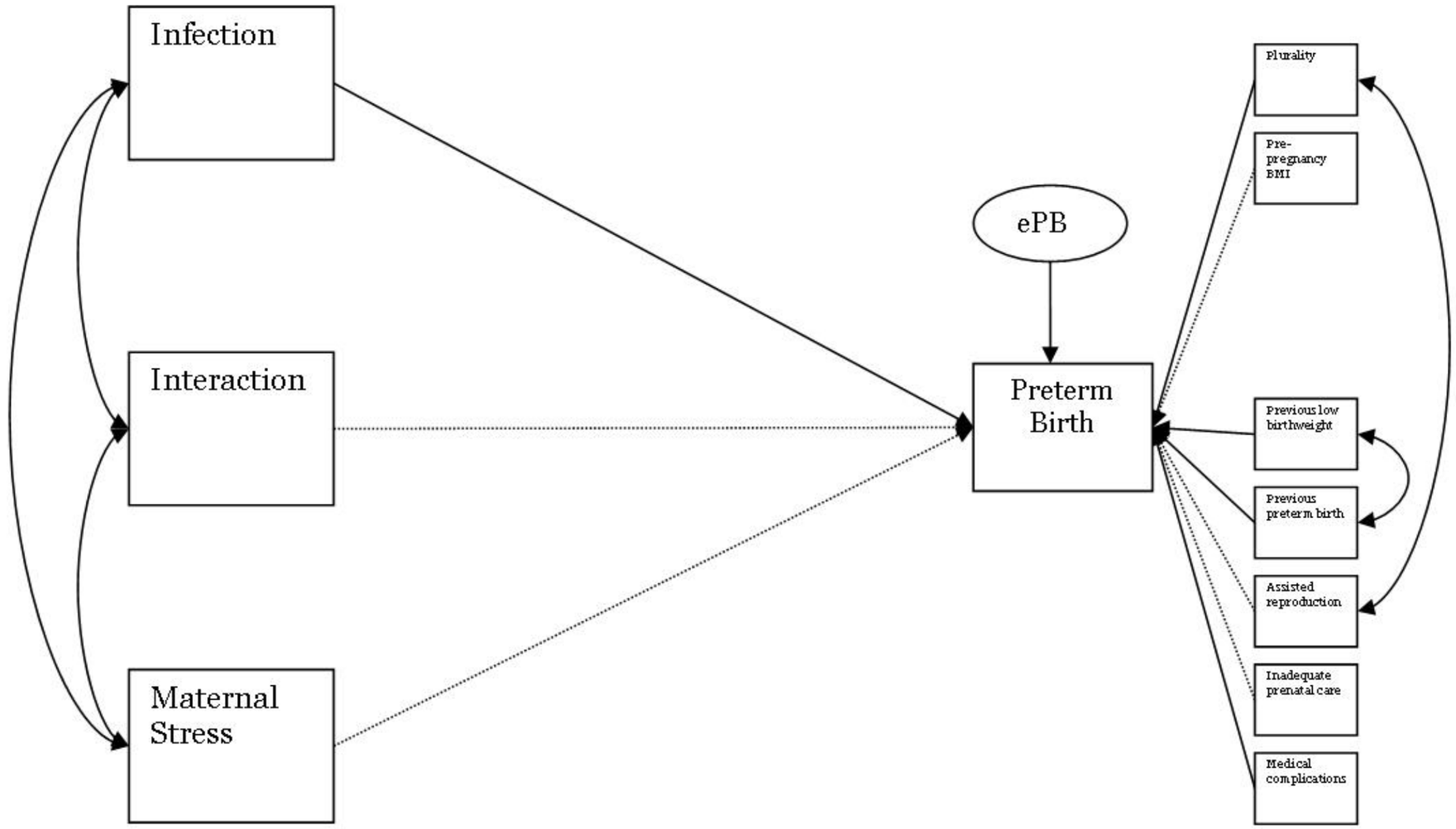

Figure 3. Interaction Model for Racial/Ethnic Groups. Significant predictors are depicted by solid arrows. Non-significant predictors are depicted by dashed arrows. The maternal stress/infection interaction was not a significant predictor of preterm birth in the overall sample or by racial/ethnic group. However, infection was a significant predictor of preterm birth in the overall sample and among Asian mothers.

Table 18

Direct and Interaction Effects with Covariates on Preterm Birth by Racial/Ethnic Group

Sample	Path	B	S.E.	C.R.	β	p
Total	Interaction → Preterm birth	0.005	0.006	0.922	0.022	0.357
	Maternal stress → Preterm birth	0.002	0.006	0.286	0.007	0.775
	Infection → Preterm birth	-0.026	0.014	-1.812	-0.043	0.070
	Interaction ↔ Maternal stress	0.599	0.096	6.209	0.155	<0.001
	Interaction ↔ Infection	0.320	0.038	8.349	0.210	<0.001
	Maternal stress ↔ Infection	0.314	0.041	7.648	0.192	<0.001
	Pre-pregnancy BMI → Preterm birth	0.003	0.002	1.505	0.034	0.132
	Previous low birthweight → Preterm birth	0.083	0.043	1.920	0.053	0.055
	Previous preterm birth → Preterm birth	0.193	0.040	4.792	0.132	<0.001
	Assisted reproduction → Preterm birth	0.100	0.057	1.753	0.041	0.080
	Inadequate prenatal care → Preterm birth	0.002	0.024	0.098	0.002	0.922
	Medical complications → Preterm birth	0.246	0.024	10.250	0.233	<0.001
	Plurality → Preterm birth	0.416	0.041	10.117	0.238	<0.001
	Previous preterm birth ↔ Previous low birthweight	0.056	0.003	19.885	0.562	<0.001
	Plurality ↔ Assisted reproduction	0.014	0.001	10.003	0.254	<0.001
White	Interaction → Preterm birth	0.005	0.009	0.508	0.018	0.612
	Maternal stress → Preterm birth	0.012	0.009	1.467	0.053	0.142
	Infection → Preterm birth	-0.032	0.024	-1.369	-0.050	0.171
	Interaction ↔ Maternal stress	0.630	0.143	4.402	0.170	<0.001
	Interaction ↔ Infection	0.356	0.054	6.627	0.261	<0.001
	Maternal stress ↔ Infection	0.311	0.058	5.346	0.208	<0.001
	Pre-pregnancy BMI → Preterm birth	0.002	0.003	0.835	0.029	0.404
	Previous low birthweight → Preterm birth	0.092	0.079	1.168	0.054	0.243
	Previous preterm birth → Preterm birth	0.222	0.070	3.156	0.145	0.002

Table 18. Continued

Sample	Path	B	S.E.	C.R.	β	p
White, cont.	Assisted reproduction → Preterm birth	0.099	0.073	1.350	0.050	0.177
	Inadequate prenatal care → Preterm birth	-0.011	0.040	-0.286	-0.010	0.775
	Medical complications → Preterm birth	0.271	0.036	7.503	0.261	<0.001
	Plurality → Preterm birth	0.349	0.057	6.093	0.224	<0.001
	Previous preterm birth ↔ Previous low birthweight	0.057	0.004	14.377	0.654	<0.001
	Plurality ↔ Assisted reproduction	0.024	0.003	8.191	0.328	<0.001
Black	Interaction → Preterm birth	-0.004	0.009	-0.390	-0.016	0.696
	Maternal stress → Preterm birth	<0.001	0.010	-0.006	<0.001	0.995
	Infection → Preterm birth	-0.024	0.021	-1.156	-0.048	0.248
	Interaction ↔ Maternal stress	0.969	0.208	4.666	0.210	<0.001
	Interaction ↔ Infection	0.322	0.096	3.348	0.149	<0.001
	Maternal stress ↔ Infection	0.324	0.090	3.584	0.16	<0.001
	Pre-pregnancy BMI → Preterm birth	-0.001	0.003	-0.264	-0.011	0.792
	Previous low birthweight → Preterm birth	0.017	0.066	0.256	0.012	0.798
	Previous preterm birth → Preterm birth	0.225	0.064	3.502	0.165	<0.001
	Assisted reproduction → Preterm birth	0.085	0.173	0.495	0.02	0.621
	Inadequate prenatal care → Preterm birth	-0.013	0.041	-0.306	-0.012	0.760
	Medical complications → Preterm birth	0.301	0.046	6.579	0.267	<0.001
	Plurality → Preterm birth	0.402	0.081	4.964	0.202	<0.001
	Previous preterm birth ↔ Previous low birthweight	0.063	0.006	10.286	0.507	<0.001
	Plurality ↔ Assisted reproduction	0.001	0.001	0.939	0.041	0.348
Asian	Interaction → Preterm birth	0.079	0.068	1.162	0.160	0.245
	Maternal stress → Preterm birth	0.047	0.066	0.722	0.089	0.470
	Infection → Preterm birth	-0.250	0.107	-2.338	-0.315	0.019
	Interaction ↔ Maternal stress	0.279	0.213	1.312	0.228	0.189
	Interaction ↔ Infection	-0.369	0.152	-2.425	-0.449	0.015

Table 18. Continued

Sample	Path	B	S.E.	C.R.	β	p
Asian, cont.	Maternal stress ↔ Infection	0.073	0.129	0.567	0.096	0.570
	Pre-pregnancy BMI → Preterm birth	-0.007	0.015	-0.479	-0.056	0.632
	Previous low birthweight → Preterm birth	0.394	0.444	0.886	0.219	0.376
	Previous preterm birth → Preterm birth	0.335	0.404	0.828	0.204	0.408
	Assisted reproduction → Preterm birth	-0.447	0.258	-1.737	-0.218	0.082
	Inadequate prenatal care → Preterm birth	-0.295	0.141	-2.097	-0.246	0.036
	Medical complications → Preterm birth	0.087	0.134	0.649	0.076	0.516
	Plurality → Preterm birth	0.815	0.311	2.621	0.330	0.009
	Previous preterm birth ↔ Previous low birthweight	0.096	0.024	3.909	0.880	<0.001
	Plurality ↔ Assisted reproduction	0.023	0.011	2.032	0.366	0.042
Other non-White	Interaction → Preterm birth	0.060	0.045	1.334	0.164	0.182
	Maternal stress → Preterm birth	-0.044	0.032	-1.354	-0.163	0.176
	Infection → Preterm birth	-0.004	0.079	-0.055	-0.007	0.956
	Interaction ↔ Maternal stress	-0.217	0.369	-0.589	-0.093	0.556
	Interaction ↔ Infection	-0.174	0.154	-1.131	-0.182	0.258
	Maternal stress ↔ Infection	0.035	0.208	0.169	0.027	0.866
	Pre-pregnancy BMI → Preterm birth	0.010	0.010	0.934	0.112	0.351
	Previous low birthweight → Preterm birth	0.506	0.197	2.564	0.348	0.010
	Previous preterm birth → Preterm birth	0.156	0.248	0.631	0.086	0.528
	Assisted reproduction → Preterm birth	-0.085	0.422	-0.202	-0.047	0.840
	Inadequate prenatal care → Preterm birth	-0.089	0.123	-0.726	-0.087	0.468
	Medical complications → Preterm birth	0.079	0.123	0.648	0.078	0.517
	Plurality → Preterm birth	0.743	0.370	2.006	0.464	0.045
	Previous preterm birth ↔ Previous low birthweight	0.040	0.015	2.680	0.468	0.007
	Plurality ↔ Assisted reproduction	0.066	0.016	4.109	0.855	<0.001

Table 18. Continued

Sample	Path	B	S.E.	C.R.	β	p
Hispanic	Interaction → Preterm birth	0.020	0.015	1.359	0.071	0.174
	Maternal stress → Preterm birth	-0.002	0.011	-0.213	-0.011	0.831
	Infection → Preterm birth	-0.050	0.040	-1.253	-0.067	0.210
	Interaction ↔ Maternal stress	0.099	0.173	0.571	0.030	0.568
	Interaction ↔ Infection	0.364	0.055	6.605	0.371	<0.001
	Maternal stress ↔ Infection	0.294	0.068	4.332	0.234	<0.001
	Pre-pregnancy BMI → Preterm birth	0.009	0.004	2.221	0.107	0.026
	Previous low birthweight → Preterm birth	0.057	0.089	0.640	0.035	0.522
	Previous preterm birth → Preterm birth	0.087	0.081	1.073	0.059	0.283
	Assisted reproduction → Preterm birth	0.234	0.124	1.884	0.091	0.060
	Inadequate prenatal care → Preterm birth	0.021	0.048	0.445	0.022	0.656
	Medical complications → Preterm birth	0.158	0.048	3.323	0.161	<0.001
	Plurality → Preterm birth	0.575	0.091	6.349	0.308	<0.001
	Previous preterm birth ↔ Previous low birthweight	0.041	0.005	8.023	0.467	<0.001
	Plurality ↔ Assisted reproduction	0.004	0.002	1.549	0.082	0.121

The biobehavioral model was also tested for robustness in the different racial/ethnic groups. Model fit for White mothers produced a similar pattern as the total sample, i.e., the fit was less than acceptable-to-good: $\chi^2(40, N = 691) = 140.17$, $p < 0.001$; GFI = 0.964, CFI = 0.872, NFI = 0.832; RMSEA = 0.06. The interaction of maternal stress and infection did not significantly predict preterm birth (Table 18; $\beta = 0.018$, $p = 0.612$), nor did maternal stress or infection. However, maternal stress had a significant correlational effect on infection ($\beta = 0.208$, $p < 0.001$). In this analysis, 17% of the variance in preterm birth was accounted for by these direct and indirect effects in addition to the covariates.

Similarly, among Black mothers, the fit of the model to the data was less than acceptable-to-good: $\chi^2(40, N = 518) = 69.973$, $p = 0.002$; GFI = 0.977, CFI = 0.902, NFI = 0.805; RMSEA = 0.038. The maternal stress and infection interaction did not significantly predict preterm birth (Table 18; $\beta = -0.016$, $p = 0.696$), while neither did maternal stress or infection. Additionally, maternal stress had significant correlational effects on infection ($\beta = 0.16$, $p < 0.001$). In this model, 15% of the variance in preterm birth was accounted for by the direct/indirect effects and covariates.

The model fit for Asian mothers was less than acceptable: $\chi^2(40, N = 36) = 60.647$, $p = 0.019$; GFI = 0.791, CFI = 0.774, NFI = 0.586; RMSEA = 0.121. The interaction effects did not significantly predict preterm birth in this group (Table 18; $\beta = 0.16$, $p = 0.245$). The direct effects of maternal stress did not significantly predict preterm birth ($\beta = 0.089$, $p = 0.47$); yet, the direct effects of

infection significantly predicted preterm birth (β = -0.315, p = 0.019). Additionally, no significant correlation effects were noted between maternal stress and infection in this group. However, 52% of the variance in preterm birth was accounted for by the direct and indirect effects and covariates in the model.

Model fit for Other non-White mothers produced a similar pattern as White mothers, i.e., the fit was less than acceptable-to-good: $\chi^2(40, N = 41) = 36.249$, p = 0.64; GFI = 0.88, CFI = 1.00, NFI = 0.706; RMSEA < 0.001. Furthermore, the interaction of maternal stress and infection did not significantly predict preterm birth (Table 18; β = 0.164, p = 0.182), while neither maternal stress nor infection were significant predictors of preterm birth. Maternal stress and infection exhibited no significant correlational effects on each other in this group. Yet, the direct and indirect effects along with the covariates accounted for 42% of the variance in preterm birth.

Among the Hispanic mothers, the fit of the model to the data was less than acceptable-to-good: $\chi^2(40, N = 361) = 55.59$, p = 0.052; GFI = 0.972, CFI = 0.932, NFI = 0.803; RMSEA = 0.033. The interaction of maternal stress and infection did not significantly predict preterm birth (Table 18; β = 0.071, p = 0.174). Additionally, neither maternal stress nor infection significantly predicted preterm birth. However, significant correlational effects were noted between maternal stress and infection in this group (β = 0.234, p < 0.001). In

this analysis, 17% of the variance in preterm birth was accounted for by the direct/indirect effects and covariates tested in the model.

Discussion

The purpose of this study was to examine the racial/ethnic disparities in maternal stressful life events, urogenital infection, and their interaction effect on preterm birth using a biobehavioral model. To the author's knowledge, this is the first to study to investigate this relationship.

Maternal stress affects all racial/ethnic groups during pregnancy; however, some stressful life events have greater impact on mothers based on their race/ethnicity (Lu & Halfon, 2003). In the present study, White mothers were at increased risk for preterm birth when they became homeless or if they had a partner serving jail time during the 12 months prior to delivery. Additionally, the risk for preterm birth declined among Other non-White mothers experiencing illness among family members prior to delivery. These findings contradict Lu and Chen's (2004) suggestion that the relationship between race and preterm delivery is probably not mediated through stress. However, additional findings of the current study support Lu and Chen's research. No significant differences were noted in the gestation term and maternal stressful life events among Black mothers in this study. This finding concurs with Lu and Chen's finding indicating that the relationship between Black women and preterm birth was weakened with inclusion of sociodemographic, medical, and behavioral variables and stress constructs (Lu &

Chen, 2004). These findings suggest that prevention programs that include counseling and coping resources should be culturally tailored based on the stressors of greatest risk for preterm birth among participants by racial/ethnic group.

Although several urogenital infections were assessed in this study, only herpes infection during pregnancy significantly impacted the gestation term by racial/ethnic group. Among Black women diagnosed with herpes, fewer preterm births as compared to full-term births were noted. Prior research has attempted to explain disparities in preterm birth by differences in the prevalence of infections among ethnic groups. Bacterial vaginosis has been reported as being more prevalent among Black and socially disadvantaged women (Fiscella, 2004; Kramer et al., 2001). This finding supports the hypothesis that urogenital infection could be an important mediating variable in explaining the high risk of preterm birth among the socially disadvantaged (Kramer et al., 2001). The hypothesis gained additional support, in 2007, when Hitti and colleagues found that Black women with bacterial vaginosis, chlamydia, and trichomoniasis were at increased risk for preterm birth. Additionally, Hitti et al. (2007) reported that Hispanic women with trichomoniasis were at increased risk for preterm birth. In the present study, Black women with bacterial vaginosis, chlamydia, and trichomoniasis were at increased risk for preterm birth; however, the risk for preterm birth was not increased among Hispanic women with trichomoniasis. Additionally, White women with trichomoniasis were at increased risk for

preterm birth. These findings suggest that prevention programs that include diagnosis and treatment services should be culturally tailored based on the infections of greatest risk for preterm birth among participants by racial/ethnic group.

In this study, a significant positive correlation was found between maternal stressful life events and urogenital infection in preterm birth even after controlling for covariates in White, Black, and Hispanic mothers. However, the relationship was stronger in White and Hispanic mothers. This finding differs from that of Hitti and colleagues (2007) who identified a stronger relationship between maternal stress and urogenital infection during pregnancy among Blacks and Hispanics as compared to Whites. This difference may indicate that the effects of maternal stress and infection during pregnancy manifest differently in pregnancy outcome based on the mother's race/ethnicity.

The fit of the biobehavioral model to the data showed similar goodness-of-fit measures as well as direct, indirect, and interaction effects on preterm birth for Whites, Blacks, Other non-Whites, and Hispanics. However, the model fit for Asians was vastly different from that of the other racial/ethnic groups. Additionally the variance in preterm birth accounted for by the model ranged from 15% in Blacks to 52% in Asians. These findings indicate that a one-size-fits-all approach to preventing to preterm birth may not be the most appropriate approach. This finding further supports the need for culturally specific prevention programs.

Lack of statistical significance of the effect of the two independent variables, maternal stress and infection, and their interaction, on preterm birth by racial/ethnic group should not diminish the importance of the study's findings. Given that cause of racial/ethnic disparities in preterm birth have yet to be identified and prevention efforts have not been successful in reducing the rates of preterm birth (IOM, 2006), the constraints related to statistical significance may not be appropriate for studies that simultaneously investigate risk factors of preterm birth, especially those that evaluate racial/ethnic disparities. Prevention specialists may need to seek alternative benchmarks for designing culturally tailored programs that focus on multiple risk factors.

This study is not without limitations. Although the sample size is large and representative of Florida's birth profile (48.3% White, 21.6% Black, 2.8% Asian, 0.6% Other non-White, and 28.6% Hispanic; March of Dimes, 2007), the analyses were conducted on unequal group sizes. Also, the stressful life events and urogenital infections represented in the study are not exhaustive. Additionally, research indicates that acculturation may play a role in disparities in preterm birth (Fiscella, 1996). Black mothers born in the United States have higher preterm birth rates as compared to those born outside the U.S. (Fiscella, 1996). This is phenomenon is not assessed in the current study, as it does not distinguish between Black mothers who are American-born, Caribbean-born, and African-born. Additionally, research has indicated that birth outcomes among Hispanic women worsen with duration of stay in the United States (Ruiz

et al., 2003). The current study does not assess this variable. However, the current findings provide insight for future studies analyzing racial/ethnic disparities in the relationship between maternal stressful life events and urogenital infection. Future studies should employ samples with equal group sizes and explore more comprehensive measures of maternal stress and infection. Additionally, future studies should explore the role of acculturation in explaining differences among racial/ethnic groups.

In conclusion, the study's findings have implications for targeted prevention programs, as a one-size-fits-all approach may not improve preterm birth rates among all racial/ethnic groups. These programs must include culturally specific counseling, as well as problem-solving and emotion-focused coping strategies. Additionally, these programs must include culturally specific awareness campaigns that highlight stressors and infections of greatest risk for preterm birth by racial/ethnic group.

CHAPTER V

CONCLUSION

The purpose of this dissertation was to examine the relationship and interaction between maternal stressful life events and urogenital infection in preterm birth. The project was comprised of three research studies. The first study examined prior research regarding the relationship between maternal stress and infection during pregnancy. The methodological quality of the research varied and the researchers failed to assess the relationship in pregnancy outcome. The findings of this investigation were inconclusive; thereby, prompting an investigation of maternal stress and infection in pregnancy outcome.

The second study examined the relationship between maternal stress and infection in preterm birth using a biobehavioral model. Maternal stress was measured by stressful life events experienced by the mother during the 12 months prior to delivery, while infection was measured by mother's diagnosis with urogenital infection during pregnancy. No significant differences were noted in the gestation term of mothers experiencing stressful life events prior to delivery. However, significant differences were revealed in the gestation term of mothers diagnosed with Group B streptococcus during pregnancy. Additionally, the relationship between maternal stress and infection was significant. However, the interaction between these variables was not significant.

The final study examined the racial/ethnic differences in maternal stress and infection in preterm birth. White mothers who became homeless or had a partner in jail during the 12 months prior to delivery gave birth to more preterm infants as compared to full-term infants. Other non-Whites who had an ill family member prior to delivery gave birth to fewer preterm infants as compared to full-term infants. Black mothers diagnosed with herpes during pregnancy delivered more full-term infants than preterm infants. The relationship between maternal stress and infection was significant among White, Black, and Hispanic mothers. However, the interaction of these two variables was not significant among any racial/ethnic group.

This study on maternal stressful life events and urogenital infection in preterm birth adds to the body of literature, since no prior studies have examined these risk factors simultaneously. Prevention specialists should advocate prevention programs that include counseling and coping resources. These programs should help women cope with multiple risk factors simultaneously. Additionally, these programs should be culturally tailored, as a one-size-fits-all approach may not yield significant reductions in preterm birth rates among all racial/ethnic groups.

REFERENCES

Bale, T. L. (2005). Is mom too sensitive? Impact of maternal stress during gestation. *Front Neuroendocrinol, 26*(1), 41-49.

Bennett, N. L., & Botti, J. J. (1989). New strategies for preterm labor. *Nurse Pract, 14*(4), 27-30, 33-24, 37-28.

Buekens, P., & Klebanoff, M. (2001). Preterm birth research: From disillusion to the search for new mechanisms. *Paediatr Perinat Epidemiol, 15*(Suppl 2), 159-161.

Coussons-Read, M., Okun, M., & Simms, S. (2003). The psychoneuroimmunology of pregnancy. *Journal of Reproductive and Infant Psychology, 21*(2), 103-112.

Culhane, J. F., Rauh, V., McCollum, K. F., Elo, I. T., & Hogan, V. (2002). Exposure to chronic stress and ethnic differences in rates of bacterial vaginosis among pregnant women. *Am J Obstet Gynecol, 187*(5), 1272-1276.

Culhane, J. F., Rauh, V., McCollum, K. F., Hogan, V. K., Agnew, K., & Wadhwa, P. D. (2001). Maternal stress is associated with bacterial vaginosis in human pregnancy. *Matern Child Health J, 5*(2), 127-134.

Darwish, A. M., Makarem, M. H., Alnashar, E. M., & Hamadeh, S. M. (2005). Screening for bacterial vaginosis in high-risk pregnancy: The experience of a developing country. *Acta Obstet Gynecol Scand, 84*(5), 483-485.

de Weerth, C., & Buitelaar, J. K. (2005). Physiological stress reactivity in human pregnancy – A review. *Neurosci Biobehav Rev, 29*(2), 295-312.

Dole, N., Savitz, D. A., Hertz-Picciotto, I., Siega-Riz, A. M., McMahon, M. J., & Buekens, P. (2003). Maternal stress and preterm birth. *Am J Epidemiol, 157*(1), 14-24.

Fiscella, K. (1996). Racial disparities in preterm births. The role of urogenital infections. *Public Health Rep, 111*(2), 104-113.

Fiscella, K. (2004). Racial disparity in infant and maternal mortality: Confluence of infection, and microvascular dysfunction. *Matern Child Health J, 8*(2), 45-54.

Florida Department of Health (Florida DOH). (2007). *Pregnancy Risk Assessment Monitoring System (PRAMS).* Retrieved September 3, 2007 from http://www.doh.state.fl.us/Disease_ctrl/epi/Chronic_Disease/PRAMS/Intro.htm.

Garrard, J. (2004). *Health sciences literature review made easy: The matrix method* (2nd ed.). Boston, MA: Jones & Bartlett Publishers.

Gennaro, S., & Hennessy, M. D. (2003). Psychological and physiological stress: Impact on preterm birth. *J Obstet Gynecol Neonatal Nurs, 32*(5), 668-675.

Glanz, K., Rimer, B.K., & Lewis, F.M. (2002). *Health behavior and health education: Theory, research, and practice* (3rd ed.). San Francisco: Jossey-Bass.

Goldenberg, R. L., Culhane, J. F., & Johnson, D. C. (2005). Maternal infection and adverse fetal and neonatal outcomes. *Clin Perinatol, 32*(3), 523-559.

Goodson, P., Buhi, E.R., & Dunsmore, S.C. (2006). Self-esteem and adolescent sexual behaviors, attitudes, and intentions: A systematic review. *Journal of Adolescent Health, 38*, 310-319.

Green, N. S., Damus, K., Simpson, J. L., Iams, J., Reece, E. A., Hobel, C. J., et al. (2005). Research agenda for preterm birth: Recommendations from the March of Dimes. *Am J Obstet Gynecol, 193*(3 Pt 1), 626-635.

Harville, E. W., Savitz, D. A., Dole, N., Thorp, J. M., Jr., & Herring, A. H. (2007). Psychological and biological markers of stress and bacterial vaginosis in pregnant women. *BJOG, 114*(2), 216-223.

Hitti, J., Nugent, R., Boutain, D., Gardella, C., Hillier, S. L., & Eschenbach, D. A. (2007). Racial disparity in risk of preterm birth associated with lower genital tract infection. *Paediatr Perinat Epidemiol, 21*(4), 330-337.

Hobel, C., & Culhane, J. (2003). Role of psychosocial and nutritional stress on poor pregnancy outcome. *J Nutr, 133*(5 Suppl 2), 1709S-1717S.

Huck, S. W. (2003). *Reading statistics and research* (4th ed.). Boston: Allyn & Bacon.

Institute of Medicine (IOM). (2006). *Preterm birth: Causes, consequences, and prevention. Executive summary*. Washington, DC: National Academy Press.

Institute of Medicine (IOM). (2007). *Preterm birth: Causes, consequences, and prevention*. Washington, DC: National Academy Press.

King, J., & Flenady, V. (2002). Prophylactic antibiotics for inhibiting preterm labour with intact membranes. *Cochrane Database Syst Rev*(4), CD000246.

Krabbendam, L., Smits, L., de Bie, R., Bastiaanssen, J., Stelma, F., & van Os, J. (2005). The impact of maternal stress on pregnancy outcome in a well-educated Caucasian population. *Paediatr Perinat Epidemiol, 19*(6), 421-425.

Kramer, M. S., Goulet, L., Lydon, J., Seguin, L., McNamara, H., Dassa, C., et al. (2001). Socio-economic disparities in preterm birth: Causal pathways and mechanisms. *Paediatr Perinat Epidemiol, 15*(Suppl 2), 104-123.

Lee, K., Schotland, M., Bacchetti, P., & Bero, L.A. (2002). Association of journal quality indicators with methodological quality of clinical research articles. *JAMA, 287*(21), 2805-2808.

Lothian, J. (2006). Saying “no” to induction. *J Perinat Educ., 15*(2), 43–45.

Lu, M. C., & Chen, B. (2004). Racial and ethnic disparities in preterm birth: The role of stressful life events. *Am J Obstet Gynecol, 191*(3), 691-699.

Lu, M. C., & Halfon, N. (2003). Racial and ethnic disparities in birth outcomes: A life-course perspective. *Matern Child Health J, 7*(1), 13-30.

Majeroni, B. A., & Ukkadam, S. (2007). Screening and treatment for sexually transmitted infections in pregnancy. *Am Fam Physician, 76*(2), 265-270.

March of Dimes (2007). *Peristats*. Retrieved on October 8, 2007 from http://www.marchofdimes.com/peristats/

McGregor, J. A., French, J. I., Parker, R., Draper, D., Patterson, E., Jones, W., et al. (1995). Prevention of premature birth by screening and treatment for common genital tract infections: results of a prospective controlled evaluation. *Am J Obstet Gynecol, 173*(1), 157-167.

Moutquin, J. M. (2003a). Classification and heterogeneity of preterm birth. *BJOG, 110*(Suppl 20), 30-33.

Moutquin, J. M. (2003b). Socio-economic and psychosocial factors in the management and prevention of preterm labour. *BJOG, 110*(Suppl 20), 56-60.

Mulder, E. J., Robles de Medina, P. G., Huizink, A. C., Van den Bergh, B. R., Buitelaar, J. K., & Visser, G. H. (2002). Prenatal maternal stress: Effects on pregnancy and the (unborn) child. *Early Hum Dev, 70*(1-2), 3-14.

Mullick, S., Watson-Jones, D., Beksinska, M., & Mabey, D. (2005). Sexually transmitted infections in pregnancy: Prevalence, impact on pregnancy outcomes, and approach to treatment in developing countries. *Sex Transm Infect, 81*(4), 294-302.

Nelson, D. B., Bellamy, S., Nachamkin, I., Ruffin, A., Allen-Taylor, L., & Friedenberg, F. K. (2008). Characteristics and pregnancy outcomes of pregnant women asymptomatic for bacterial vaginosis. *Matern Child Health J, 12*(2), 216-222.

O' Neill, M. S., Hertz-Picciotto, I., Pastore, L. M., & Weatherley, B. D. (2003). Have studies of urinary tract infection and preterm delivery used the most appropriate methods? *Paediatr Perinat Epidemiol, 17*(3), 226-233.

Othman, M., Neilson, J. P., & Alfirevic, Z. (2007). Probiotics for preventing preterm labour. *Cochrane Database Syst Rev*(1), CD005941.

Pararas, M. V., Skevaki, C. L., & Kafetzis, D. A. (2006). Preterm birth due to maternal infection: Causative pathogens and modes of prevention. *Eur J Clin Microbiol Infect Dis, 25*(9), 562-569.

Patel, R. R., Steer, P., Doyle, P., Little, M. P., & Elliott, P. (2004). Does gestation vary by ethnic group? A London-based study of over 122,000 pregnancies with spontaneous onset of labour. *Int J Epidemiol, 33*(1), 107-113.

Roberts, J. K. (1999, January). *Basic concepts of confirmatory factor analysis*. Paper presented at the annual meeting of the Southwest Educational Research Association, San Antonio (ERIC Document Reproduction Service No. ED 427 091).

Ruiz, R. J., Fullerton, J., Brown, C. E. L., & Dudley, D. J. (2002). Predicting risk of preterm birth: The roles of stress, clinical risk factors, and corticotropin-releasing hormone. *Biol Res Nurs, 4*(1), 54-64.

Ruiz, R. J., Fullerton, J., Brown, C. E. L., & Schoolfield, J. (2001). Relationships of cortisol, perceived stress, genitourinary infections, and fetal fibronectin to gestational age at birth. *Biol Res Nurs, 3*(1), 39-48.

Ruiz, R. J., Fullerton, J., & Dudley, D. J. (2003). The interrelationship of maternal stress, endocrine factors and inflammation on gestational length. *Obstet Gynecol Surv, 58*(6), 415-428.

Stapleton, C. (1997, January). *Basic concepts and procedures of confirmatory factor analysis*. Paper presented at the annual meeting of the Southwest Educational Research Association, Austin (ERIC Document Reproduction Service No. ED 407 416).

Steer, P. (2005). The epidemiology of preterm labor – A global perspective. *J Perinat Med, 33*(4), 273-276.

Stevens, J. P. (2002). *Applied multivariate statistics for the social sciences* (4th ed.). Mahwah: Lawrence Erlbaum Associates, Inc.

Sun, J. (2005). Assessing goodness of fit in confirmatory factor analysis. *Measurement and Evaluation in Counseling and Development, 37*, 240-256.

Thompson, B. (2004). *Exploratory and confirmatory factor analysis: Understanding concepts and applications*. Washington DC: American Psychological Association.

Trabert, B., & Misra, D. P. (2007). Risk factors for bacterial vaginosis during pregnancy among African American women. *Am J Obstet Gynecol, 197*(5), 477.e1-477.e8.

U.S. Department of Health of Human Services (USDHHS). (2000). *Healthy people 2010: Understanding and improving health.* Rockville, MD, Office of Disease Prevention and Health Promotion.

U.S. Department of Health of Human Services (USDHHS). (2007). *Pregnancy Risk Assessment Monitoring System (PRAMS): Home*. Retrieved September 3, 2007 from http://www.cdc.gov/PRAMS/.

Wadhwa, P .D., Culhane, J. F., Rauh, V., Barve, S. S., Hogan, V., Sandman, C. A., et al. (2001). Stress, infection and preterm birth: A biobehavioural perspective. *Paediatr Perinat Epidemiol, 15*(Suppl 2), 17-29.

Windsor, R., Clark, N., Boyd, N., Goodman, R. (2004). *Evaluation of health promotion, health education, and disease prevention programs* (3rd ed.). New York: McGraw-Hill.

Yaris, F., Kadioglu, M., Kesim, M., Unsal, M., Yaris, E., Ulku, C., et al. (2004). Urinary tract infections in unplanned pregnancies and fetal outcome. *Eur J Contracept Reprod Health Care, 9*(3), 141-146.

VITA

Name: Joy Lavonne Anderson

Address: 4243 TAMU
College Station, TX 77843-4243

Email Address: jlanderson@hlkn.tamu.edu

Education: Ph.D., Health Education, Texas A&M University, 2008
M.S., Health Education, Florida State University, 2004
B.S., Chemistry Pre-medicine, Florida A&M University, 1999

Professional Experience: (08/2005 – 05/2008) Graduate Research Assistant, *Center for the Study of Health Disparities*, *Texas A&M University*, College Station, Texas

(04/2000 - 08/2005) Regulatory Specialist, *Florida Public Service Commission*, Tallahassee, Florida

Publications:

Anderson, J.L., Warren, C.A., Perez, E., Louis, R.I., Phillips, S., Wheeler, J., Cole, M., Misra, R. (in press). Gender and ethnic differences in hand hygiene practices among college students. *American Journal of Infection Control.*

Anderson, J.L. (2006). An evaluation of African American adolescent health status with gender comparison. *Californian Journal of Health Promotion, 4*(2) 168-174.