THE EFFECTS OF EARLY LIFE FACTORS AND PRECONCEPTION STRESS ON BIRTH WEIGHT IN A NATIONAL SAMPLE OF U.S. WOMEN

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A dissertation submitted to the faculty of the University of North Carolina at Chapel Hill in partial fulfillment of the requirements for the degree of Doctor of Philosophy in the Department of Maternal and Child Health.

Chapel Hill
2013

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ABSTRACT

KELLY KATHRYN STRUTZ: The Effects of Early Life Factors and Preconception Stress on Birth Weight in a National Sample of U.S. Women
(Under the direction of Dr. Jon M. Hussey)

The purpose of this dissertation is to examine the impact of preconception stress on offspring birth weight, racial/ethnic birth weight disparities, and the intergenerational transmission of birth weight. Analysis of singleton live first and second births to non-Hispanic White, Mexican- and other-origin Latina, and non-Hispanic Black women in the National Longitudinal Study of Adolescent Health was used to address this topic. The first paper of the dissertation examined the roles of latent factors for preconception acute and chronic stress on offspring birth weight and birth weight disparities, including the potentially mediating role of preconception depressive symptoms. Linear regression models indicated that chronic stressors, but not acute stressors or depressive symptoms, were inversely associated with birth weight for both first and second births and partially explained the disparities in birth weight between the minority racial/ethnic groups and non-Hispanic Whites. The second paper of the dissertation examined the persistence of birth weight across generations, including the potentially moderating roles of preconception acute and chronic stressors. Linear regression models indicated that maternal birth weight partially explained the Black-White disparity in offspring birth weight, but that preconception stress did not modify the positive association between maternal and

offspring birth weight. The findings suggest that both a woman's birth weight and her experience of chronic stressors through the reproductive period have individual effects on the birth weights of her offspring.

For Beatrice Lee Strutz,

with apologies for the preconception

(and prenatal, and postpartum)

stress that it occasionally caused.

ACKNOWLEDGEMENTS

I found this to be the most difficult section of the dissertation to write, because I have so many people to thank. First, I couldn't have asked for a better advisor and chair than Jon Hussey. Jon provided me with just the right mix of independence and oversight, challenged me to think more deeply and critically about my research, and modeled thoughtful and reasoned scholarship while maintaining balance in life. Our collaborations with Liana Richardson have been particularly enriching; I am grateful to Liana for her additional mentorship on so many aspects of research and professional development.

I'm very thankful to my committee members who've supported me, not just during the dissertation process, but throughout my doctoral program. Anna Maria Siega-Riz and the other faculty and trainees on the Reproductive, Perinatal, and Pediatric Epidemiology Training Grant (NICHD T32-HD052468-02) taught me to think like an epidemiologist and to situate my research in a wider disciplinary conversation. Vijaya Hogan expanded and refined everything I thought I knew about health disparities, and helped me to get my first first-authored paper published through the MCHB Epidemiology Health Disparities traineeship. Carolyn Halpern and our colleagues on the Sexual Risk Trajectories Project broadened my ideas about adolescent and young adult health and development (with a healthy dose of

humor). Chirayath Suchindran was always generous with his time, providing extensive answers to my analysis questions for this and other projects.

The Department of Maternal and Child Health provided a collaborative environment in which I could do my best work. I am very grateful for departmental financial support through the Cynthia H. Cassell Doctoral Dissertation Award in MCH, a nomination for the Graduate School Doctoral Merit Assistantship, and GRA positions. Thanks to all of the MCH faculty who've provided me with opportunities to develop my research and professional skills, especially Sherri Green and Jonathan Kotch. Thanks also to Yvette Thompson, Carrie Aldrich, and Lisa Perry for keeping me and the other students organized and on track. I feel fortunate to have spent this time around so many impressive MCH students, and would particularly like to thank my cohortmates Laili Irani, Layla Lavasani, Winnie Luseno, Martha Skiles, and Khadija Turay: Martha, Layla, and Winnie, for sharing so many meals and conversations over the years; Laili, for the fun we've had on conference trips; and Khadija, my fellow last woman standing, for filling our last year with commiseration, motivation (WWHRCD?) and CSA produce. Thanks also to Shoshana Goldberg and Leslie deRosset for being great collaborators, and to Alane Murdock, Khadija, and Shoshana for helpful feedback on my defense presentation.

My education was also enhanced by my traineeship with the Carolina Population Center (NICHD R24-HD050924) and its wealth of resources, with thanks to Jan Hendrickson-Smith for her uncanny ability to connect the trainees to exactly what we need before we know we need it. I am very grateful to the PIs, staff, and participants of Add Health, a program project directed by Kathleen Mullan Harris and

designed by J. Richard Udry, Peter S. Bearman, and Kathleen Mullan Harris at the University of North Carolina at Chapel Hill, and funded by grant P01-HD31921 from the *Eunice Kennedy Shriver* National Institute of Child Health and Human Development, with cooperative funding from 23 other federal agencies and foundations. Special acknowledgment is due Ronald R. Rindfuss and Barbara Entwisle for assistance in the original design. Information on how to obtain the Add Health data files is available on the Add Health website (http://www.cpc.unc.edu/addhealth). No direct support was received from grant P01-HD31921 for this analysis. All Add Health procedures were approved by the Public Health Institutional Review Board (IRB) at the University of North Carolina at Chapel Hill; the present dissertation analyses were deemed exempt. I would especially like to thank the Add Health Stress Measures Working Group for identifying available measures of acute and chronic stressors, and Joyce Tabor, Eric Whitsel, Quynh Nguyen, Ping Chen, and Mariah Chang for assistance in navigating this rich and complex data source.

Finally, I'd like to thank all of my family and friends for everything they've done for me over the years. In particular, my parents, Carol and Donald LaPlante, have always encouraged me to pursue my goals, intellectual and otherwise, and were my biggest cheerleaders when those pursuits resulted in a PhD. I couldn't have done any of this, and wouldn't even have wanted to, without the unwavering support and patience of my husband, Jason Strutz. Thank you to Jason and to everyone who made our time in North Carolina so meaningful. I'm looking forward to our next adventure.

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LIST OF ABBREVIATIONS

Add Health	The National Longitudinal Study of Adolescent Health
BMI	body mass index
BRFSS	Behavioral Risk Factor Surveillance System
CARDIA	Coronary Artery Risk Development in Young Adults
CES-D	Center for Epidemiologic Studies – Depression scale
CFI	comparative fit index
CI	confidence interval
kg	kilograms
m	meters
MCH	maternal and child health
MEPS	Medical Expenditure Panel Survey
PRAMS	Pregnancy Risk Assessment and Monitoring System
PSID	Panel Survey of Income Dynamics
TLI	Tucker-Lewis index
U.S.	United States
RMSEA	root mean square error of approximation

CHAPTER 1

INTRODUCTION

Birth Weight and Preconception Health

Birth weight is a marker of infant health at birth and an important predictor of an infant's survival and subsequent health status. Low birth weight, defined as weight at birth of less than 2500 grams, is associated with an increased risk of developing both short-term and long-term health and educational problems.[1] In 2007, 8.2% of infants in the United States (U.S.) were born at low birth weight; the *Healthy People 2020* target calls for a 5% improvement over this baseline to 7.8%.[2,3] Although copious risk factors for restricted birth weight have been identified, they have failed to explain how the prevalence of restricted birth weight has increased over time[2] or how marked racial/ethnic disparities persist.[1,4-6] In addition, birth weight has been demonstrated to be correlated across generations, in a relationship believed to have genetic, environmental, and gene-environment interaction (including epigenetic) components[7] that may contribute to racial/ethnic disparities.[8,9]

Because prenatal care and other pregnancy interventions have not been sufficient for mitigating the increase and disparities in prevalence of adverse birth outcomes, attention has shifted to preconception health, defined broadly as health before a pregnancy.[10] The concept of preconception health, situated within a life

course perspective,[11-13] acknowledges that birth outcomes are affected by maternal development prior to the nine-month prenatal period. Preconception health includes interconception health, or health between pregnancies, and is equally important for promoting women's own well-being and healthy development.[14] Although "preconception" could theoretically describe any period in the life course prior to a pregnancy, this term is often used in clinical and public health practice to refer specifically to the reproductive years of women (and, to a lesser extent, men).

Interest in preconception health was rooted in prenatal care,[15] and has since expanded beyond the health care sector to encompass social and behavioral determinants of health and preventive interventions for women and their potential offspring.[10] Efforts to examine the prevalence and impact of preconception risk and protective factors in women of childbearing age have been hindered by the lack of comprehensive surveillance systems for preconception health in the U.S.[16] To address this limitation, state maternal and child health (MCH) epidemiologists compiled a list of measures available in existing state-level surveys such as the Pregnancy Risk Assessment and Monitoring System (PRAMS) and the Behavioral Risk Factor Surveillance System (BRFSS).[17] These surveys provide some information about the prevalence of preconception exposures in postpartum women or in the general population; for example, 18.5% of postpartum women responding to PRAMS surveys in 2004 reported experiencing four or more stressors in the preconception period.[18] The identification of indicators and baseline statistics enabled the introduction of preconception health and behavior objectives in *Healthy People 2020*.[3] Although data sources to examine the impact of these preconception

factors on health outcomes also have been limited, research is developing on the effects of preconception exposures for women and their infants.

Life Course Theory

The increased emphasis on preconception health exemplifies the growing interest in life course theory in the fields of MCH and epidemiology. Originally developed by sociologist Glen Elder, Jr.[19,20] and popularized in public health by Lu and Halfon[21] and Kuh and Ben-Shlomo,[11] life course theory emphasizes the health consequences of exposures across an individual's entire life span rather than limiting to proximal determinants of health conditions.[13] Key principles of life course theory pertinent to preconception and infant health include the principle of timing, which proposes that the timing and chronology of exposures, including duration and sequencing, is as influential as the exposures themselves, and the principle of linked lives, which acknowledges the interconnection among family members and other social networks in altering health status.[12,22]

Three life course models have been propounded in the public health literature: the latency model, in which exposures in a critical or sensitive period affect later health independent of intervening exposures; the cumulative model, in which exposures accumulate over time to produce a greater impact on health than any single exposure; and the pathway model, in which early exposures lead to later risk factors and subsequently to health outcomes.[13,23-25] With respect to these models, the preconception period could represent either a critical/sensitive period, an accumulation of exposure from early life, an intervening period that modifies a

relationship between early life factors and the outcome of interest, or a combination of the above. In addition, the influence of a mother's own health at birth on her offspring could represent a critical period or the beginning of an accumulation or pathway of exposures. The application of life course theory to empirical testing of MCH topics is relatively nascent and will require further refinement to distinguish among competing models or model combinations.

Stress Theory and Mechanisms

The health effects of stress throughout the life course have been of particular interest to public health and social science researchers alike. One key theory linking social stress to health conditions is Pearlin's stress process model.[26-28] This model posits that social characteristics including gender, race/ethnicity, and socioeconomic status lead to stress exposures such as stressful life events (acute stressors) and stressful life conditions (chronic stressors). These stressors, in turn, affect physical and mental health status. The model also accounts for effect modification of the relationship between stressors and health by social and personal resources.[29] The stress process model has been explicitly integrated with life course theory,[30] and is increasing in popularity as a framework to understand elevated risks of adverse health outcomes among minority groups and groups with low social status.[29,31]

In perinatal health research, physiologic mechanisms have been hypothesized to connect maternal stress to reproductive function and infant health outcomes. It has been suggested that acute stressful events in early life can program stress reactivity that persists into adulthood, while cumulative exposure to

chronic stress has been hypothesized to result in accelerated aging, or “weathering”, wearing down the body’s adaptive systems and affecting hormones during pregnancy.[32-36] These neuroendocrine and immunological dysregulation pathways represent possible biologic mechanisms by which stress over the life course can lead to low birth weight and birth weight disparities. In addition, other mechanisms, including depressive symptoms, are likely to mediate the relationship between stress and physical health outcomes, including birth outcomes.[37] Depressive symptoms can result from stressors and are thought to affect the same neuroendocrine pathways as stress itself.[38] Because African-American women report higher levels of depressive symptoms than non-Hispanic White women, this has also been proposed as a contributor to the Black-White disparity in adverse birth outcomes.[39-41]

Stress Measurement

It is difficult to operationalize a complex construct such as stress. Therefore, studies of the health consequences of stress have utilized varied measures. Historically, stress has been defined most frequently as exposure to an inventory of life events within a specified period of time.[42] These acute stressors are relatively brief in duration although they may have continued ramifications; examples include experiencing a natural disaster or termination of employment. The use of acute life events as indicators of stress can be problematic, because respondents may differ in their subjective perception of the negativity or lasting impact of an event.[37,42] To overcome this limitation, some studies have been able to incorporate subjective perceptions of acute stressors into life event scales.[36] Further, heterogeneity in the

timing of the events with respect to the health outcome of interest can bias estimates of associations, and thus researchers must be careful in defining the relevant period of exposure.[43] Despite their limitations, counts of stressful life events remain the most common measure of stress in health research,[42] likely due to the relative ease in obtaining these measures and to consistency with prior studies.

In contrast, more recent studies have defined stress using measures of stressful life conditions.[36] These chronic stressors recur or accumulate throughout a respondent's life; examples include persistent socioeconomic disadvantage or daily exposure to hassles.[36,37] Moreover, chronic stressors include measures specific to particular racial/ethnic groups or subgroups thereof, such as exposure to racial discrimination/racism and to negative aspects of immigrant acculturation.[36,44,45] Consistent with a life course perspective on health and with hypothesized mechanisms of biologic dysregulation resulting from stress, the use of chronic life conditions as indicators of stress has increased rapidly in recent years. However, measurement of stressful life conditions is less standardized across studies than that of stressful life events; while validated scales of acute events have been developed,[36] the same is not true of chronic conditions. In addition, estimates of associations between chronic stressors and health conditions can be biased by differences in physiological and behavioral responses to chronic stressors both across and within individuals.[46,47] Although the measurement of chronic stressors requires further development, initial studies have demonstrated promising results.

Stress and Birth Outcomes

The vast majority of studies assessing the effects on birth outcomes of stress and depression have relied on measurement during the prenatal period only.[36,48-64] Not surprisingly, results of these studies have been mixed. It is likely that much of the variation in the results can be attributed to variations in definitions and measurement of stress[36] or of variations in sampled populations. While these studies suggest an effect of stress on infant health status, the prenatal period may not have been the most appropriate window of time in which to measure stress and related factors. It is likely that the more robust effects of chronic stressors measured in pregnancy were capturing cumulative exposures from the preconception period.

A smaller number of studies have examined the effects of acute or chronic stressors in the reproductive period on birth outcomes,[65-67] with no studies including both acute and chronic stressors. Although the examination of stressful life events is common in the prenatal literature, only one study examined preconception stress as measured by a psychosocial hassles scale,[65] demonstrating a negative but nonsignificant effect of high stress on birth weight limited by a small sample size. Additional studies of low birth weight or preterm birth in national cohorts suggested effects of specific chronic stressors, but were limited to U.S. births at younger maternal ages[66] or utilized a British birth cohort that may prevent generalizability of the findings to the U.S.[67] In addition, three studies[68-70] examined preconception depressive symptoms as measured by the Center for Epidemiologic Studies - Depression (CES-D) [71] on gestational age, finding mixed results. These studies were hampered by the use of retrospectively reported depressive symptoms[69] or

prospective measures reported only in a narrow window of time prior to the pregnancy.[70] Moreover, none of these studies examined nationally representative samples of U.S. women or compared racial/ethnic differences for racial/ethnic groups besides non-Hispanic White and non-Hispanic Black. The sole nationally representative study of preconception mental health on birth outcomes[72] did not examine depressive symptoms specifically, but instead used a global mental health rating. In this analysis of data from the Medical Expenditure Panel Survey (MEPS), self-reported fair or poor mental health was associated with increased odds of low birth weight and other pregnancy complications.

It is worth acknowledging that an additional line of inquiry has focused on the effects of chronic stressors early in a woman's life (prior to the reproductive period). Studies of household or neighborhood poverty at the time of the mother's own birth suggest an effect of poverty in early life on offspring birth weight that may be independent of or modified by maternal socioeconomic status measured during pregnancy,[73-76] while evaluations of the weathering hypothesis, using age as a marker for the accumulation of social stressors, have shown some support for the hypothesis for low birth weight and other infant outcomes.[35,77-84] These studies of early life provide evidence that stressors prior to pregnancy affect offspring health.

Studies of the role of stress in birth weight transmission have been more limited given that most have come from birth registry linkages across generations with little information on stressors. However, examinations of the moderating role of low socioeconomic status suggest that maternal socioeconomic status and support factors at the time of the offspring birth may be less important than those existing

earlier in her life,[85,86] although evidence is mixed.[87,88] No studies examined socioeconomic factors in the reproductive period, or additional aspects of chronic or acute stress.

Study Overview

To address a number of gaps in the literature reviewed above and in further detail in the following chapters, this dissertation evaluates the effects of both acute and chronic preconception stressors on birth weight. Informed by life course and stress theories, it tests hypotheses about the effects of acute stressors (stressful life events) and chronic stressors (stressful life conditions) in the preconception period on birth weight, including the roles of acute and chronic stressors in racial/ethnic disparities in birth weight and in the intergenerational transmission of birth weight. Further, this project explores preconception depressive symptoms as a pathway through which stress may impact birth weight. By assessing preconception (including interconception) factors, this project goes beyond traditional analyses of prenatal risk factors to identify novel influences on suboptimal birth weight and its persistence across generations.

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CHAPTER 2

PRECONCEPTION STRESS, BIRTH WEIGHT, AND BIRTH WEIGHT DISPARITIES AMONG U.S. WOMEN

INTRODUCTION

Birth Weight and Preconception Health

Birth weight is a marker of infant health at birth and an important predictor of an infant's survival and subsequent health status. Low birth weight, defined as weight at birth of less than 2500 grams, is associated with an increased risk of developing both short-term and long-term health problems.[1] The prevalence of restricted birth weight has been increasing since the 1980s in the United States (U.S.);[2] and marked differences in birth weight persist by race/ethnicity[3,4] Although copious risk factors for restricted birth weight have been identified, they have failed to explain the increase over time or the disparities in prevalence.[1,5]

Failures of prenatal care and other pregnancy interventions to address the increase and disparities in prevalence of adverse birth outcomes have led to a focus on preconception health, defined broadly as health before a pregnancy (although often used in public health practice to denote health during the reproductive years) and including interconception health, or health between pregnancies.[6,7] Drawing on a life course framework,[8-10] the concept of preconception health suggests that

infants are affected not only by maternal exposures in the nine-month prenatal period, but also by maternal development before the pregnancy.

Stress Theory and Measurement

One preconception exposure of interest is stress. Pearlin's stress process model posits that social characteristics including those surrounding race/ethnicity in the U.S. lead to stress exposures that affect health,[11-13] and has been used to understand elevated risk of adverse health outcomes among minority groups.[14,15] It is worth noting that elevated stress is not inherent to persons of minority race/ethnicity, given that race/ethnicity is a social construct and not a biological one. Rather, stress results from historical and societal constraints leading to differential life chances across groups.[16]

In studies of its health consequences, stress has been defined most frequently as exposure to an inventory of life events within a specified period of time.[17] These acute stressors are relatively brief in duration but may have continued ramifications.[17,18] Consistent with a life course perspective, more recent studies have examined chronic stressors as a risk factor for health outcomes.[18,19] These stressful life conditions recur or accumulate throughout a respondent's life. However, measurement of chronic stressors is less standardized across studies than that of acute stressors; validated scales of acute events[19] but not chronic conditions have been developed.

Stress and Birth Outcomes

Physiologic mechanisms have been hypothesized to link maternal stress to maternal and infant health.[20-22] For example, cumulative exposure to stress has been hypothesized to result in accelerated aging, or "weathering," wearing down the body's adaptive systems.[21,22] Weathering in particular was proposed as a source of racial/ethnic disparities in perinatal health, such that the higher stress experienced by African-American women causes their reproductive functioning to deteriorate more rapidly than that of White women.[22] Other possible pathways through which stress can lead to birth outcome disparities include infection[23] and nutrition.[19] In addition, depressive symptoms can result from stressors and may mediate the relationship between stress and birth outcomes by affecting the same neuroendocrine pathways as stress itself.[18,24]

The vast majority of studies assessing the effects on birth outcomes of stress and related factors have relied on measurement during the prenatal period, with mixed results.[19,24-26] A smaller number of studies have examined effects of acute stressors, specific chronic stressors, or depressive symptoms in the reproductive period on birth outcomes.[27-32] Although several of these analyses suggested associations, this work included limitations such as small sample sizes,[27] European cohorts not generalizable to the U.S.,[29] or retrospectively reported preconception measures.[30] Further, none included both acute and chronic stressors or compared racial/ethnic differences for groups besides non-Hispanic Black and non-Hispanic White.

Study Objectives and Hypotheses

To address these gaps in the literature, the objective of this study was to examine the impact of maternal preconception acute stressors (or stressful life events) and preconception chronic stressors (or stressful life conditions) on offspring birth weight and racial/ethnic birth weight disparities, including exploration of preconception depressive symptoms as a pathway through which stress may impact these outcomes. Our hypotheses were as follows: 1) acute and chronic stressors will be inversely associated with birth weight; 2) preconception depressive symptoms will be inversely associated with birth weight and will partially mediate associations between stress and birth weight where such associations exist; 3) the distributions of birth weight and stress will vary by maternal race/ethnicity; and 4) stress will partially explain racial/ethnic differences in birth weight where such differences exist. The conceptual model guiding our hypotheses is shown in Figure 2.1.

METHODS

Data Source and Analytic Sample

This study used contractual data from the National Longitudinal Study of Adolescent Health (Add Health), which began as a nationally representative probability sample of U.S. adolescents in grades 7 through 12 in the 1994-1995 school year.[33] Students were sampled from participating schools to complete an in-home interview. From April to December of 1995, 20,745 Wave I in-home interviews were conducted (79% response rate), accompanied by an interview with a parent or

guardian. Three follow-up in-home interviews of the same panel of respondents have been completed: Wave II in 1996 (88% response rate); Wave III in 2001-2002 with 15,170 respondents aged 18-26 years (77% response rate); and Wave IV in 2007-2008 with 15,701 respondents aged 24-32 years (80% response rate). The Wave IV interviews included a full pregnancy and birth history assessed within each relationship for each respondent. For this study, responses from Wave II were not included because the participants who were in 12th grade in Wave I were not surveyed at Wave II; follow-up of these participants was restored for Waves III and IV. Further information on Add Health is available elsewhere.[33]

The analytic sample consisted of all first or second singleton live births conceived and born between Waves I and IV to non-Hispanic White (hereafter, "White"), Mexican-origin Latina, other-origin Latina, and non-Hispanic Black ("Black") female respondents with valid sampling weights. Only live births were included for comparability of outcome and completeness of reporting, and only singleton births were assessed because the causes and consequences of low birth weight for multiple births differ from those of singleton births.[34] Further, births to women of other racial/ethnic groups were excluded due to small sample size. The total sample sizes were 3512 first births (2035 White, 349 Mexican-origin Latina, 295 other Latina, and 833 Black) and 1901 second births (1072 White, 216 Mexican-origin Latina, 128 other Latina, and 485 Black).

Measures

Preconception acute and chronic stressors were examined as exposures and modeled as indicators of continuous latent factors. A list of acute stressors reported at Waves I and III has been established for Add Health,[35,36] composed of negative events of sudden onset and limited duration occurring within 12 months before the interview. Items reported at both Waves I and III, or for which comparable measures are found at both waves, are listed in Table 2.1. The acute stressors came from Wave I for births conceived before Wave III, and from Wave III for births conceived after Wave III. Items from the prior studies[35,36] that were available on the Wave I interview only, on the Wave III interview only, or pertaining to parenting were excluded from the present study to maintain comparability across waves and across analyses by parity. Additionally, items that loaded poorly onto the acute stressors factor in the confirmatory factor analysis were also excluded.

A list of chronic stressors was identified to reflect occurrences of longer duration pertaining to similar broad domains. These items are listed in Table 2.1. Chronic stressors pertaining to the respondent's family of origin and early experiences came from Wave I for all births regardless of conception date, while the others came from responses before or during the preconception Wave as shown in Table 2.1. Additional items had been considered and rejected if loaded poorly onto the chronic stressors factor in the confirmatory factor analysis.

The outcome, birth weight, was utilized as a continuous measure. Respondent report of birth weight was assessed with the question "How much did {baby's name} weigh at birth? (pounds and ounces)." This value was converted to

grams (where 1 pound = 453.59 grams) to be consistent with clinical measurements of birth weight.[37]

The direct effects of preconception depressive symptoms on birth weight were assessed, as well as depressive symptoms as a potential mediator of associations between preconception stressors and birth weight. Depressive symptoms from the preconception Wave (I or III) were represented by a latent factor with self-reported indicators based on the Center for Epidemiologic Study of Depression scale (CES-D), specifically designed to measure depressive symptoms in general population samples.[38] The 9 questions from the CES-D in common between Waves I and III used here are listed in Table 2.1.

Moreover, variations in the distributions of birth weight and preconception stress by maternal race/ethnicity were determined, and the potential for preconception stress to explain racial/ethnic differences in birth weight was assessed. Race, ethnicity, and Hispanic/Latina background were self-identified by the respondent on the Wave I questionnaire.

Although parity is often treated as a confounder in perinatal epidemiologic analyses, it is likely that the effects of stress on birth weight differ for primiparas vs. multiparas. Therefore, parity was used as an effect modifier here; more specifically, as a stratification variable. The respondent's parity at the time of the birth was constructed from pregnancy outcome variables in the respondent's pregnancy history. All models were run for the first live birth to each respondent. Subsequently, where applicable, they were re-run for the second live birth to each respondent.

Those respondents whose first birth occurred before Wave I and second birth occurred afterward were included in the models for second births.

Proposed confounders may be associated with birth weight and with stress or depression. Respondent's preconception weight status was calculated as body mass index (BMI) from respondent height and weight (self-reported at Wave I or measured at Wave III) at the same Wave from which preconception stressors and depressive symptoms were taken. Respondent's preconception cigarette smoking was calculated from the number of cigarettes reported in the last 30 days on the preconception Wave, and dichotomized into none versus any. Respondent's preconception alcohol consumption was calculated from number of drinks per day and drinks per week in the past year reported at the preconception Wave, and dichotomized as heavy alcohol consumption (yes/no) defined as consuming more than 3 drinks per day or more than 7 drinks per week.[39] Respondent's age at the birth was calculated from date of respondent's birth and the date of the child's birth. The respondent's marital or cohabitation status at the time of birth was categorized as married, cohabiting, or neither married nor cohabiting from respondent report at Wave IV. The time between the preconception interview and the birth was calculated by subtracting the date of the Wave I or Wave III interview from the infant's birth date as reported at Wave IV.

Statistical Analyses

First, univariate distributions and bivariate associations were examined using Stata v.12 (Stata Corp, College Station, TX). This was followed by a confirmatory

factor analysis to generate factor scores. Based on the findings, adjustments to the measurement of the latent variables were made. Specifically, potential indicators that did not load onto the factors were removed and errors were correlated among the variables as suggested by modification indices with theoretical justification (e.g., among the three neighborhood indicators of the chronic stressors factor). This confirmatory factor analysis was conducted using Mplus[40] software v.7 and accounted for categorical variables and other non-normality in the data using weighted least squares estimation.[41]

Factor scores were exported to Stata and linear regressions were used to test the study hypotheses, generating risk differences with 95% confidence intervals (CIs). Thus, coefficients represent the change in grams of birth weight for a one-unit change in each predictor. Sensitivity analyses were run as logistic regression models with low birth weight (yes/no) as the outcome and did not appreciably change the findings. Thus, only the results with a continuous outcome are presented here. Mediation hypotheses were tested using the Baron and Kenny criteria:[42] 1) the main exposure must be significantly associated with the outcome; 2) the proposed mediator must be significantly associated with both the exposure and the outcome; and 3) a substantial change must occur in the point estimate for the exposure after including the mediator in the model. All analyses included sampling weights and cluster variables to account for the complex survey sampling design of Add Health.

RESULTS

Sample Characteristics

Descriptive characteristics of the sample by parity are shown in Table 2.1. In each of the two subsamples, approximately two-thirds of the mothers were White and one in five were Black. Among the Latina mothers, two-thirds were of Mexican origin and one-third were of other origin. Prevalence of each acute stressor was fairly low, with the exception of having forgone needed medical care in the past year. In contrast, chronic stressors were more prevalent. The distributions of responses to the CES-D questions varied across symptoms; “(not) feeling just as good as others” was most prevalent symptom while “feeling disliked” was least prevalent. Mean preconception BMI was 23.6 before first births and 25.3 before second births. Preconception smoking and heavy drinking were prevalent. The mean age at first birth was 22 and at second birth was 24, and the proportions of women who were married and cohabiting increased for second births compared to first births. The average time between the preconception interview and birth was over 4 years for each birth.

Preconception Stress and Depression

Fit indices demonstrated that the confirmatory factor analysis models for each parity subsample fit well (first births: comparative fit index [CFI]=0.954, Tucker-Lewis index [TLI]=0.951, root mean square error of approximation [RMSEA]=0.015 with 90% CI:0.014, 0.017); second births: CFI=0.958, TLI=0.954, RMSEA=0.015 with

90% CI:0.013, 0.017); with good fit defined as CFI and TLI>0.95 and RMSEA<0.05).[43] All correlations among acute and chronic stressors and depressive symptoms were significant (Table 2.2). Before and after controlling for confounders, chronic stressors were inversely associated with birth weight for both first and second births (Table 2.3). Neither acute stressors nor depressive symptoms were significantly associated with birth weight for either parity subsample.

Maternal Race/Ethnicity

Mexican-origin Latinas, other Latinas, and Black women had higher factor scores than White women for both acute and chronic stressors in both the first and second birth subsamples (Table 2.4). In addition, first births to women in all three of these racial/ethnic groups had significantly lower birth weights than White women, while only Black women had significantly lower birth weights for their second births (Table 2.5, Model 1). After including chronic stressors in the models, the magnitudes of the coefficients for each racial/ethnic group decreased, and the coefficient for first births to other-origin Latinas was no longer significant (Table 2.5, Model 2). Chronic stressors remained significantly associated with birth weight for first but not for second births in these models. Interaction terms between maternal race/ethnicity and chronic stressors were tested but omitted due to lack of significance. Although acute stressors were not significantly associated with birth weight, models were run testing interactions between maternal race/ethnicity and acute stressors (not shown). The coefficients for the interaction terms were significant for other-origin Latinas and Blacks for second births; therefore, stratified models were run (Table

2.6). Only other-origin Latinas demonstrated a significant inverse association between acute stressors and birth weight in the stratified models, although it was no longer significant after controlling for chronic stressors.

DISCUSSION

We found partial support for our first hypothesis, as preconception chronic stressors but not acute stressors had a statistically significant inverse association with birth weight among both first and second births. Our results for acute stressors were consistent with a study from Central Pennsylvania,[27] which demonstrated a negative but nonsignificant effect of high preconception psychosocial hassles on birth weight. In addition, our findings for chronic stressors were consistent with studies of low birth weight or a related outcome, preterm birth, suggesting that stressful life conditions in adolescence such as neighborhood and family disadvantage[28] and family structure disruptions or contact with social services[29] affect perinatal outcomes.

We did not find support for our second hypothesis, as preconception depressive symptoms were not significantly associated with birth weight and therefore did not meet the second Baron and Kenny criterion[42] for mediation between stress and birth weight. The lack of association in our study was consistent with two studies finding no relationship between preconception depressive symptoms and preterm birth[30,31] but differed from one analysis of the Coronary

Artery Risk Development in Young Adults (CARDIA) study in which higher depressive symptoms slightly increased the odds of preterm birth.[32]

Our third hypothesis was supported, as women in all minority racial/ethnic groups had higher scores for acute and chronic stressors and lower offspring birth weights for first births than White women. Additionally, Black women had lower birth weights for their second births. Our findings for racial/ethnic differences in stress and for the Black-White disparity in birth weight were consistent with expectations.[5,14] Although Mexican-origin Latinas have similar rates of low birth weight as non-Hispanic White women,[4] our use of a continuous birth weight measure allowed detection of more subtle differences in birth weight distribution. Birth weight distributions vary across Latina subgroups,[44] and our data source allowed us to assess effects separately for women of Mexican origin vs. all other origins. Our finding that the relationship between acute stressors in the interconception period and birth weight was moderated by race/ethnicity, such that acute stressors impacted second births for other-origin Latinas, was new to the literature, suggests a weathering role of acute stressors, and merits further investigation.

Furthermore, we found support for our fourth hypothesis, as chronic stressors partially mediated the associations between maternal race/ethnicity and birth weight. This finding that a portion of the racial/ethnic disparities in birth weight is explained by chronic stressors accumulating through the reproductive period is comparable to results of studies evaluating the weathering hypothesis using age as a marker for cumulative social stressors.[21,45-50] Although most evaluations of the weathering

hypothesis have addressed the Black-White disparity in birth outcomes,[21,45-48] extensions of this hypothesis to Mexican-origin women found no evidence of weathering.[49,50] Our findings for Latina mothers contrast with these studies, suggesting that weathering among both Mexican- and other-origin Latinas should not be discounted but instead examined longitudinally in national samples.

Limitations

Limitations of the study include those inherent in using secondary data. All measures are based on self-report, and may be recalled inaccurately or reflect social desirability. Information is limited to that collected in the interviews, and may not reflect the complete stress history of each participant. Of particular relevance, the Add Health data are missing preconception measures of racism and discrimination which may represent the most important elements of stress for racial/ethnic disparities in birth outcomes.[51] In the absence of these measures, however, the effects we found for stress are likely to be conservative estimates.

In addition, the dataset is limited in its coverage of pregnancy information. Birth weight assessment is reliant on maternal recall rather than clinical report, although this method has been validated in other data sources.[52-55] In addition, the distribution of birth weights in Add Health is comparable to those in birth certificates.[2] Further, the Add Health data also contain no measures of prenatal stress. Therefore, we cannot evaluate whether preconception stress operates independently of or is mediated through prenatal stress. Similarly, we could not

examine other potential pathways, such as perinatal infections or gestational weight gain, through which stress could affect birth weight.

An additional limitation of this study is that only births occurring to women up to 32 years of age could be examined. Although this age range represents the majority of births in the U.S.,[2] the timing of birth may be associated with exposure to acute or chronic stressors.[56] This study ameliorates this limitation somewhat by controlling for time between the interview and the birth, but the results likely are not generalizable to births at older ages.

Conclusions

Despite these limitations, the study contains a number of strengths. To our knowledge, it is the first to apply prospective measures of both acute and chronic stressors to the study of preconception stress and birth weight, and to evaluate disparities in preconception stress and birth weight across multiple racial/ethnic groups in a diverse, nationally representative sample. Results of the study suggest that broad social policies mitigating stressful conditions throughout the life course will be needed to improve birth weights and decrease birth weight disparities. In addition, our results provide support for inclusion of Latinas in studies of preconception stress and disparities, and for preconception and interconception interventions tailored to the needs of women in different racial/ethnic groups. For example, our findings suggest that Latinas of ethnic origins outside of Mexico may benefit particularly from interventions between conceptions to protect their offspring from the impact of acute stressors. Additional research is needed to confirm this

finding and to determine the optimal content of such interventions, which could include improved access to culturally competent health services and improvements in community safety.

FIGURES AND TABLES

Figure 2.1. Conceptual Model Depicting Hypothesized Relationships among Maternal Race/Ethnicity, Preconception Stress, Depression, and Offspring Birth Weight, National Longitudinal Study of Adolescent Health, 1994-2008.

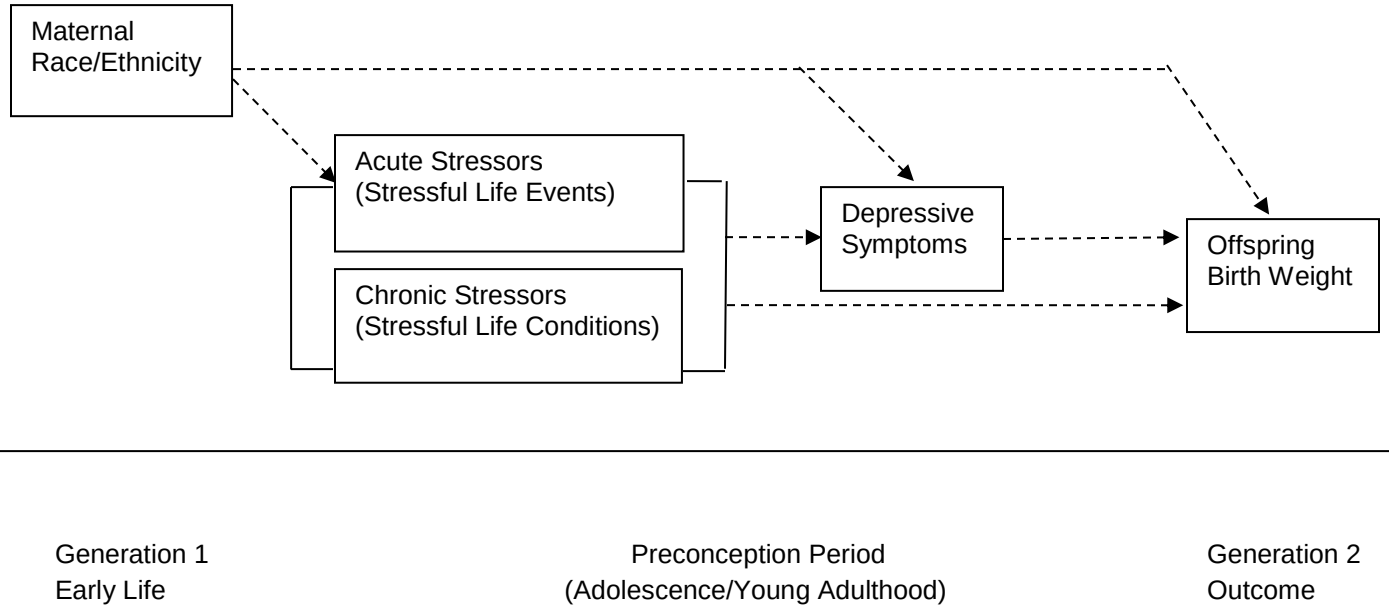

Table 2.1. Characteristics of the Study Population by Parity, National Longitudinal Study of Adolescent Health, 1994-2008.

Characteristic	First Births (n = 3512)	Second Births (n = 1901)
Birth weight, mean (SE), grams	3271 (15)	3320 (17)
Maternal race/ethnicity, %		
Non-Hispanic White	69.7	67.1
Mexican-origin Latina	8.3	8.6
Other-origin Latina	4.9	4.1
Non-Hispanic Black	17.1	20.3
Acute Stressors[a]		
Death of parent, %	1.5	1.6
Friend committed suicide, %	3.9	3.4
Relative committed suicide, %	1.3	1.6
Suicide attempt, %	5.1	4.0
Forgone medical care, %	23.0	23.0
Saw violence, %	8.8	7.9
Threatened by knife or gun, %	6.6	5.9
Threatened other with knife or gun, %	2.4	2.2
Shot by or shot someone, %	1.2	1.0
Stabbed, %	2.4	1.9
Jumped, %	5.6	5.2
Injured in a physical fight, %	5.1	5.5
Hurt other in a physical fight, %	10.1	7.1
Chronic Stressors		
Low parent educational attainment,[b] %	68.2	71.3
Parent received public assistance,[c] %	37.3	39.7
Low parent income,[b] %	31.5	35.8
Parent could not pay bills,[b] %	16.6	18.2
Low respondent educational attainment,[a] %	18.3	22.3
No health insurance,[d] %	19.4	28.0
English as a second language,[b] %	6.4	6.0
Not born a U.S. citizen,[b] %	3.2	3.0
Living without either bioparent,[b] %	51.2	56.3
Low neighborhood household education,[d] %	16.3	20.7
High neighborhood poverty,[d] %	31.4	40.4
High neighborhood unemployment,[d] %	29.9	38.3
Depressive Symptoms[a]		

Feeling bothered by things that don't usually bother, %		
Never or rarely	52.1	47.0
Sometimes	36.0	40.2
A lot of the time	8.7	9.5
Most or all of the time	2.9	3.3
Feeling unable to shake off the blues, %		
Never or rarely	65.2	65.8
Sometimes	23.2	23.2
A lot of the time	7.9	7.7
Most or all of the time	3.5	3.2
Feeling just as good as others, %		
Never or rarely	10.3	9.3
Sometimes	21.7	20.3
A lot of the time	29.2	24.7
Most or all of the time	38.5	45.5
Having trouble keeping mind on activities, %		
Never or rarely	41.5	45.1
Sometimes	41.9	39.8
A lot of the time	12.7	12.1
Most or all of the time	3.7	3.0
Feeling depressed, %		
Never or rarely	57.6	59.0
Sometimes	30.4	28.5
A lot of the time	8.2	8.7
Most or all of the time	3.4	3.8
Feeling too tired for activities, %		
Never or rarely	43.1	39.5
Sometimes	42.8	44.0
A lot of the time	10.6	11.5
Most or all of the time	3.4	4.9
Enjoying life, %		
Never or rarely	4.0	3.8
Sometimes	19.4	17.7
A lot of the time	28.6	29.4
Most or all of the time	48.0	49.1
Feeling sad, %		
Never or rarely	47.5	44.6
Sometimes	42.1	44.6
A lot of the time	7.1	7.7
Most or all of the time	3.1	3.1
Feeling disliked, %		
Never or rarely	70.2	70.3
Sometimes	24.2	24.7
A lot of the time	4.0	3.3
Most or all of the time	1.4	1.6
Confounders		
Preconception BMI, mean (SE), kg/m^2	23.6 (0.1)	25.3 (0.2)
Preconception cigarette smoking, %	34.4	35.5
Preconception heavy drinking, %	29.3	27.9
Age at birth, mean (SE), years	22.6 (0.2)	24.2 (0.2)

Marital/cohabitation status at birth, %		
Married	44.3	54.5
Cohabiting	25.7	29.0
Neither	30.0	16.6
Time between preconception interview and birth, mean (SE), days	1599 (22)	1494 (20)

Abbreviations: %, weighted percent; BMI, body mass index; kg, kilograms; m, meters; SE, standard error of the mean; U.S., United States

[a] Reported at the immediate preconception Wave.

[b] Reported at adolescence (Wave I) for all respondents.

[c] Reported by the parent at Wave I or retrospectively for childhood by the respondent in later Waves.

[d] Reported at any Wave before conception.

Table 2.2. Correlations among Preconception Acute and Chronic Stressors and Depressive Symptoms by Parity, National Longitudinal Study of Adolescent Health, 1994-2008.

	First Births (n=3512)			Second Births (n=1901)		
	Acute Stressors	Chronic Stressors	Depressive Symptoms	Acute Stressors	Chronic Stressors	Depressive Symptoms
Acute Stressors	1[a,b]			1		
Chronic Stressors	0.38	1		0.38	1	
Depressive Symptoms	0.50	0.25	1	0.53	0.25	1

[a] Correlations are based on weighted data.

[b] All correlations are significant $p<0.001$.

Table 2.3. Linear Regression Results for the Effects of Preconception Acute and Chronic Stressors and Depressive Symptoms on Birth Weight by Parity, National Longitudinal Study of Adolescent Health, 1994-2008.

	First Births (n=3512)		Second Births (n=1901)	
	Unadjusted b[a] (95% CI)	**Adjusted b[a,b] (95% CI)**	**Unadjusted b[a] (95% CI)**	**Adjusted b[a,b] (95% CI)**
Acute Stressors	-45 (-124, 34)	-30 (-118, 54)	-36 (-124, 53)	-20 (-110, 70)
Chronic Stressors	-210 (-291, -130)	-192 (-270, -113)	-181 (-297, -65)	-178 (-313, -43)
Depressive Symptoms	-52 (-125, 22)	-59 (-139, 22)	-52 (-154, 50)	-49 (-153, 56)

Abbreviations: CI, confidence interval.

[a] Coefficients are based on weighted data.

[b] Adjusted for preconception body mass index, cigarette smoking, heavy drinking; age at birth; marital/cohabitation status at birth; and time between preconception interview and birth.

Table 2.4. Linear Regression Results for the Associations between Maternal Race/Ethnicity and Preconception Acute and Chronic Stressors by Parity, National Longitudinal Study of Adolescent Health, 1994-2008.

	First Births (n=3512)		Second Births (n=1901)	
	Acute Stressors Unadjusted b[a] (95% CI)	**Chronic Stressors Unadjusted b[a] (95% CI)**	**Acute Stressors Unadjusted b[a] (95% CI)**	**Chronic Stressors Unadjusted b[a] (95% CI)**
Maternal Race/ Ethnicity: Non-Hispanic White	0 [Referent]	0 [Referent]	0 [Referent]	0 [Referent]
Mexican-origin Latina	0.17 (0.09, 0.24)	0.27 (0.20, 0.35)	0.16 (0.04, 0.28)	0.20 (0.11, 0.28)
Other-origin Latina	0.15 (0.09, 0.21)	0.30 (0.19, 0.41)	0.23 (0.06, 0.40)	0.26 (0.16, 0.37)
Non-Hispanic Black	0.20 (0.15, 0.25)	0.28 (0.24, 0.33)	0.21 (0.15, 0.27)	0.24 (0.19, 0.28)

Abbreviations: CI, confidence interval.

[a] Coefficients are based on weighted data

Table 2.5. Linear Regression Results for the Effects of Maternal Race/Ethnicity and Preconception Chronic Stressors on Birth Weight by Parity, National Longitudinal Study of Adolescent Health, 1994-2008.

	First Births (n=3512)		Second Births (n=1901)	
	Model 1: Adjusted b[a,b] (95% CI)	**Model 2: Adjusted b[a,c] (95% CI)**	**Model 1: Adjusted b[a,b] (95% CI)**	**Model 2: Adjusted b[a,c] (95% CI)**
Maternal Race/Ethnicity				
Non-Hispanic White	0 [Referent]	0 [Referent]	0 [Referent]	0 [Referent]
Mexican-origin Latina	-144 (-246, -42)	-112 (-219, -4)	-124 (-270, 22)	-104 (-253, 46)
Other-origin Latina	-130 (-240, -19)	-86 (-204, 32)	-45 (-179, 89)	-13 (-153, 126)
Non-Hispanic Black	-180 (-253, -106)	-146 (-229, -64)	-171 (-278, -64)	-148 (-261, -34)
Chronic Stressors		-139 (-233, -45)		-124 (-266, 18)
Confounders				
BMI	-2 (-8, 4)	-2 (-8, 4)	11 (6, 17)	12 (6, 17)
Smoking	-47 (-113, 20)	-39 (-106, 28)	-78 (-168, 12)	-75 (-164, 14)
Heavy drinking	91 (29, 152)	90 (28, 152)	29 (-44, 102)	28 (-45, 101)
Age at birth	1 (-10, 12)	-2 (-13, 9)	-13 (-25, -0.4)	-15 (-28, -2)
Marital/cohabitation status at birth				
Married	0 [Referent]	0 [Referent]	0 [Referent]	0 [Referent]
Cohabiting	-79 (-160, 1)	-66 (-146, 13)	-79 (-168, 10)	-67 (-156, 21)
Neither	-35 (-116, 45)	-30 (-110, 50)	-69 (-172, 35)	-58 (-161, 44)
Time to birth	-0.008 (-0.04, 0.03)	-0.008 (-0.04, 0.02)	0.03 (-0.03, 0.1)	0.04 (-0.03, 0.1)

Abbreviations: BMI, body mass index; CI, confidence interval.

[a] Coefficients are based on weighted data.

[b] Adjusted for preconception BMI, cigarette smoking, heavy drinking; age at birth; marital/cohabitation status at birth; and time between preconception interview and birth.

[c] Adjusted for preconception chronic stressors, BMI, cigarette smoking, heavy drinking; age at birth; marital/cohabitation status at birth; and time between preconception interview and birth.

Table 2.6. Linear Regression Results for the Effects of Preconception Acute Stressors on Birth Weight for Second Births by Maternal Race/Ethnicity, National Longitudinal Study of Adolescent Health, 1994-2008.

	Second Births (n=1901)	
	Model 1: Adjusted b[a,b] (95% CI)	**Model 2: Adjusted b[a,c] (95% CI)**
Non-Hispanic White (n=1072)		
Acute Stressors	109 (-15, 233)	154 (5, 303)
Chronic Stressors		-165 (-384, 54)
Confounders		
BMI	9 (2, 16)	9 (2, 17)
Smoking	-128 (-226, -31)	-125 (-222, -28)
Heavy drinking	45 (-52, 141)	40 (-57, 136)
Age at birth	-11 (-26, 4))	-13 (-28, 3)
Marital/cohabitation status at birth		
Married	0 [Referent]	0 [Referent]
Cohabiting	-111 (-221, -1)	-97 (-208, 14)
Neither	36 (-92, 164)	47 (-80, 174)
Time to birth	0.05 (-0.02, 0.1)	0.05 (-0.02, 0.1)
Mexican-origin Latina (n=216)		
Acute Stressors	-53 (-299, 193)	74 (-208, 355)
Chronic Stressors		-346 (-807, 116)
Confounders		
BMI	3 (-23, 29)	7 (-17, 31)
Smoking	69 (-213, 351)	32 (-236, 300)
Heavy drinking	32 (-146, 210)	-4 (-169, 161)
Age at birth	-10 (-49, 30)	-2 (-52, 47)
Marital/cohabitation status at birth		
Married	0 [Referent]	0 [Referent]
Cohabiting	-221 (-438, -4)	-204 (-414, 5)
Neither	-10 (-407, 212)	-74 (-371, 222)
Time to birth	-0.3 (-0.5, -0.1)	-0.4 (-0.6, -0.2)

Other-origin Latina (n=128)		
Acute Stressors	-238 (-460, -16)	-220 (-489, 48)
Chronic Stressors		-58 (-406, 290)
Confounders		
BMI	-8 (-32, 17)	-8 (-32, 17)
Smoking	-19 (-250, 213)	-31 (-262, 201)
Heavy drinking	-69 (-215, 77)	-69 (-218, 80)
Age at birth	-17 (-40, 5)	-18 (-40, 5)
Marital/cohabitation status at birth		
Married	0 [Referent]	0 [Referent]
Cohabiting	-92 (-347, 162)	-88 (-341, 165)
Neither	-402 (-702, -101)	-396 (-693, -99)
Time to birth	0.03 (-0.1, 0.2)	0.03 (-0.1, 0.2)
Non-Hispanic Black (n=485)		
Acute Stressors	-147 (-333, 39)	-106 (-303, 92)
Chronic Stressors		-126 (-380, 128)
Confounders		
BMI	16 (7, 24)	16 (7, 25)
Smoking	121 (-85, 328)	107 (-102, 316)
Heavy drinking	-40 (-220, 141)	-49 (.236, 137)
Age at birth	-12 (-35, 12)	-12 (-35, 12)
Marital/cohabitation status at birth		
Married	0 [Referent]	0 [Referent]
Cohabiting	-11 (-207, 186)	-1 (-201, 199)
Neither	-153 (-324, 23)	-147 (-327, 32)
Time to birth	0.04 (-0.5, 0.1)	0.04 (-0.05, 0.1)

Abbreviations: BMI, body mass index; CI, confidence interval.

[a] Coefficients are based on weighted data.

[b] Adjusted for preconception BMI, cigarette smoking, heavy drinking; age at birth; marital/cohabitation status at birth; and time between preconception interview and birth.

[c] Adjusted for preconception chronic stressors, BMI, cigarette smoking, heavy drinking; age at birth; marital/cohabitation status at birth; and time between preconception interview and birth.

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CHAPTER 3

MATERNAL AND OFFSPRING BIRTH WEIGHT CORRESPONDENCE AND PRECONCEPTION STRESS IN A NATIONAL U.S. COHORT

INTRODUCTION

Infant birth weight is a marker of health associated with both short-term and long-term health outcomes.[1] The prevalence of restricted birth weight has been increasing since the 1980s,[2] with racial/ethnic disparities persisting.[3,4] Moreover, birth weight has been demonstrated to be correlated across generations. A systematic review of 12 studies indicated that maternal low birth weight (defined as weight at birth of less than 2500 grams)[1] more than doubles the odds of infant low birth weight.[5] This relationship is believed to have genetic, environmental, and gene-environment interaction (including epigenetic) components;[6] estimates of the environmental contribution range from 14%-53% but specific mechanisms have not been established.[7-9] In addition, higher rates of offspring low birth weight have been recorded for African-American mothers born at low birth weight than for non-Hispanic White mothers born at low birth weight,[10,11] suggesting a possible contributing role of intergenerational transmission of adverse health at birth to the disparity.

Prenatal risk factors for restricted birth weight have provided limited explanation for the increase over time or the disparities in prevalence.[1,12] Therefore, interest in preconception exposures (those occurring in the reproductive period prior to a pregnancy, including those between pregnancies) has increased.[13,14] Preconception stress is one such exposure of interest[15-17] and may include both acute and chronic stressors. Physiologic mechanisms have been hypothesized to connect maternal stress to reproductive function and infant health outcomes. It has been suggested that acute stressful events in early life can program stress reactivity that persists into adulthood to affect childbearing, while cumulative exposure to chronic stress has been hypothesized to result in accelerated aging, or "weathering", wearing down the body's adaptive systems and affecting hormones during pregnancy.[18-22] These neuroendocrine and immunological dysregulation pathways represent possible biologic mechanisms by which stress over the life course can modify the correspondence between maternal and offspring birth weight.

Studies of birth weight transmission in the United States (U.S.) and other developed countries have come mainly from birth registry linkages across generations, and thus are limited in the stressors that can be assessed. For this reason, existing studies have focused on aspects of low socioeconomic status in correlations of low birth weight or birth weight distributions. These suggest that maternal socioeconomic status and support factors at the time of the offspring birth are less important than those existing earlier in her life,[23,24] although findings were mixed.[25,26] None of these studies examined socioeconomic factors in the reproductive period. Given that low socioeconomic status is only one aspect of

chronic stress, correlated to other concurrent and future stressors,[27] stress mechanisms have been hypothesized to play a role in the persistence of adverse health at birth across generations.[23]

To fill the gaps in the literature, the purpose of this study was to examine the distributions of birth weight across generations in a national cohort, including the role of preconception acute and chronic stressors in the intergenerational transmission of birth weight. Our hypotheses were as follows: 1) maternal birth weight will be positively associated with offspring birth weight; 2) the distributions of maternal and offspring birth weight will vary by maternal race/ethnicity; 3) maternal birth weight will partially explain racial/ethnic differences in offspring birth weight where such differences exist; and 4) the effect of maternal birth weight on offspring birth weight will be modified by preconception stress, such that correspondence will be stronger for mothers with higher stress. The conceptual model guiding our hypotheses is shown in Figure 3.1.

METHODS

Data Source and Analytic Sample

This study used contractual data from the National Longitudinal Study of Adolescent Health (Add Health), which began as a nationally representative probability sample of U.S. adolescents in grades 7 through 12 in the 1994-1995 school year.[28] From April to December of 1995, 20,745 students completed the Wave I in-home interview (79% response rate), accompanied by an interview with a

parent or guardian. Three additional in-home interviews of the same panel of respondents have been completed: Wave II (88% response rate) in 1996, approximately one year after Wave I; Wave III in 2001-2002 with 15,170 respondents aged 18-26 years (77% response rate); and Wave IV in 2007-2008 with 15,701 respondents aged 24-32 years (80% response rate). The Wave IV interviews included a full pregnancy and birth history assessed within each relationship for each respondent. For this study, responses from Wave II were not included because the participants who were in 12th grade in Wave I were not surveyed at Wave II; follow-up to these participants was restored for Waves III and IV. Further information about Add Health is available elsewhere.[28]

The analytic sample consisted of all first or second singleton live births conceived and born between Waves I and IV to non-Hispanic White (hereafter, "White"), Mexican-origin Latina, other-origin Latina, and non-Hispanic Black ("Black") female respondents with valid sampling weights. Only singleton births were included because the causes and consequences of low birth weight for twins and other multiple births differ from those of singleton births,[29] and only live births were assessed for comparability of outcome and completeness of reporting. Further, births to women of other racial/ethnic groups were excluded due to small sample size. The total sample sizes were 3512 first births (2035 White, 349 Mexican-origin Latina, 295 other Latina, and 833 Black) and 1901 second births (1072 White, 216 Mexican-origin Latina, 128 other Latina, and 485 Black).

Measures

The main effects of maternal respondent birth weight, race/ethnicity, and acute and chronic stressors were examined. Maternal birth weight was reported by the respondent's parent (typically mother) at Wave I with the question "What was {respondent's name}'s birth weight? (pounds and ounces)" and converted to grams (where 1 pound = 453.59 grams) in order to be consistent with clinical measurements of birth weight[30] and to scale the variable.

Respondent race/ethnicity was included to assess differences in the distributions of maternal and offspring birth weight by race/ethnicity, as well as the potential of maternal birth weight to explain racial/ethnic differences in offspring birth weight. Race, ethnicity, and Hispanic/Latina background were self-identified by the respondent on the Wave I questionnaire.

Indicators of acute stress have been used in previous papers[31,32] and were as follows: death of a parent, friend committed suicide, relative committed suicide, respondent attempted suicide, skipped necessary medical care, saw violence, threatened by a knife or gun, threatened someone with a knife or gun, was shot by or shot someone, was stabbed, was jumped, was injured in a physical fight, and hurt someone in a physical fight. These came from Wave I for births conceived prior to Wave III, and from Wave III for births conceived after Wave III (the "preconception Wave"). Indicators of chronic stress pertaining to the respondent's family of origin and early experiences came from Wave I for all births; these were low parent educational attainment, parent received public assistance, low parent income, parent did not have enough money to pay bills, English as a second language, not born a

U.S. citizen, and living without either bioparent. Additional indicators of chronic stress came from the preconception Wave (low respondent educational attainment for age) or report on any Wave before or including the preconception Wave (no health insurance, low neighborhood household education, high neighborhood poverty, high neighborhood unemployment).

The outcome, offspring birth weight, was assessed with the question "How much did {baby's name} weigh at birth? (pounds and ounces)," and converted to grams.

Parity was treated as an effect modifier to determine if the effects differ for primiparae and multiparae, and therefore all models were run separately for first births and for second births. Respondent's parity at the time of birth was constructed from the pregnancy history; respondents whose first birth occurred before Wave I and second birth occurred afterward were included in the models for second births only.

Covariates included as potential confounders of the relationship between preconception stress and offspring birth weight were maternal preconception body mass index (BMI) from respondent height and weight (self-reported at Wave I or measured at Wave III), cigarette smoking dichotomized as any vs. none from the number of cigarettes reported in the last 30 days at the preconception Wave, and alcohol consumption from the number of drinks per day and per week in the past year at the preconception Wave, dichotomized as heavy alcohol consumption yes/no where "heavy" was defined as >3 drinks/day or >7 drinks/week;[33] age at the birth (in years, calculated from respondent and offspring birth dates); marital or cohabitation

status at the birth (married, cohabiting, or neither); and time between the preconception interview and the birth (in days). In addition, for all analyses including maternal birth weight, the respondent's mother's self-reported smoking status at Wave I (grandmaternal smoking, any/none) was included as a proxy of the grandmother's smoking behavior at the time of the respondent's birth.

Analyses

First, univariate distributions and bivariate associations were examined using Stata 12 (Stata Corp, College Station, TX). In prior work (Strutz et al., unpublished manuscript), latent factor scores for acute and chronic stressors were generated in a confirmatory factor analysis using Mplus software[34] v.7. Factor scores were exported to Stata and linear regression was used to test the study hypotheses, generating risk differences with 95% confidence intervals (CIs). Mediation of the effect of race/ethnicity on offspring birth weight by maternal birth weight was tested using the Baron and Kenny criteria:[35] 1) the main exposure must be significantly associated with the outcome; 2) the proposed mediator must be significantly associated with both the exposure and the outcome; and 3) a substantial change must occur in the point estimate for the exposure after including the mediator in the model. In addition, potential modification of the effect of maternal birth weight by acute and chronic stressors was assessed by examining the magnitude and statistical significance of product terms. Because a sizeable proportion of respondents were missing maternal birth weight, missing values were imputed using chained multiple imputation techniques. Results did not differ between the imputed

and unimputed data and thus only the findings for the imputed data are presented here (with the exception of correlation coefficients due to software limitations). All analyses included sampling weights and cluster variables to account for the complex survey sampling design of Add Health.

RESULTS

Sample Characteristics

Descriptive characteristics of the sample by parity are shown in Table 3.1. In each of the two subsamples, the majority of mothers were White, approximately two-thirds of the Latina mothers were of Mexican origin, and approximately one in five mothers overall were Black. Mean maternal and offspring birth weights were similar within and across subsamples, and mean factor scores for acute and chronic stressors were similar across subsamples as well. Mean preconception BMI was 23.6 before first births and 25.3 before second births. Preconception smoking and heavy drinking and grandmaternal smoking were prevalent. Mean ages at first and second births were 22 and 24, respectively, and a higher proportion of women were unmarried or not cohabiting at first birth compared to second birth. The average time between the preconception interview and each birth was over 4 years.

Correlation and Regression Results

Maternal birth weight was positively correlated with offspring birth weight in the full sample and in most race/ethnicity by parity strata as shown in Table 3.2.

Crude associations between maternal race/ethnicity and both maternal and offspring birth weight are shown in Table 3.3. Compared to Whites, both Mexican- and other-origin Latinas had significantly lower offspring weights for first births only, but did not have significant differences in their maternal birth weights in either subsample. In contrast, Black mothers had significantly lower maternal and offspring birth weights in both parity subsamples.

Further results are shown in Table 3.4 for first births and Table 3.5 for second births. First-born offspring birth weights remained lower for the three minority racial/ethnic groups after controlling for potential confounders (Table 3.4, Model 1). Maternal birth weight did not change the coefficient for Mexican-origin Latinas appreciably, slightly strengthened the relationship for other-origin Latinas, and attenuated the coefficient for Blacks (Table 3.4, Model 2). Although chronic stressors but not acute stressors were associated with offspring birth weight when added to the models (Table 3.4, Model 3), neither significantly interacted with maternal birth weight (Table 3.4, Model 4). For second births, the Black-White gap in offspring birth weight persisted after control for confounders (Table 3.5, Model 1) and was somewhat attenuated by maternal birth weight (Table 3.5, Model 2). Neither acute nor chronic stressors were associated with birth weight in these analyses (Table 3.5, Model 3) and did not significantly interact with birth weight (Table 3.5, Model 4).

DISCUSSION

We found support for our first hypothesis. As seen throughout the literature,[5-9,37] maternal birth weight was positively associated with offspring birth weight in this U.S. national cohort. In addition, our second hypothesis was supported. Racial/ethnic disparities in birth weight were evident, particularly for Black women who had significantly lower maternal and offspring birth weight in both parity subsamples as expected.[12] However, Mexican- and other-origin Latinas did not themselves have significantly lower birth weights, and only their first-born offspring did. Birth outcomes for Latinas differ across ethnic background, immigrant generation, and duration of residence in the U.S., with those of Mexican origin in particular experiencing similar rates of low birth weight as Whites[4] and birth weight distributions shifting downward with time in the U.S.[38,39] The Latinas in our sample, all of whom resided in the U.S. in 7th through 12th grade, may differ from more recent immigrants.

We found partial support for our third hypothesis, as maternal birth weight met the Baron and Kenny criteria for mediation for Black women but not for Mexican- or other-origin Latinas. The birth weights of Black women were significantly lower than those of White mothers, and maternal birth weight decreased the magnitudes of the birth weight differences for their first- and second-born offspring. This finding can be compared to studies of transgenerationally linked state vital statistics data. An analysis of data from Illinois demonstrated that maternal low birth weight accounted for a greater percentage of low birth weight offspring among Black mothers than

among White mothers.[10] This was confirmed in a linkage of California birth records, although the effect of maternal birth weight as a continuous measure was similar between Black and White mothers.[24] In contrast, a more recent examination in Virginia found the opposite effect with maternal low birth weight having a greater effect for Whites.[40] These conflicting results may reflect differences in the population composition of these states. Analysis of the linked data for Washington State included Latina mothers and, as in our study, suggested that the lower maternal birth weights for Blacks but not Latinas partially explained differences in offspring birth weights.[41]

Finally, we did not find support for our fourth hypothesis, as neither acute nor chronic preconception stressors modified the effect of maternal on offspring birth weight. Previous work in theCalifornia birth record linkage demonstrated a moderating effect of household income at the mother's birth on birth weight correspondence, such that the relationship between maternal and offspring birth weight was stronger for mothers born into low-income households than for those born into high-income households.[24] However, neighborhood income in early life did not modify birth weight transmission in an analysis of Chicago birth records from the aforementioned Illinois linkage,[25] while a study of national Panel Survey of Income Dynamics (PSID) data demonstrated that grandparent-fixed effect models to control for family socioeconomic status did not fully explain birth weight persistence.[26]

Limitations

Limitations of the study include its reliance on self-reported measures subject to inaccurate recall or biased by social norms. The dataset is limited in its coverage of stress measures and of pregnancy information. For example, birth weight assessment relies on maternal recall. However, this method has been validated in other data sources,[42-45] and distributions of birth weights in Add Health are comparable to those found in vital statistics.[2,46]

Further, the respondents were aged 24-32 when surveyed on their pregnancy histories. Timing of birth, particularly for young mothers, has been associated with socioeconomic status[47] and with exposure to acute and chronic stressors.[48] Therefore, the results are not generalizable to births occurring to women at older ages, who would be expected to have higher socioeconomic status and lower stress exposures, despite our efforts to control for timing effects using age at birth and time between interview and birth.

Finally, birth weight is a composite measure of gestational age and intrauterine growth retardation, and may not be the best representation of maternal and infant health. It is worth noting that the utility of birth weight to explain later morbidity and mortality has been debated.[49-54] Although the associations between birth weight and later health outcomes are likely not causal, birth weight remains a marker of infant health status that is measurable, available in many data sources, and of interest to public health practice.

Strengths and Implications

The study contains a number of strengths as well. To our knowledge, it is the first study to evaluate the role of birth weight correspondence in disparities across multiple racial/ethnic groups in a national sample and to examine the potentially moderating roles of prospectively measured acute and chronic stressors in the reproductive period. Results of the study suggest that the persistence of birth weight across generations is not easily altered by the presence or absence of stressors despite an individual effect of chronic stressors on offspring birth weight. These findings provide some explanation for the difficulty in improving birth weight and mitigating birth weight disparities. However, they also suggest that improvements in birth weight may persist into the next generation as well. Future longitudinal research in families will be needed to examine this supposition.

FIGURES AND TABLES

Figure 3.1. Conceptual Model Depicting Hypothesized Relationships among Maternal Race/Ethnicity, Maternal and Offspring Birth Weight, and Preconception Stress, National Longitudinal Study of Adolescent Health, 1994-2008.

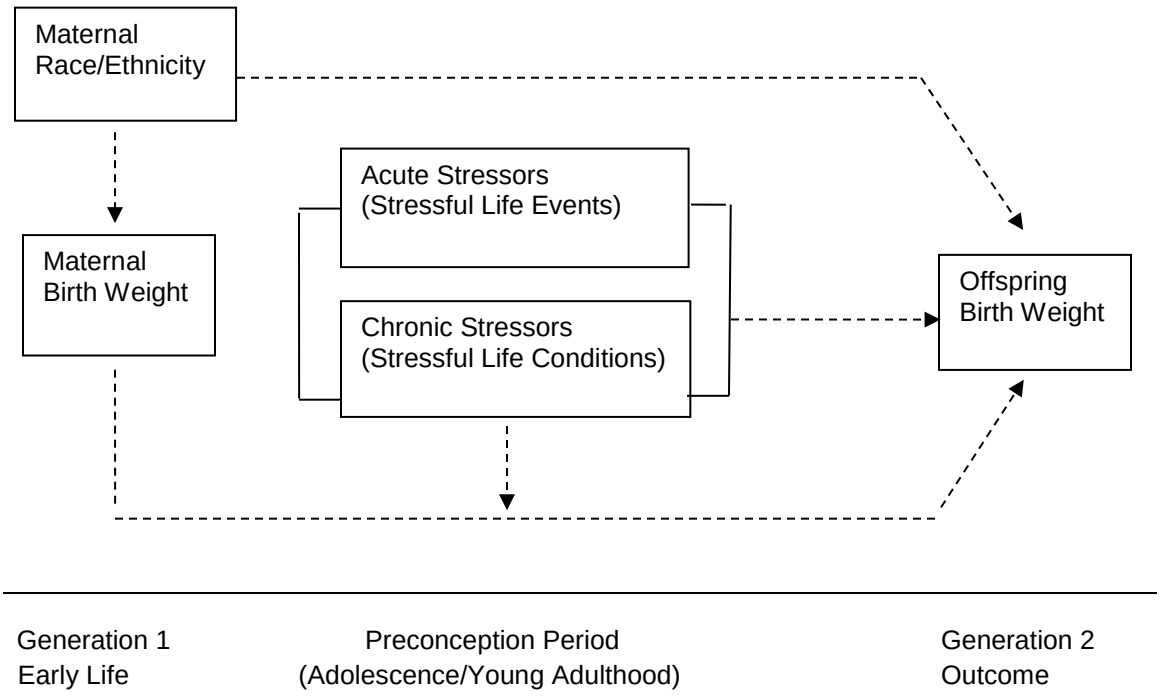

Table 3.1. Characteristics of the Study Population by Parity, National Longitudinal Study of Adolescent Health, 1994-2008.

Characteristic	First Births (n = 3512)	Second Births (n = 1901)
Offspring birth weight, mean (SE), grams	3271 (15)	3320 (17)
Maternal birth weight, mean (SE), grams	3299 (17)	3271 (21)
Maternal race/ethnicity, %		
Non-Hispanic White	69.7	67.1
Mexican-origin Latina	8.3	8.6
Other-origin Latina	4.9	4.1
Non-Hispanic Black	17.1	20.3
Acute stressors, mean (SE)	0.04 (0.01)	0.05 (0.02)
Chronic stressors, mean (SE)	0.02 (0.02)	0.01 (0.02)
Confounders		
Preconception BMI, mean (SE), kg/m^2	23.6 (0.1)	25.3 (0.2)
Preconception cigarette smoking, %	34.6	35.8
Preconception heavy drinking, %	29.5	28.1
Age at birth, mean (SE), years	22.6 (0.2)	24.2 (0.2)
Marital/cohabitation status at birth, %		
Married	44.3	54.5
Cohabiting	25.7	29.0
Neither	30.0	16.6
Time between preconception interview and birth, mean (SE), days	1599.4 (21.6)	1493.8 (20.1)
Grandmaternal cigarette smoking, %	40.2	44.0

Abbreviations: %, weighted percent; BMI, body mass index; kg, kilograms; m, meters; SE, standard error of the mean; U.S., United States

Table 3.2. Correlations between Maternal and Offspring Birth Weight by Maternal Race/Ethnicity and Parity, National Longitudinal Study of Adolescent Health, 1994-2008.

	First Births (n = 3512)	Second Births (n = 1901)
Non-Hispanic White	0.19[a,c]	0.14[c]
Mexican-origin Latina	0.24[c]	0.08
Other-origin Latina	0.26[b]	0.40[c]
Non-Hispanic Black	0.20[c]	0.27[c]
Full sample	0.21[c]	0.18[c]

[a] All correlations are based on weighted data.

[b] Significant at $p<0.05$.

[c] Significant at $p<0.001$.

Table 3.3. Linear Regression Results for the Associations between Maternal Race/Ethnicity and Maternal and Offspring Birth Weight by Parity, National Longitudinal Study of Adolescent Health, 1994-2008.

	First Births (n=3512)		Second Births (n=1901)	
	Maternal Birth Weight Unadjusted b[a] (95% CI)	**Offspring Birth Weight Unadjusted b[a] (95% CI)**	**Maternal Birth Weight Unadjusted b[a] (95% CI)**	**Offspring Birth Weight Unadjusted b[a] (95% CI)**
Maternal Race/Ethnicity				
Non-Hispanic White	0 [Referent]	0 [Referent]	0 [Referent]	0 [Referent]
Mexican-origin Latina	-7 (-119, 105)	-116 (-207, -26)	-8 (-143, 127)	-93 (-204, 18)
Other-origin Latina	73 (-38, 184)	-122 (-218, -27)	92 (-79, 263)	-56 (-190, 78)
Non-Hispanic Black	-216 (-280, -152)	-213 (-280, -146)	-204 (-295, -112)	-168 (-257, -79)

Abbreviations: CI, confidence interval.

[a] Coefficients are based on weighted data.

Table 3.4. Linear Regression Results for the Effects of Maternal Race/Ethnicity, Maternal Birth Weight, and Preconception Stress on Offspring Birth Weight for First Births, National Longitudinal Study of Adolescent Health, 1994-2008.

	Offspring Birth Weight, First Births (n=3512)			
	Model 1: Adjusted b[a,b] (95% CI)	**Model 2: Adjusted b[a,c] (95% CI)**	**Model 3: Adjusted b[a,d] (95% CI)**	**Model 4: Adjusted b[a,e] (95% CI)**
Maternal Race/Ethnicity				
Non-Hispanic White	0 [Referent]	0 [Referent]	0 [Referent]	0 [Referent]
Mexican-origin Latina	-117 (-211, -23)	-118 (-208, -28)	-96 (-189, -3)	-95 (-188, -2)
Other-origin Latina	-111 (-215, -7)	-128 (-234, -21)	-101 (-212, 11)	-100 (-212, 13)
Non-Hispanic Black	-192 (-268, -116)	-152 (-227, -77)	-136 (-218, -54)	-138 (-219, -56)
Maternal birth weight		201 (150, 251)	197 (146, 247)	200 (149, 251)
Acute stressors			92 (-3, 186)	214 (-270, 697)
Chronic stressors			-140 (-241, -40)	-129 (-711, 452)
Maternal birth weight x Acute stressors				-37 (-180, 106)
Maternal birth weight x Chronic stressors				-3 (-175, 168)
Confounders				
BMI	-2 (-8, 4)	-4 (-10, 2)	-4 (-10, 2)	-4 (-10, 2)
Maternal smoking	-46 (-108, 15)	-47 (-108, 14)	-53 (-115, 9)	-54 (-116, 8)
Heavy drinking	88 (30, 146)	79 (19, 139)	68 (10, 127)	69 (10, 127)
Age at birth	0.5 (-10, 11)	-0.1 (-10, 9)	0.3 (-10, 11)	0.2 (-10, 11)
Marital/cohabitation status at birth				
Married	0 [Referent]	0 [Referent]	0 [Referent]	0 [Referent]
Cohabiting	-73 (-151, 4)	-65 (-138, 8)	-59 (-132, 13)	-58 (-131, 14)
Neither	-30 (-107, 47)	-25 (-100, 54)	-23 (-51, 63)	-23 (-98, 53)

Time to birth	-0.004 (-0.04, 0.03)	-0.01 (-0.04, 0.02)	-0.01 (-0.05, 0.02)	-0.01 (-0.05, 0.02)
Grandmaternal smoking		-3 (-60, 54)	6 (-51, 63)	7 (-50, 64)

Abbreviations: BMI, body mass index; CI, confidence interval.

[a] Coefficients are based on weighted data.

[b] Main effect of maternal race/ethnicity adjusted for preconception BMI, cigarette smoking, heavy drinking; age at birth; marital/cohabitation status at birth; and time between preconception interview and birth.

[c] Main effects of maternal race/ethnicity and maternal birth weight adjusted for preconception BMI, cigarette smoking, heavy drinking; age at birth; marital/cohabitation status at birth; time between preconception interview and birth; and grandmaternal smoking.

[d] Main effects of maternal race/ethnicity, maternal birth weight, and preconception acute and chronic stressors adjusted for preconception BMI, cigarette smoking, heavy drinking; age at birth; marital/cohabitation status at birth; time between preconception interview and birth; and grandmaternal smoking.

[e] Main effects of maternal race/ethnicity, maternal birth weight, and preconception acute and chronic stressors with interaction terms adjusted for preconception BMI, cigarette smoking, heavy drinking; age at birth; marital/cohabitation status at birth; time between preconception interview and birth; and grandmaternal smoking.

Table 3.5. Linear Regression Results for the Effects of Maternal Race/Ethnicity, Maternal Birth Weight, and Preconception Stress on Offspring Birth Weight for Second Births, National Longitudinal Study of Adolescent Health, 1994-2008.

	Offspring Birth Weight, Second Births (n=1901)			
	Model 1: Adjusted b[a,b] (95% CI)	**Model 2: Adjusted b[a,c] (95% CI)**	**Model 3: Adjusted b[a,d] (95% CI)**	**Model 4: Adjusted b[a,e] (95% CI)**
Maternal Race/Ethnicity				
Non-Hispanic White	0 [Referent]	0 [Referent]	0 [Referent]	0 [Referent]
Mexican-origin Latina	-114 (-234, 7)	-104 (-225, 17)	-90 (-215, 35)	-88 (-215, 38)
Other-origin Latina	-58 (-193, 76)	-70 (-197, 57)	-52 (-190, 86)	-52 (-191, 86)
Non-Hispanic Black	-178 (-286, -71)	-149 (-256, -42)	-138 (-254, -21)	-136 (-252, -19)
Maternal birth weight		147 (90, 205)	144 (84, 203)	139 (78, 201)
Acute stressors			38 (-66, 142)	-68 (-607, 471)
Chronic stressors			-104 (-270, 62)	-236 (-1025, 552)
Maternal birth weight x Acute stressors				32 (-131, 196)
Maternal birth weight x Chronic stressors				40 (-196, 276)
Confounders				
BMI	11 (5, 17)	9 (3, 15)	9 (3, 15)	9 (3, 15)
Maternal smoking	-66 (-152, 20)	-72 (-158, 15)	-74 (-161, 14)	-73 (-160, 14)
Heavy drinking	22 (-49, 93)	11 (-59, 80)	5 (-65, 76)	4 (-66, 75)
Age at birth	-14 (-26, -2)	-11 (-23, 1)	-11 (-23, 1)	-11 (-23, 1)
Marital/cohabitation status at birth				
Married	0 [Referent]	0 [Referent]	0 [Referent]	0 [Referent]
Cohabiting	-93 (-180, -7)	-85 (-171, 2)	-78 (-164, 8)	-79 (-164, 6)
Neither	-79 (-184, 26)	-74 (-179, 31)	-68 (-172, 35)	-70 (-173, 33)

Time to birth	0.04 (-0.02, 0.1)	0.03 (-0.03, 0.1)	0.03 (-0.03, 0.1)	0.03 (-0.03, 0.1)
Grandmaternal smoking		35 (-40, 110)	41 (-37, 119)	41 (-36, 119)

Abbreviations: BMI, body mass index; CI, confidence interval.

[a] Coefficients are based on weighted data.

[b] Main effect of maternal race/ethnicity adjusted for preconception BMI, cigarette smoking, heavy drinking; age at birth; marital/cohabitation status at birth; and time between preconception interview and birth.

[c] Main effects of maternal race/ethnicity and maternal birth weight adjusted for preconception BMI, cigarette smoking, heavy drinking; age at birth; marital/cohabitation status at birth; time between preconception interview and birth; and grandmaternal smoking.

[d] Main effects of maternal race/ethnicity, maternal birth weight, and preconception acute and chronic stressors adjusted for preconception BMI, cigarette smoking, heavy drinking; age at birth; marital/cohabitation status at birth; time between preconception interview and birth; and grandmaternal smoking.

[e] Main effects of maternal race/ethnicity, maternal birth weight, and preconception acute and chronic stressors with interaction terms adjusted for preconception BMI, cigarette smoking, heavy drinking; age at birth; marital/cohabitation status at birth; time between preconception interview and birth; and grandmaternal smoking.

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CHAPTER 4

CONCLUSIONS

Overview of Findings

This dissertation examined the impact of preconception acute and chronic stressors on offspring birth weight and racial/ethnic birth weight disparities for both first and second births. In addition, it explored a potentially mediating role of the stress-birth weight relationship by depressive symptoms, the potential for maternal birth weight to explain racial/ethnic disparities in offspring birth weight, and a potentially moderating role of stress in maternal-offspring birth weight correspondence. Findings included that chronic but not acute stressors were inversely associated with offspring birth weight, and this relationship was not mediated by depressive symptoms. Chronic stressors partially explained disparities in offspring birth weight for all minority racial/ethnic groups (non-Hispanic Blacks, Mexican-origin Latinas, and other-origin Latinas), while maternal birth weight partially explained the Black-White disparity. Despite individual main effects of both chronic stressors and maternal birth weight on offspring birth weight, neither chronic nor acute stressors modified the effect of maternal on offspring birth weight. A significant interaction was found for acute stressors and racial/ethnic group, such that Latinas of origins outside Mexico had an inverse relationship between acute

stressors and weight of second-born offspring. Aside from this result, most findings were similar across the parity subsamples.

The Use of Add Health to Study Preconception Stress

The shift in emphasis from prenatal exposures to preconception exposures is not new in the maternal and child health (MCH) field,[1] but the effects of preconception exposures on infant health have been underresearched due to limited sources of data.[2] This dissertation utilized data from the National Longitudinal Study of Adolescent Health (Add Health), a cohort not explicitly designed to study preconception or pregnancy but instead general health and development from adolescence through young adulthood.[3] Strengths of this cohort to address preconception exposures include that it is nationally representative and diverse. Furthermore, the large majority of participants were interviewed before any childbearing. Measures of stressors, depressive symptoms, behaviors, and other constructs not included in the present study were reported prospectively in relation to the conceptions and births, although some measures involve a retrospective report of a longer period before the time of the survey.

As elucidated further in the previous chapters, the main limitation of this data for examining preconception health is self-reported measures that may exclude key indicators of stress (particularly racism and discrimination) and pregnancy information. For example, the information on offspring gestational age in the Add Health data appears to be subject to considerable measurement error, based on comparisons to known distributions of gestational age in the population.[4] Therefore,

this study did not adjust for gestational age. This decision was supported by the fact that gestational age is not a cause of the exposures under examination,[5] and by interest in the overall effects of stressors on birth weight[6,7] whether restriction of birth weight is due to preterm birth or to intrauterine growth retardation.

Concordance between the Findings and the Guiding Theories

One theoretical perspective informing this dissertation was life course theory, which, as a public health framework, emphasizes that exposures across an individual's life span impact that individual's health status.[8] As mentioned in the introductory chapter to this dissertation, there are three life course models commonly used in public health:[8-11] 1) the latency model, in which the preconception period represents a critical or sensitive period affecting offspring health regardless of prenatal exposures; 2) the cumulative model, in which exposures measured in the reproductive period represent accumulation of exposures from earlier life; and 3) the pathway model, in which preconception is an intervening period modifying the effects of early life factors on offspring birth weight. The mother's own birth weight and accompanying health at birth would represent a critical period or the beginning of an accumulation or pathway of exposures in these models.

In this study, acute stressors were most consistent with the latency model because these indicators were limited to the 12 months preceding each interview. However, the interviews occurred an average of 4 years before the births, and thus this 12 month period may not have represented the critical period for preconception stress to affect infant birth weight. It is possible that acute stressors occurring closer

to conception have greater impact. Moreover, the latency model could not be fully tested in the present study because the data does not include prenatal measures of stress.

Chronic stressors, on the other hand, were most consistent with the cumulative model, because the indicators measured exposures across a longer duration before the preconception interview. This model was supported by the study findings. The pathway model was tested in the second dissertation paper with both acute and chronic stressors representing an intervening moderator between maternal and offspring birth weight, but this model was not supported. This dissertation represents only a first step towards disentangling the roles of early life and preconception factors in life course models of health.

The other theoretical perspective underlying this dissertation was the stress process model, which posits that social characteristics lead to acute and chronic stressors that affect physical and mental health status,[12-14] hypothesized to account for racial/ethnic disparities in health.[15,16] It should be noted that it is nothing inherent in the social characteristics themselves, but instead how people are treated because of them, that increases stress. Birth weight distributions for non-Hispanic Blacks have been consistently proven to be shifted downward compared to those for non-Hispanic Whites,[17] and this project's findings were no exception. Maternal birth weight and both first- and second-born offspring birth weights were significantly lower for Black mothers than for White mothers. Additionally, the present studies found that both maternal birth weight and chronic stressors individually explained portions of this disparity for offspring birth weights.

In contrast, birth weight distributions for Latinas in the U.S. differ by ethnic origin, immigrant generation, and duration of residence in the U.S.[18,19] Although this is also true for recent Black immigrants and their children,[20] they represent only a small proportion of the Black respondents in this and other national samples. For Latinas in this study, first-born offspring but not second-born offspring or the mothers themselves had significantly lower birth weights than Whites, with chronic stressors explaining a portion of this disparity. This project's similar findings for Mexican- and other-origin Latinas contrast with the literature demonstrating a health advantage for those of Mexican descent.[21] The differences in results between this project and published work could be due to several factors. The other-origin Latina group here is particularly heterogenous, and sample size restrictions will not allow for further distinction between lower-risk and higher-risk groups. Further, this study used full birth weight distributions instead of dichotomizing into low vs. normal birth weight, and was therefore able to detect a more subtle difference in first-born offspring birth weight that may be obscured by a binary measure. Finally, the current sample of Latinas resided in the U.S. in 7th through 12th grades and may differ from Latinas who immigrate at older ages. Regardless, this project provides some evidence for the stress process model as an explanation of racial/ethnic birth weight disparities.

Implications

This study has several implications for future research, practice, and policy. First, it confirms the utility of using existing prospective longitudinal cohorts to study preconception exposures. In a parsimonious funding climate, and with

preconception enrollment presenting logistical challenges for pregnancy cohorts,[22] leveraging existing resources can help MCH researchers and perinatal epidemiologists understand the impact of preconception health on the next generation. Although the findings of this dissertation did not differ greatly between first and second births, the effects of other exposures and interventions may differ by timing. Thus, future work should continue to examine potential differences between the preconception and interconception periods. Additional studies will be needed to test the proposed biological mechanisms[23-26] for how chronic stress gets "under the skin" to result in adverse birth outcomes; the present study found no evidence to support that depressive symptoms are a pathway.

Moreover, while the results of the dissertation provide continued support for reducing maternal and infant health disparities for Black women, they also indicate that Latinas, who now account for almost one-quarter of all U.S. births,[27] should not be overlooked in this line of research and practice. Preconception and interconception interventions must be tailored to the needs of women in different racial/ethnic groups. Chronic stressors impacted offspring birth weight for all included minority groups. These stressors reflect social conditions that are difficult to improve, necessitating policies beyond those of the health sector to enact real change. Furthermore, offspring birth weight was tightly linked to maternal birth weight, and therefore improvements made in one generation may not be visible until the next generation. Although no one factor, including maternal birth weight or chronic stressors, has a large effect on birth weight, the findings of this dissertation demonstrate that history affects health. To the extent to which policymakers can

adopt a longer-term view, studies such as this one build the evidence base for enacting social safety nets that buffer the effects of chronic stress and promote health equity for women and their infants.

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