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A Multilevel Analysis of Prenatal Maternal Stress and Birth Weight

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Not long ago, the study of pregnancy and birth was the sole domain of the field of medicine and the allied health professions. In recent decades, psychosocial and sociocultural factors have been increasingly incorporated into theory and research on pregnancy in order to improve our scientific understanding of the factors that elevate or reduce risk. When we began our work on pregnancy in the 1980s, it was recognized that even a complete analysis of medical risk factors did not account for much of the variation in rates of such adverse birth outcomes as preterm birth or low birth weight. However, approaches to the identification of psychosocial risk factors were still evolving in rigor and acceptance by biomedical scientists. In the intervening decades, psychosocial and interdisciplinary approaches to the study of pregnancy and birth have evolved considerably; they are not only accepted today but often welcomed. Many behavioral, sociological, cultural, and biomedical scientists now work together in collaboration to understand the complex interplay of multiple levels and types of concepts that contribute to maternal and child outcomes. Moreover, biopsychosocial integration is producing exciting findings and important insights that promise to improve future health outcomes for pregnant women and their offspring.

In this chapter, we provide brief background on the epidemiology of birth outcomes, especially focusing on low birth weight, and we outline a multilevel approach to understanding the etiology of low birth weight. We review the evidence regarding various types of prenatal maternal stress in the prediction of birth weight, and we focus on the theory and evidence on three modifiers of these effects—namely, medical risk, race and ethnicity, and social support. We explain the major mechanisms at physiological and behavioral levels, focusing in detail on research on smoking, physical activity, nutrition, and healthy lifestyles. Thereafter, there is a discussion of integrative models and we present a schematic diagram differing from others in its greater detail of psychological processes. Our conclusions offer suggestions for studying stress better in order to understand low birth weight. We propose studying health behaviors further as a set of mechanisms, and argue that we are not yet ready to intervene to reduce low birth weight with psychosocial approaches. A subtheme throughout the chapter is the importance of race and ethnicity as they pertain to disparities in birth outcomes and their understanding.

BACKGROUND ON BIRTH OUTCOMES

We use the term *adverse birth outcomes* broadly here to refer to preterm birth (PTB), low birth weight (LBW), infant mortality and related complications for the mother or infant at birth or shortly after. In this chapter, we build upon our earlier one entitled "Stress Processes in Pregnancy and Birth" (Dunkel Schetter, Gurung, Lobel, & Wadhwa, 2001), which appeared in the first edition of this handbook. Here, we focus on a broader range of issues but on only one outcome—LBW (low birth weight)—while still emphasizing stress, a primary area of our individual programs of research. Other recent reviews exist on stress and PTB (Dunkel Schetter & Glynn, 2010; Hobel, Goldstein, & Barrett, 2008), but none of which we are aware on stress and LBW. These two outcomes are objectively measured, and national and international data on population rates are published regularly. Although PTB and LBW are moderately correlated, the complex and multiple etiological pathways to them are increasingly distinguishable (Dunkel Schetter, 2010). Furthermore, disentangling these two outcomes and their precursors is critical to moving forward on national agendas to reduce rates of PTB, LBW, and infant mortality in high-risk populations.

Rates and Significance of LBW

LBW is defined as infant weight at birth of less than 2,500 grams (5 pounds, 8 ounces). Fetal growth is the trajectory of growth, usually estimated through ultrasound examinations over the course of gestation. At 8.3%, the rate of LBW in the United States in 2006 was the highest in four decades (Martin, Hamilton, & Sutton, 2009) and higher than the rate for other industrialized countries. This high LBW rate reflects, in part, the growing number of multiple births in the United States (e.g., twins, triplets), more than half of which are LBW. However, the rate of LBW among singleton births is also considered high at 6.5% nationwide and has risen by approximately 10% in the last several years (Martin et al., 2009).

LBW co-occurs with PTB in one half to two thirds of cases. Babies born early are likely to weigh less; yet many babies born at full term (37 to 40 weeks) are LBW. Ultrasound and newborn exams are used to differentiate premature LBW from more mature growth-retarded LBW infants. Intrauterine growth restriction (IUGR) refers to a fetus not reaching its genetically predetermined size, based on population standards, usually as a result of pathological changes in the placenta (Marsal, 2002). A newborn that is *small for gestational age* (SGA) is below the 10th percentile of weight for gestational age at birth, a characteristic sometimes also referred to as *fetal growth retardation*, or FGR. These infants are at higher risk of abnormalities and risk of neonatal death compared to higher weight babies, and those babies below the fifth percentile are especially at risk of these outcomes.

Both underweight and preterm infants are at increased risk of infant mortality and other problems in infancy and childhood, including respiratory illness, impaired growth, cognitive deficits, and longer term neurological problems that may require special education (Hack, Klein, & Taylor, 1995; Newnham, 1998; Thompson et al., 1997). Research also suggests that LBW infants are at increased risk in adulthood for cardiovascular disease, noninsulin-dependent diabetes, obesity, and psychiatric conditions (Behrman & Stith Butler, 2007).

Studies of LBW examine both the clinically defined dichotomous outcome (i.e., normal vs. low birth weight) and the continuous variable of weight in grams at birth (ranging from 500 grams or less to more than 3,000 grams). LBW is further subdivided into *very low birth weight* (VLBW), defined as less than 1,500 grams at birth, and *extremely low birth weight* (ELBW), which is less than 1,000 grams at birth. Very large samples are needed to provide the statistical power to assess predictors of the categorical variables of LBW, VLBW, or ELBW; whereas studies with smaller samples can examine the correlates of the continuous birth weight variable with sufficient power. Both are valuable in understanding the effects of such psychosocial factors as stress. In fact, there is evidence that some child and adult health outcomes are influenced by small gradations in birth weight within the normal birth weight range (Breslau, Chilcoat, DelDotto, Andreski, & Brown, 1996; Matte, Bresnahan, Begg, & Susser, 2001; Richards, Hardy, Kuh, & Wadsworth, 2001; Sorensen et al., 1997). In addition, the effects of psychosocial variables on birth weight are often linear. Therefore, there is more than adequate justification, both empirically and clinically, for studying both birth weight variables. We believe that a focus on the etiology of LBW from a multilevel and, especially, psychosocial perspective is very valuable at this juncture.

Racial and Ethnic Disparities in LBW

LBW occurs disproportionately among ethnic groups within the United States. African Americans have approximately twice the LBW rate (14.0%) compared to non-Hispanic Whites (7.3%) and Hispanics (7.0%) and have a higher rate of LBW than other groups as well (Martin et al., 2009). African Americans also have higher rates of PTB. However, African American infants have a survival advantage at lower birth weights and younger gestational ages compared to European American infants (Alexander, Tompkins, & Hulsey, 2000; Wilcox & Russell, 1990). Furthermore, there is evidence suggesting that African American fetuses mature faster than European American fetuses (e.g., Alexander, Hulsey, Robillard, De Caunes, & Papiernik, 1994; Robillard et al., 1994). Thus, race-specific analyses are essential, and race-specific criteria for defining LBW may be needed.

In addition to the disparity between African Americans and other groups in LBW, there are differences among ethnic subgroups of Latinas. For instance, Puerto Rican women have higher rates of LBW (10% in 2003) than do other Latinas, and there is considerable variability within the Hispanic population as a function of socioeconomic status (SES) and acculturation. Campos and colleagues (2008) discussed two important and counterintuitive patterns within the Latina population that are relevant to studying pregnancy. First, Latino or Hispanic individuals of lower SES have better health outcomes in general, including pregnancy outcomes, than do Latinos of higher SES (Abraido-Lanza, Dohrenwend, Ng-Mak, & Turner, 1999; Hessol & Fuentes-Affick, 2000). Second, more acculturated Latina women often have higher rates of adverse pregnancy outcomes than do less acculturated women. For example, in a study of nearly 1,100 poor minority women, two thirds of whom were of Mexican origin or descent and all of whom were having their first child, more acculturated women had lower birth weight infants and higher stress and substance use (Zambrana, Scrimshaw, Collins, & Dunkel-Schetter, 1997). They were also less enthusiastic about the pregnancy compared to women who were less acculturated.

In a similar study of a cohort of 1,064 Latina women (Campos, Dunkel Schetter, Walsh, & Schenker, 2007), two components of acculturation were distinguished, one involving use of English and contact with Anglos (labeled *Anglo orientation*), and the other involving immersion in Mexican and Spanish-speaking culture (labeled *Mexican orientation*). Although there were no associations of Mexican orientation with birth weight in this study, higher Anglo orientation was associated with lower infant birth weight (OR = 1.28, $p < .01$). That is, Latina pregnant women who reported that they associated more often with Anglos and spoke, wrote, and thought more in English had lower birth weight babies, controlling for gestational age, compared to those who were lower on this factor.

Both of these studies suggest that increased involvement in Anglo culture poses pregnancy risk. Further consistent evidence comes from a recent study on racial and ethnic disparities in LBW in 2,412 unmarried mothers classified as non-Hispanic Black, non-Hispanic White, U.S.-born Mexican origin, and foreign-born Mexican origin, which found a Mexican-origin advantage in birth weight that was explained, in part, by better prenatal health and behaviors in Mexican-born women (Reichman, Hamilton, Hummer, & Padilla, 2008). Researchers are just beginning to understand the sometimes paradoxical differences among Latino subgroups in pregnancy. It appears that the Latina pregnant women most at risk may be those less likely to have cultural resilience factors such as larger social networks and stronger support, or highly valued family connections and mutual

obligations (i.e., *familialism*), and they are less likely to highly value pregnancy and childbearing (Abdou et al., 2010; Campos et al., 2008; Campos et al, 2007).

Paradoxical findings also occur for African American women of higher SES. This group does not experience the lower rates of adverse birth outcomes that European American women of similar SES do (e.g., Din-Dzietham & Hertz-Picciotto, 1998). This may be partially because African American women with greater education or higher job status are less connected to a supportive community and more exposed to racism in ethnically integrated neighborhoods and places of work (Buka, Brennan, Rich-Edwards, Raudenbush, & Earls, 2003; Clark, Anderson, Clark, & Williams, 1999). The observation that higher SES Black women in the United States have poorer pregnancy outcomes on average than do poor White women points to critical interactions of SES, race, and ethnicity in pregnancy risk.

MULTILEVEL ANALYSES OF BIRTH OUTCOMES

Clearly, any comprehensive and useful understanding of LBW will involve thoughtful and detailed behavioral and sociocultural analyses, reaffirming that a complete understanding of pregnancy and birth outcomes requires interdisciplinary approaches. Many processes influence the rate of growth of a fetus and resultant birth weight of a baby. Table 19.1 contains a summary of a number of risk and resilience factors at each level that are known or commonly hypothesized to influence birth weight. Individual-level factors, interpersonal factors, sociocultural factors, and community factors all figure into the comprehensive prediction of LBW. Within these categories, there are further subcategories of variables that influence birth weight. These levels of analysis and specific factors have been examined in past research, although mostly in isolation rather than conjointly. There is increasing recognition that the prediction of birth outcomes is multifactorial, involving joint and interactive effects of many variables at several different levels. Thus, the taxonomy depicted in Table 19.1 may help to advance a comprehensive understanding of the processes that influence birth outcomes.

To expand on the multiple influences on birth outcomes, we note that medical, behavioral, and psychological factors all exert influence at the individual level. Medical risk factors include maternal history of disease, obstetric and gynecological risk factors including complications in prior pregnancies, and emerging symptoms and conditions in the present pregnancy. Behavioral risk factors of primary interest at the individual level include preconception and prenatal smoking, drug and alcohol use, nutrition, physical activity and fitness, and health care. Psychological factors at the individual level include exposure to stressors of many types, emotional and cognitive responses to stress, and personal resources that directly influence birth weight or modify effects of stressors on birth weight. In this chapter, we focus in greater detail on this level, that is, psychological factors and their incorporation into integrative predictive models, because this level involves areas of our unique past contributions and topics especially relevant to the field of health psychology.

At the interpersonal level, social integration, networks, support, and close relationship quantity and quality (e.g., with partner or baby's father) are all key factors, along with conflict and interpersonal violence in close relationships. Group- and sociocultural-level factors include race, ethnicity, nativity, acculturation, socioeconomic status, and social position. Community- and societal-level factors include community stressors and resources, neighborhood characteristics, and such institutional characteristics as systemic discrimination.

As can be seen in Table 19.1, it would be impossible to capture all these influences in a single study. However, in sum total, they represent the large array of established or likely influences on rate of fetal growth and resultant infant birth weight. Figure 19.1 is a simple schematic diagram of these multilevel categories of factors on birth weight. It depicts them as direct and equally important causal influences, although they may actually have different degrees of importance and, in some cases, are likely to interact in their effects.

TABLE 19.1
Multilevel Predictors, Mediators, and Moderators of Birth Weight

Level of Analysis	Risk and Resilience Factors
Individual: *Biological/medical*	Maternal medical history/risk factors
	Maternal pregnancy risk conditions and symptoms
	Preconception dysregulated HPA, immune, or other systems
	Genetic factors
Individual: *Behavioral*	Smoking in pregnancy
	Drug use in pregnancy
	Alcohol use in pregnancy
	Physical fitness
	Nutrition in pregnancy
	Vitamin use in pregnancy
	Health behaviors preconception
	Regular and preventive health care (immunizations, dental care)
	Prenatal care utilization
Individual: *Psychological*	Stress and emotion
	Major life events
	State anxiety
	Pregnancy anxiety/pregnancy distress
	Chronic stress
	Traumatic events
	Work stress
	Perceived racism
	Mental health indicators
	Depressed mood
	Anxiety disorders
	PTSD
	Personal resources
	Optimism
	Mastery
	Self-esteem
	Perceived available support
	Attitudes toward pregnancy
	Intendedness
	Coping style
	Approach/avoidance
	Positive emotions
	Religiosity/spirituality
Interpersonal: *Dyadic/familial/relational*	Social support received
	Social network properties
	Relationship characteristics of baby's father
	Family composition
	Quality of relationships
	Responsiveness
	Marital satisfaction
	Intergenerational factors
	Interpersonal violence (IPV)
Group: *Sociocultural*	Race/ethnicity
	Nativity
	Acculturation

continued

TABLE 19.1 (Continued)
Multilevel Predictors, Mediators, and Moderators of Birth Weight

Level of Analysis	Risk and Resilience Factors
	SES/social class
	Familism
	Collectivism
Community: *Societal*	Community stressors (e.g., noise, traffic, toxins, violence, pollution, crime, density of housing)
	Community cohesion, collectivism
	Neighborhood poverty
	Residential segregation
	Institutional discrimination
	Cultural norms and values

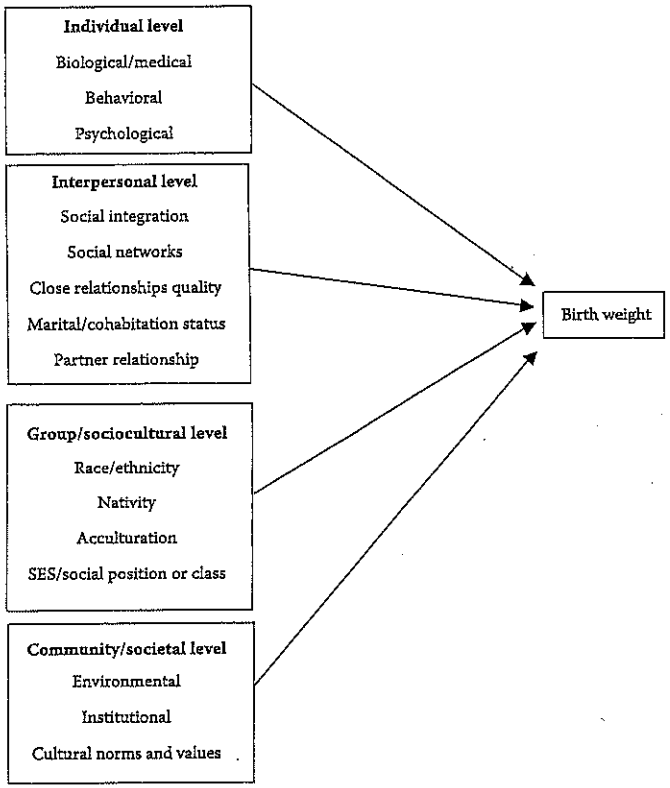

FIGURE 19.1 Multilevel direct predictors of birth weight.

Evidence Regarding Prenatal Maternal Stress and Birth Outcomes

The individual-level factor influencing birth outcomes that we have focused on most in a number of studies is stress. There is now compelling scientific evidence that prenatal maternal stress in various forms predicts PTB (Dunkel Schetter, 2009; Dunkel Schetter & Glynn, 2010), and there is also considerable evidence regarding LBW. For example, Beydoun and Saftlas (2008), who reviewed the human and animal evidence for prenatal maternal stress and adverse outcomes, indicate that 9 out of the 10 studies on LBW that they reviewed show significant effects. Stress has been operationalized in these studies in numerous ways, typically by measuring either major life events, perceived stress, general state anxiety, or pregnancy-specific anxiety or distress.

In our first study on this topic, we followed 130 women throughout their pregnancies with assessments of stress at every prenatal visit using multiple stress measures (Lobel, Dunkel-Schetter, & Scrimshaw, 1992). Using structural equation modeling to combine measures of perceived stress, state anxiety, and distress ensuing from life events, we tested whether the composite stress factor predicted time of delivery (weeks gestation) and birth weight (in grams) when controlling for medical risk factors and risk behaviors, including smoking. Women participating in this study were interviewed from four to 12 times in pregnancy in the public clinic where they received prenatal care. They were diverse in ethnicity and were mostly low income. Some were undocumented residents. Approximately half were interviewed in Spanish.

As hypothesized, we found that the composite stress factor was predictive of earlier delivery (i.e., younger gestational age at birth) and lower birth weight (see Figure 19.2). At that time, there was relatively little prospective evidence for the effects of stress on these outcomes. Stress assessment had been problematic in many prior studies that used measures of unknown reliability or validity (Lobel, 1994). Utilizing a composite stress factor and multivariate modeling techniques offered several statistical and conceptual advantages. However, these results called for replication. Furthermore, physicians and epidemiologists preferred to see the prediction of dichotomous outcomes and risk ratios for PTB and LBW in order to consider the findings clinically relevant.

In subsequent prospective studies, stress consistently predicted gestational age at birth (GA) or PTB. In particular, studies by Dunkel Schetter and colleagues (Dunkel Schetter, 1998, 2009, 2010; Rini, Dunkel-Schetter, Wadhwa, & Sandman, 1999; Roesch, Dunkel Schetter, Woo, & Hobel, 2004; Wadhwa, Sandman, Porto, Dunkel-Schetter, & Garite, 1993; Zambrana et al., 1997) have shown that pregnancy-related anxiety—that is, fears and concerns about this pregnancy and baby specifically—is a unique predictor of the timing of delivery (cf. Lobel, Cannella, et al., 2008). Furthermore, three large epidemiological studies in the United States and Canada (Dole et al., 2003; Kramer et al., 2009; Orr, Reiter, Blazer, & James, 2007) report odds ratios of 1.5 to 2 times greater risk of PTB among women with high pregnancy-linked anxiety during pregnancy.

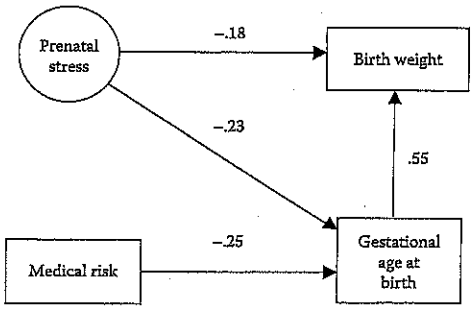

FIGURE 19.2 Effects of stress on birth outcomes. (Adapted from Lobel, M., Dunkel-Schetter, C., and Scrimshaw, S. C. M., *Health Psychology, 11*, 32–40, 1992.)

Thus, pregnancy-related anxiety appears to be a reliable and robust independent predictor of PTB. However, the effects of stress or anxiety on birth weight or LBW are less clear. For this reason, we believe that a review of the specific evidence about stress effects on birth weight and LBW is valuable in order to determine what forms of stress predict fetal growth outcomes and for whom.

Evidence Regarding Stress and LBW

Does prenatal (or prepregnancy) stress significantly influence infant birth weight independent of confounds including time of delivery (GA or PTB)? In order to examine this question, we systematically compiled available strong evidence on stress and birth weight or LBW published since 1990. Searches of relevant databases and citations in relevant articles yielded approximately 45–50 studies. The majority of the studies were prospective, a few utilized retrospective case-control designs, and a small group utilized state or national archives. Investigations were conducted in the United States and in several other countries including Brazil, Denmark, the Netherlands, Sweden, and New Zealand. We narrowed our review to investigations with at least 50 participants, but these studies usually had much larger samples sizes ranging up to 2,000 or more (Copper et al., 1996; Sable & Wilkinson, 2000), and in the archival studies, millions of births are involved (e.g., Khashan et al., 2008). Samples in the United States include teens, women of low-SES, African Americans, and residents from many regions of the country. Many of these investigations focused on the prediction of birth weight as the primary goal.

The assessment of stress varied greatly across studies which included measures of life events, depression, anxiety, general distress, and perceived stress and hassles, all of which are common measures. Still other approaches involve composite indices designed for pregnancy (e.g., Lobel, Canella, et al., 2008; Lobel, DeVincent, Kaminer, & Meyer, 2000; Pryor et al., 2003) or the examination of stress in the form of specific community events such as national tragedies in Sweden and the United States (Catalano & Hartig, 2001; Smits, Krabbendam, De Bie, Essed, & Van Os, 2006). Another form of stress studied is *racism*. These literatures virtually all measure stress at the individual level, in contrast to studies on neighborhoods that capture such stressful environmental conditions as neighborhood poverty (e.g. Collins, Wambach, David, & Rankin, 2009). A thorough review of evidence on neighborhood stressors and LBW is beyond the scope of this review, although it is necessary for any complete multilevel analysis.

The vast majority of studies that we reviewed found that measures of stress or emotion predicted either birth weight or the clinical outcome, LBW. Those studies that did not find such effects tended to have smaller samples, providing weak tests of the hypotheses, and had less rigorous study designs though a few exceptions to this rule exist. The nature of the measures used provides some clues to the patterns of findings, so we reviewed them by type of stress measure.

Several studies use the Perceived Stress Scale (PSS; Cohen, Kamarck, & Mermelstein, 1983) to study prenatal stress. This is a very general measure of stress reflecting inability to cope and being overwhelmed and not in control of stressful demands. A large prospective study in Sweden with 826 women giving birth for the first time found no effects of prenatal PSS scores on SGA births (i.e., births that are small for gestational age), although they included only four of the items on the original scale (Dejin-Karlsson et al., 2000). Similarly, a New Zealand case-control study of over 836 SGA births compared their PSS scores to those of 870 births that were average for gestational age and found no significant differences between the two groups (Pryor et al., 2003); this study does not report whether the researchers abbreviated the PSS or not. In contrast, Krabbendam et al. (2005) administered the full PSS to 5,511 Dutch women at 14 and 30 weeks of pregnancy and found that high perceived stress at 14 weeks increased risk of SGA but the effect was small after adjustment for education and smoking (OR = 1.16). Sable and Wilkinson (2000) conducted a retrospective case-control study of 2,378 pregnant women in Missouri, dividing them according to whether the infant was normal, low, or very low weight (with the latter, VLBW, defined as < 1500 g). They found that high perceived stress, reported retrospectively after birth, was associated with

1.5 times greater risk of a VLBW infant after adjusting for education, income, marital status, inadequate prenatal care, and smoking. A few other studies reported effects of stress on birth weight when the PSS was a part of a composite index (Dominguez, Dunkel Schetter, Mancuso, Rini, & Hobel, 2005; Lobel et al., 1992; Zambrana et al., 1997). However, others reported no effects of PSS as part of composite scores on birth weight (Rondo et al., 2003). Overall, stress as measured by the PSS does not seem to be a strong indicator of risk for LBW, especially when examined as the only stress measure.

The use of composite measures is one solution to stronger measurement in pregnancy (Lobel, 1994), and some studies suggest that this strategy increases predictive power (Herrera, Salmeron, & Hurtado, 1997; Lobel et al., 1992; Zambrana et al., 1997). For example, among 178 African American women studied prospectively beginning before 20 weeks' gestation, a composite of standardized prenatal stress variables accounted for 7% of the variance in birth weight adjusted for GA (Dominguez et al., 2005). However, composite stress variables have not always shown effects on birth weight (Lobel et al., 2000; Rini et al., 1999). Notably, these studies all included similar stress measures.

There is more consistent evidence that major life events contribute to LBW. Khashan et al. (2008) studied 1.38 million singleton Danish births over more than 20 years by merging birth data with records on specific severe life events. They found that the death of a relative during pregnancy, or in the six months prior to it, was associated with significantly higher risk of having small infants (RR = 1.17 for SGA; RR = 1.22 < 5th percentile). In another such study, Danish national databases were used to identify retrospectively 3,402 cases of LBW and 19,551 control cases (Precht, Andersen, & Olsen, 2007). Mothers exposed to severe life events before 32 weeks had twice the risk of SGA in these analyses. Another investigation showed that prepregnancy life events predicted birth weight after adjusting for controls in 100 low-educated, mostly White women studied prospectively (Pagel, Smilkstein, Regan, & Montano, 1990). In addition, a study of 92 mothers whose infants were LBW and 92 control women utilized objective coding of life events and chronic stress and found both were associated significantly with LBW, with some confounding variables controlled (Mutale, Creed, Maresh, & Hunt, 1991). A recent retrospective study examined 8,064 births in the South Carolina Pregnancy Risk Assessment and Monitoring System (PRAMS) and tested effects of life events in pregnancy reported after birth and of neighborhood poverty on both LBW and PTB (Nkansah-Amankra, Luchok, Hussey, Watkins, & Liu, 2010). In brief, they found significant effects of life events on LBW that were moderated in part by neighborhood context, with stronger effects for women in disadvantaged neighborhoods. Despite these findings, many of which involved strong measures and very large samples, a few studies have not found life event effects on birth weight (e.g., Hoffman & Hatch, 2000; McCormick et al., 1990).

Interesting results by Catalano and Hartig (2001) on Swedish birth data pertain to understanding communal life event stress and birth weight. They used time-series analyses to examine the effects of two national events that caused "communal bereavement:" a death of a national hero and a ship disaster that killed many people. The incidence of VLBW was higher in the months following the events compared to the months before. Similarly, Smits et al. (2006) found that pregnant women in the Netherlands exposed through the media to the September 11 attacks in New York City had lower birth weight infants (cf. Endara et al., 2009; Engels, Berkowitz, Wolff, & Yehuda, 2005; Eskenazi, Marks, Catalono, Bruckner, & Toniolo, 2007; Lederman et al., 2004). The possibility that community-wide tragedies pose risk of LBW is intriguing because pregnant women exposed to such events would be particularly amenable to intervention possibly in the context of prenatal care.

Investigators have also measured anxiety, depression, and general distress as predictors of LBW, birth weight, or related outcomes. General distress measured with the General Health Questionnaire (GHQ) during pregnancy was a significant risk factor for LBW in 865 Brazilian pregnant women (OR = 1.97; Rondo et al., 2003). Similarly, Hoffman and Hatch (2000) reported that depression measured with the Center for Epidemiological Studies Depression Scale (CES-D)

at week 28 of pregnancy among 666 women was associated with smaller infants but only in the low SES group. A Dutch study of 396 first births found depressed mood posed significant risk for SGA after adjusting for smoking, education, weight, and height (Paarlberg et al., 1999), although the effect size was modest (OR = 1.12). However, Zimmer-Gembeck and Helfand (1996) reported that when adjusted for ethnicity, medical risk, and health behaviors, LBW was associated with negative mood in a sample of 3,073 low-income women (OR =1.65). Furthermore, a prospective study of 712 inner city pregnant teens and adults composed of mostly minority women (Steer, Scholl, Hediger, & Fischer, 1992) found that depression measured with the Beck Depression Inventory (BDI) predicted LBW and SGA with relatively large effect sizes (OR = 3.97; 3.02 respectively). Clearly, among the psychosocial stress factors studied, distress and depressed mood seem to be stronger risk factors for LBW relative to other stress measures, although at least a few epidemiological studies report no significant effects (Chung, Lau, Yip, Chiu, & Lee, 2001).

In addition to conducting studies of depressed mood, some investigators have looked at clinically significant diagnoses of depressive disorders. For example, LBW was predicted by depressive disorder in 1,100 pregnant women who were screened for psychiatric disorders during pregnancy, with an odds ratio of 1.82 (Rogal et al., 2007). In addition, Kelly et al. (2002) conducted a population-based retrospective cohort study of 521,490 California births using hospital discharge data and found that psychiatric diagnoses predicted normal birth weight and VLBW (OR = 2.0, 2.9) adjusted for marital status, ethnicity, and prenatal care adequacy. Thus, results of tests of psychiatric diagnoses with birth weight are consistent in size and direction with those results on depressed mood or symptomatology (cf. Berle et al., 2005).

Regarding anxiety, a study of 1,500 indigent pregnant women in Alabama found that trait anxiety in the second trimester significantly predicted IUGR (Goldenberg, Cliver, Cutter, & Hoffman, 1991). Although only marginally significant, depressed mood had a larger effect (OR = 1.47 trait anxiety; OR = 2.00 depressed mood). Another study of a smaller sample of 132 pregnant women studied prospectively found that highly anxious women were more likely to have an LBW infant (Field et al., 2008). However, the sample size precluded control of some confounds, among them medical risk. Furthermore, a number of studies reported no significant effects of anxiety or anxiety diagnoses as a predictor of LBW (e.g., Berle et al., 2005; Catov, Abatemarco, Markovic, & Roberts, 2010; Rondo et al., 2003). Rini et al. (1999) tested the effects of anxiety on GA and birth weight in a sample of 221 women followed during pregnancy and postpartum using multivariate techniques that enabled distinguishing predictors of each outcome and controlling for medical risk, ethnicity, and other confounds. What resulted was a clear pathway from anxiety to earlier delivery but no direct effect on birth weight, only an indirect effect through GA. Thus, evidence for the effects of anxiety on LBW is not compelling, especially compared to the consistency of the effects of prenatal anxiety on PTB (Dunkel Schetter & Glynn, 2010). In contrast, depression appears to be a more powerful affective state than anxiety with respect to fetal growth and LBW.

Still another approach to studying stress prenatally is to examine chronic stressors. For example, a study of 1,363 pregnant, low-income women in Illinois used multiple measures of stress tailored to this population and reported that LBW was associated with such chronic stressors as unemployment or crowding, with odds ratios of 2.7 to 3.8 (Borders, Grobman, Amsden, & Hall, 2007). Similarly, Orr et al. (1996) examined stressors in family, work, finances, neighborhood, and housing in a sample of 2,000 low-income women, the majority of whom were African American. They found that exposure to these chronic stressors predicted LBW adjusted for medical and behavioral risks (OR = 1.52). Similarly, a study of 480 Black and White women in Alabama who were prospectively assessed regarding job strain (perceived demands and control) showed that women with high-strain jobs had lower birth weight infants compared to those with lower strain and, with adjustment for many confounding variables, compared to those who were unemployed (Oths, Dunn, & Palmer, 2001). There was also an interaction with race such that the effects were stronger for Blacks. Finally, Pritchard and Teo (1994), in a study of 393 Scottish women who had given birth previously, measured perceived difficulties in their household roles at 20 and 30 weeks' gestation, finding that high difficulty predicted LBW after adjustment for SES and smoking (OR = 4.70). In sum, chronic stressors appear to be a robust predictor of LBW. Unfortunately, the studies of chronic stress do not generally measure mood to determine if the indirect effects of stress through a pregnant woman's mood states, particularly depression, might account for these effects.

Evidence on Racism and LBW

Another important body of evidence demonstrating effects of prenatal stress on birth weight consists of studies investigating racial discrimination in pregnant women of color, especially African Americans (e.g., Giscombé & Lobel, 2005; Rich-Edwards & Grizzard, 2005). Racial discrimination or racism has been defined as oppression, domination, and denigration of individuals by other individuals and by social and cultural institutions based on skin color or membership in a particular ethnic group (Clark et al., 1999; Jones, 2000; Krieger, Rowley, Herman, Avery, & Phillips, 1993; Utsey & Ponterotto, 1996). At least eight studies have investigated whether racial discrimination contributes to elevated risk of delivering a LBW infant, of which six focused specifically on explaining the high rate of LBW in African Americans. (The remaining two studies, of Latinas and Arab Americans, are discussed separately later.) Studies examining racism and LBW also differ in whether they are prospective or retrospective, in their measures of racism, and in whether they control for other predictors of LBW. Such differences hinder the ability to draw conclusions from these studies; nevertheless, several of these studies find associations between racism and LBW. We summarize the findings of these studies below.

Generally, studies of racism and LBW in African American women have employed two different study designs or approaches. One approach, namely a disparities analysis, involves measuring perceptions of racial discrimination and LBW in Black women compared to women of a different race (usually Whites), then examining whether group differences in discrimination accounted for disparities in LBW. One such study compared African American women to non-Hispanic White pregnant women using a measure of racism adapted from a widely used and well-validated instrument assessing experiences in such various life domains as employment, school, health care, and public settings (Dominguez, Dunkel Schetter, Glynn, Hobel, & Sandman, 2008). The measure was adapted to determine whether respondents had personally experienced racial discrimination or had witnessed others experiencing it and whether, for each of these, it occurred in childhood or as an adult. Prenatal reports by expecting mothers of both vicarious childhood exposure to racism and of lifetime exposure to racism predicted giving birth to infants of lower birth weight. These measures of perceived racism also accounted, in part, for differences in birth weight between African American and White women, with other predictors of birth weight, including gestational age at delivery, controlled.

In a retrospective study of African American and White women, perceptions of racial discrimination partially accounted for the difference in rates of LBW between these two groups of women (Mustillo et al., 2004). However, controlling for gestational age eliminated the association between racism and LBW, indicating that effects of racism on birth weight in this study were mediated fully by earlier delivery. Another comparative study of African American women and women of other racial backgrounds (Chinese, Dominican, Puerto Rican, Mexican, and White) asked study participants if they had "experienced one or more incidents of racial discrimination" during their pregnancy. The authors did not find a significant association between perceived racial discrimination experienced during pregnancy and birth weight (Shiono, Rauh, Park, Lederman, & Zuskar, 1997) after controlling for numerous other predictors. However, associations between discrimination and birth weight were not examined separately for each ethnic group in this study, and the measure of racism was of unknown reliability and validity. Potential interactive effects of discrimination and ethnicity were also not examined, although racial discrimination may have greater impact on some racial groups (e.g., African Americans) than on others (Dominguez et al., 2008).

The second design approach (within racial group analyses) involved studying African American women exclusively and testing whether women who perceive greater racism are more likely to deliver an LBW infant. Three studies utilized this approach. Two investigations used adapted versions of the Krieger (1990) discrimination measure administered to women after the birth of a normal-weight or VLBW infant (< 1500 g; Collins, David, Handler, Wall, & Andes, 2004; Collins et al., 2000). Either all or a majority of study participants were impoverished in these two samples. Both studies found that racism was associated with an increase in the likelihood that an African American woman would deliver a VLBW infant. In the first of the two studies, the variable examined was discrimination during pregnancy; the association of this variable with VLBW was strongest in women with other risk factors (e.g., late or no prenatal care; cigarette or alcohol use), suggesting interactive effects. In the latter study, lifetime exposure to discrimination showed strong, dose-response relationships with VLBW, but discrimination during pregnancy, which was not frequently experienced in this sample, was not associated with VLBW (Collins et al., 2004). The third study of low-income African American women did not find an association between racial discrimination and birth weight (Dailey, 2009), but this study used a different measure than the earlier studies and produced a complex set of findings.

Finally, two intriguing studies of groups other than African Americans have used indirect indicators of racism to examine association with birth weight. Landale and Oropesa (2005) used the darkness of skin tone among Puerto Rican women as an indicator of racism. These researchers found that among Puerto Rican women living in five eastern U.S. states, those with darker skin tones were more likely to deliver a LBW infant. This association remained significant after controlling for numerous other variables including socioeconomic background, current SES, health behaviors, and prenatal care utilization. Skin tone also predicted the continuous birth weight variable in this group of women; that is, darker skin tone was inversely associated with birth weight in a linear manner. However, skin tone was not associated with birth weight for Puerto Rican women living in their native country or those living in New York City. Because New York City has the largest concentration of Puerto Ricans in the United States and arguably has higher acceptance of immigrants, these results are consistent with an interpretation involving racism.

Another indirect indicator of racism was examined in a study that compared rates of LBW in Californian women with Arabic surnames (as listed on birth certificates) before and after the September 2001 terrorist attack on the United States, and in comparison to women of other ethnic backgrounds (Lauderdale, 2006). Prior to September 2001, Arabic-named women had rates of LBW that were equivalent to those of White women. In the six-month period following September 2001, women with Arabic names experienced a 34% increase in deliveries of LBW infants. This increase did not occur for women without Arabic names including White, Black, Hispanic, and Asian women. The authors document the widespread, extreme, and violent discrimination that Arab Americans experienced during the period following September 11, 2001. Pregnant women with Arab surnames clearly experienced or witnessed such discrimination or lived in fear of it during this time.

In sum, the available studies on racism and discrimination in pregnant women indicate likely effects on birth weight, but as discussed later in this chapter, more research is needed.

Modifiers of Stress Effects on LBW

A small number of studies have examined interactive effects of stress on birth weight. These studies examine three main modifiers of stress effects: (1) medical risk, (2) maternal ethnicity and race, and (3) social support. Medical risk is usually a count of the number of risk conditions a mother has from a total of two to as many as three dozen possible antenatal risk factors. Evidence for modification of stress effects on birth outcomes by medical risk appeared in our own prior work reviewed earlier (Lobel et al., 1992). Among the 130 women studied prospectively, there were significant interactions of medical risk and stress in effects on both PTB and LBW. That is, women who were high in both stress and medical risk were at much greater risk of LBW compared to those women with either high

stress or high risk alone or low on both variables. These interactive effects also occurred in a similar pattern for GA. Given these interaction effects, we believe that interactions of stress with such specific medical risk factors as diabetes or hypertension may be important to test further, especially in populations that experience these conditions frequently. A recent study on stress and resting blood pressure in pregnant women (Hilmert et al., 2008) revealed that African American women who had a combination of high stress and high resting blood pressure (subclinical high levels) had infants of significantly lower-than-normal birth weight. These results are consistent with the notion that stress is more likely to contribute to an adverse outcome when it occurs in combination with medical risk conditions. However, most women in the study were not hypertensive; notably, high resting blood pressure values within normal range interacted with stress in effects on birth weight.

Another potentially important modifier of the effects of stress on birth outcomes is race or ethnicity, as alluded to earlier. Disparities in birth outcomes between African Americans and most other ethnic groups are integral to studies on stress and birth outcomes (Giscombé & Lobel, 2005; Hogue & Bremner, 2005). Two groups of researchers have examined interactive effects of prenatal stress and race on birth weight (Buka et al., 2003; Oths et al., 2001). In both studies, effects of stress on birth weight were greater in African American women than in European American women. Each study examined a different type of stress. Buka et al. (2003) found that the degree of poverty present in the neighborhood where a pregnant woman lived predicted lower birth weight in African American women but not in European American women. Oths et al. (2001) focused on job strain in pregnant women and found that it had a stronger inverse association with birth weight in African American women than in European American women. More attention to tests of interactions of stress in various forms with race is needed.

The third potential modifier of stress effects on birth weight is social support. Although most of the research on social support in pregnancy has examined only whether main effects on birth outcomes occur, the dominant hypothesis in the social support literature has been that support would interact with or modify effects of stress on health outcomes; that is, support would be expected to "buffer" effects of stress on birth outcomes. However, very few tests and little evidence of this exist. Turner, Grindstaff, and Phillips (1990), in one of the earliest and stronger studies of social support in pregnancy, found evidence of support moderation. This prospective study of 268 pregnant teenagers revealed that social support moderated effects of stress in the low-SES group but not in the higher SES group.

Collins, Dunkel-Schetter, Lobel, and Scrimshaw (1993) examined the role of social support in detail in predicting various birth outcomes, including birth weight. Among women who experienced high numbers of life events during pregnancy, those reporting higher quality social support delivered infants with higher birth weight, whereas women who had low levels of life events did not show the effect. Furthermore, whereas support quality was the only social support measure that had an interactive effect with stress, other aspects of support, including social network size, were directly associated with birth outcomes in this study. These findings indicate that researchers must assess various aspects of social support (e.g., quantity, quality, network) if they hope to understand social support processes in pregnancy (see Dunkel Schetter, 2010; Dunkel Schetter & Brooks, 2009; Rini, Dunkel Schetter, Hobel, Glynn, & Sandman, 2006). In addition, social support has shown benefits for only certain ethnic or racial subgroups in some studies suggesting interactions of race/ethnicity and social support on birth weight (Buka et al., 2003; Norbeck and Anderson, 1989; Sagrestano, Feldman, Killingsworth-Rini, Woo, & Dunkel-Schetter, 1999).

Summary of Research Findings on Stress and LBW

We observe that whereas research on stress and PTB has accumulated to a point where firmer conclusions can be drawn (Dunkel Schetter, 2009, 2010; Dunkel Schetter & Glynn, 2010; Savitz & Dunkel Schetter, 2006), the evidence regarding stress and lower birth weight lags behind. Altogether, available research findings indicate that stress in various forms including major and chronic stressors,

emotional states, and perceived racism, predicts lower birth weight. However, a major problem with this literature is that the studies vary so much in design, sample, measures, and control of possible confounds that it is difficult to make sense of what the primary stressors are that pose risk and for whom. Nonetheless, the findings on depression, chronic stress, and stress composite indices are somewhat consistent; those on life events are uneven but, on balance, suggest effects of note; and results on anxiety in pregnancy and perceived stress are few or none.

Although small in number, the results of studies examining stress modifiers are particularly important for three reasons: (1) they demonstrate why tests of interactions should be conducted and reported more often; clearly, specific subgroups of women may be at greater risk of the effects of stress on birth outcomes; (2) existing research provides a strong rationale for measuring modifiers comprehensively, especially as medical risk, ethnicity and race, and social support; and (3) the studies suggest that stress may be especially important to assess in women with medical and demographic risk factors such as high blood pressure or low SES.

MECHANISMS: MULTILEVEL MEDIATING PROCESSES

In the past decade, many researchers have endeavored to study the predictors of birth outcomes, but only recently has attention been directed to the mechanisms involved. Some of the multilevel predictors shown in Table 19.1 may also mediate other factors' effects on birth weight and preterm birth. For example, the effects of community and cultural influences on health outcomes are often hypothesized to be mediated by such individual-level factors as health behavior or emotional states. Similarly, individual-level psychological factors undoubtedly influence birth outcomes through complex biological processes. However, our scientific knowledge of these multilevel pathways remains incomplete.

The mediating processes perhaps most studied are variants of stress pathways to birth outcomes. Stressors have been hypothesized to influence birth weight through both physiological and behavioral mechanisms (Dunkel Schetter et al., 2000). Stress causes dysregulation of the immune, cardiovascular, and neuroendocrine systems in nonpregnant women. In pregnancy, these processes become much more complex. Physiological mechanisms linking stress with earlier delivery and PTB have received a great deal of attention (Behrman & Stith Butler, 2007; Challis et al., 2009; Coussons-Read, Okun, & Simms, 2003; Dunkel Schetter & Glynn, 2010; Wadhwa et al., 2001), whereas the physiological pathways from stress to LBW have not been studied as often. For example, hormones that the placenta releases in response to stressors, such as corticotrophin-releasing hormone (CRH), seem to precipitate early delivery in animals and human, a phenomenon referred to as the "placental clock" (Hobel, Dunkel-Schetter, Roesch, Castro, & Arora, 1999; Majzoub et al., 1999; Sandman et al., 2006; Smith, Mesiano, & McGrath, 2002). Considerable research has been devoted to this topic and has resulted in important and interesting gains in scientific knowledge about PTB. However, stress can also impair a woman's motivation and ability to maintain healthy behavior in pregnancy, thereby contributing to adverse outcomes. We posit that this health behavioral pathway is implicated more in the etiology of LBW than that of PTB. In the remainder of our discussion of stress–birth weight mediation, we focus on these two sets of hypothesized processes: (1) physiological mediation, and (2) mediation involving various health behaviors.

Physiological Mediation of Stress: The LBW Link

A growth-restricted fetus is one that has not reached its growth potential at a given gestational age as a result of one or more causal factors (Lin & Santolaya-Forgas, 1998). Up to 70% of SGA infants who are are small simply because of constitutional factors determined by maternal weight, height, parity, and ethnicity. Normal fetal growth occurs in three stages. The first phase is during the first 16 weeks of pregnancy and involves cellular hyperplasia, or a rapid increase in cell number. The second phase at midgestation involves increase in cell number and size. The last phase is after 32

weeks and involves rapid increases in cell size. Patterns of fetal growth retardation differ depending on the causes, their duration, and the stage of gestation. In addition, growth restriction in the fetus can be symmetric or asymmetric, with the latter accounting for 79% to 80% of cases. *Asymmetric FGR* refers to greater decrease in size of the fetus's abdomen compared to the head. *Symmetric FGR* refers to growth patterns where head and abdomen are decreased proportionately. It is believed that symmetric FGR occurs when risks occur earlier in gestation, and that asymmetric FGR occurs when risks occur later.

A large number of fetal, placental, maternal, and other factors can lead to FGR. Fetal factors include chromosomal abnormalities, congenital malformations, and multiple gestations. Maternal factors include genetic or constitutional factors, obstetrical risk factors such as previous stillbirth, recurrent abortion, previous preterm births, and maternal diseases. They also include such environmental factors as altitude and, as noted earlier, such behavioral factors as nutrition, smoking, alcohol abuse, and drug use. The most common cause of FGR is chronic vascular disease related to hypertension, renal diseases, diabetes, and vascular diseases, especially when co-occurring with preeclampsia. Preeclampsia, a rapidly progressing condition related to pregnancy, is characterized by high blood pressure and the presence of protein in the urine. The fetus can be viewed as parasitic to the mother in that it depends on maternal supply of oxygen and nutrients. Although further detail is beyond the scope of this chapter, it is important to note that inadequate placental blood flow may lead to pregnancy-induced hypertension and to FGR.

One often discussed pathway from stress to LBW involves the vascular effects of stress. Specifically, chronic maternal stress can result in vasoconstriction, reduced uteroplacental perfusion, and hypoxia (reduced oxygen to the fetus), thereby contributing to fetal growth retardation and LBW (Cosmi, Luzi, Gori, & Chiodi, 1990; Shepherd & Kiel, 1992). However, evidence supporting this stress-to-birth-weight pathway is not extensive. Kurki, Hiilesmaa, Raitasalo, Mattila, and Ylikorkala (2000) studied 623 pregnancies at 10–17 weeks' gestation and at delivery. A small percentage developed preeclampsia, and both depression and anxiety predicted preeclampsia (OR = 3.1 for both together). Also, Teixeira, Fisk, and Glover (1999) found anxiety assessed at 32 weeks of pregnancy predicted uterine artery resistance, which is consistent with a vascular mechanism linking anxiety with fetal development or SGA (cf. Harville et al., 2008).

Hypothalamic pituitary adrenal (HPA) involvement as a pathway from stress to fetal growth has also been investigated. To begin with, there is evidence regarding stress hormones and growth or birth weight. For example, maternal CRH, the corticotrophin-releasing hormone released from the placenta into maternal blood as part of the HPA stress response, significantly differentiated those who later gave birth to LBW and normal birth weight infants in a sample of several hundred women (Wadhwa et al., 2004). In a more recent study, increases in plasma maternal cortisol at 15, 19, and 25 weeks and increases in placental CRH at 31 weeks in 158 pregnant women were significantly associated with lower infant maturation scores at birth using a standard clinical assessment after controlling for length of gestation, but the effects held only for male infants (Ellman, Dunkel Schetter, Hobel, Chicz-DeMet, Glynn, & Sandman, 2008). Further, a Dutch study of 2,820 women found that higher maternal cortisol levels at 13 weeks gestation predicted lower birth weight and higher SGA risk, but only among women who provided blood samples at 9 a.m. or earlier (Goedhart et al., 2010). These results are consistent with a stress-HPA-LBW meditational chain but not definitive without measuring stress explicitly.

Diego et al. (2006) utilized ultrasound data and collected first-morning urine samples to assess cortisol and norepinephrine, and obtained psychological stress measures in 98 women during midgestation (16 to 29 weeks). Daily hassles predicted higher urinary cortisol which significantly predicted lower fetal weight, controlling for gestational age at assessment and SES. There was also a significant direct effect of maternal distress (depressed and anxious mood) on fetal weight that was not mediated by cortisol, which may be a result of unmeasured behavioral mechanisms. Overall, 26% of the variance in fetal weight was explained in this study. As the authors discussed, unbound cortisol can cross the placenta and affect fetal development by dysregulation of placental CRH

levels. This study is one of very few to include sociodemographic, biological, and psychosocial variables within a reasonably large sample and to predict fetal weight assessed by ultrasound (see also Diego, Field, Hernandez-Reif, Schanberg, Kuhn, & Gonzalez-Zuintero, 2009).

Of note, the techniques for obtaining biological samples, whether they are blood, urine, or saliva samples, and the details of how they are stored, processed, and assayed are critical to evaluating findings (Latendresse & Ruiz, 2008). In addition, preliminary evidence suggests that ethnic differences in HPA hormones during pregnancy may be important to consider in examining disparities in outcomes such as LBW (e.g. Glynn, Dunkel Schetter, Chicz-DeMet, Hobel, & Sandman, 2007). Very sophisticated multidisciplinary research on these HPA pathways is needed to replicate, extend, and clarify existing research.

Another set of physiological mechanisms likely to be involved in the associations between stress and LBW is the immune–inflammatory pathway. Prenatal stress has been linked to a greater likelihood of urogenital infection (Culhane et al., 2001) and infection is a known contributor to adverse birth outcomes. Much of this research has focused on the link between urogenital infection and PTB (Lu & Goldenberg, 2000; Newton, Piper, Shain, Perdue, & Peairs, 2001), although a few recent studies find an association between infections such as bacterial vaginosis and low birth weight (Svare, Schmidt, Hansen, & Lose, 2006; Thorsen et al., 2006; Vogel et al., 2006). We did not identify any published studies at time of writing linking stress, inflammation, and birth weight.

In sum, although the pathophysiology of fetal growth restriction is somewhat understood, it is quite complex. Furthermore, the ways in which stress is involved in these pathways has not been developed much in research.

Health Behavior in Pregnancy and LBW

Health behaviors represent an understudied and potent mechanism to explain any impact of prenatal stress on birth weight. In fact, LBW may be more behaviorally influenced than PTB. Many maternal behaviors influence the growth of the fetus; whereas fewer behaviors, mainly cocaine use and smoking, have clearly been documented to influence PTB (Savitz & Dunkel Schetter, 2006). The health behaviors known to contribute to LBW include smoking and substance use, diet and nutrition, and physical activity. As a result, women are advised by their health care providers to refrain from smoking and abusing substances and to eat a balanced diet at regular intervals during pregnancy. People experiencing high stress are, however, more likely to engage in various types of unhealthful behaviors that adversely affect birth weight, including poor eating habits, inadequate physical activity, cigarette smoking, and alcohol and other substance use (e.g., Ng & Jeffery, 2003; Stetson, Rahn, Dubbert, Wilner, & Mercury, 1997). Stress may also adversely influence women's attitudes toward their pregnancies, which in turn influence health behavior. For example, women who are unhappy about being pregnant seem to practice less optimal self-care during pregnancy compared to women who feel fortunate to be pregnant or feel that it is fulfilling a life goal (DeLuca & Lobel, 1995; Zambrana et al., 1997).

Despite the scientific plausibility of these behavioral mechanisms, surprisingly few studies have investigated the extent to which health behaviors mediate the impact of prenatal stress on birth weight. One exception is a recent study in which the impact of pregnancy-specific stress on birth weight was explained in part by its association with cigarette smoking using structural equation modeling techniques (Lobel, Canella, et al., 2008). An ethnically diverse sample of 279 women of moderate socioeconomic status was interviewed three times during early, mid-, and late pregnancy about various types of stress and health behaviors. Slightly more than a fifth of the study participants reported that they smoked during pregnancy. Those who were experiencing the greatest distress from such pregnancy-specific issues as their medical care, physical symptoms and bodily changes, concerns about becoming a parent, and worries about the baby's health were more likely to be smoking cigarettes during their pregnancy and, as a result, to deliver a LBW infant. Unhealthy eating, lack of physical activity, and other risky health behaviors did not mediate the association between stress and LBW but, as expected, were associated with pregnancy-specific stress. Thus, some health behaviors may mediate effects of stress on birth weight, whereas other health behaviors may not, and the patterns may be population-specific.

Although there is a dearth of research that simultaneously examines prenatal stress, health behaviors, and birth outcomes, studies that investigate the first link in the causal chain, that is, the associations of prenatal stress with health behaviors, are more common. Recent research on associations of stress in pregnancy with smoking, physical activity, and poor nutrition is reviewed next.

Stress, Cigarette Smoking, and Birth Weight

Cigarette smoking is a well-documented contributor to LBW (Crawford, Tolosa, & Goldenberg, 2008; Jaddoe et al., 2008; Phung et al., 2003; Windham, Hopkins, Fenster, & Swan, 2000), and there is some evidence that the impact of smoking in pregnancy may be greater in such otherwise vulnerable groups as older women and women of color (Windham et al., 2000). It is estimated that 20% to 30% of all LBW deliveries in the United States are attributable to maternal smoking during pregnancy (Crawford et al., 2008). Many female smokers do, however, abstain from cigarette smoking when they become pregnant out of concern for their baby's health or in response to health care provider recommendations (Crittenden, Manfredi, Cho, & Dolecek, 2007). However, approximately 10% of women continue to smoke during pregnancy (Crawford et al., 2008; Weaver, Campbell, Mermelstein, & Wakschlag, 2008).

Smoking during pregnancy has been shown to be associated with a variety of demographic factors including low education, low income, and non-Hispanic White ethnicity (Mathews, 2001). Smoking is also associated with various forms of stress, namely interpersonal violence, job strain, and low resources such as inadequate social support (Bullock, Mears, Woodcock, & Record, 2001; Crawford et al., 2008; Jesse, Graham, & Swanson, 2006; Song & Fish, 2006). In one of the more sophisticated analyses of the influence of prenatal stress on smoking, Weaver and colleagues (2008) found that low maternal education and low household income predicted persistent prenatal smoking (defined as smoking in at least two trimesters). Further, the number of cigarettes smoked was predicted by a latent psychosocial risk variable comprised of high numbers of stressful life events, dissatisfaction with social support, and little use of community resources. These authors suggest that the association between stress and smoking may be bidirectional; that is, women who smoke during pregnancy may feel distressed as a result of their smoking in addition to smoking in response to stress.

A recent study of 1,399 women living in an urban area of Russia examined the prospective prediction of birth weight in relation to prenatal stress, housing conditions, smoking, and alcohol consumption (Grjibovski, Bygren, Svartbo, & Magnus, 2004). Results indicated that smoking, living in a shared or crowded apartment, and perceived stress at work or at home were predictors of lower birth weight. Furthermore, smoking was correlated with lower education, being unmarried, living in a shared apartment, carrying an unplanned pregnancy, and perceived stress. Although mediational pathways were not examined in this study, the pattern of results suggests that such variables as stress and living in a shared apartment may have adversely affected birth weight through their association with smoking. Also notable in this study is that prenatal alcohol use was not correlated with any of the psychosocial variables examined, nor with smoking, and it did not predict LBW.

Stress, Physical Activity, and Birth Weight

Regular, moderately intense physical activity during pregnancy has been associated with higher birth weight (Clapp, Kim, Burciu, & Lopez, 2000; Lobel, DeVincent, Kaminer, & Meyer, 2000) and with other favorable pregnancy and labor outcomes, including fewer pregnancy-related discomforts (Horns, Ratcliffe, Leggett, & Swanson, 1996; Sternfeld, Quesenberry, Eskenazi, & Newman, 1995), less pain during labor (Varrassi, Bazzano, & Edwards, 1989), shorter duration of active labor (Clapp, 1990), shorter hospital stays (Hall & Kaufmann, 1987), and reduced likelihood of cesarean section (surgical) delivery in individual studies (Bungum, Peaslee, Jackson, &

Perez, 2000; Hall & Kaufmann, 1987). A meta-analysis of randomized controlled trials on aerobic exercise in pregnancy, however, found the available data insufficient to infer benefits for birth weight (Kramer & McDonald, 2009). They did, however, find effects of exercise on maternal fitness.

Furthermore, evidence is inconsistent about effects of physical activity from employment on birth outcomes. A recent meta-analysis of 29 studies (Mozurkewich, Luke, Avni, & Wolf, 2000) indicates that physically demanding work increases the likelihood of delivering a SGA infant (OR 1.37, 95% CI 1.30, 1.44). In another review (Clapp, 1996), two types of job-related activities that were most consistently linked to PTB and fetal growth restriction across studies involved "standing for long periods" and "working double-shifts," both activities that result in high fatigue and are thought to affect uterine blood flow and reduce oxygen and nutrients to the fetus. However, two other job-related activities, "heavy lifting" and "long periods of walking" were less consistently linked to PTB or restricted fetal growth (Clapp, 1996). The failure to differentiate between different types of job-related physical activities may help explain the contradictory pattern of findings that exists across studies on the effects of employment on pregnancy outcomes (see also Pompeii, Savitz, Evenson, Rogers, & McMahon, 2005). In addition, studies on employment typically fail to examine such characteristics of employment as the psychological demands (e.g., high strain, low control) that may help account for any adverse impact on birth outcomes in employed pregnant women (Woo, 1997).

Overall, research suggests that voluntary and moderate physical activity may have a direct and beneficial influence on birth weight, although more randomized controlled trials are needed to confirm this. In addition, this type of physical activity is associated with more favorable psychological and emotional states in pregnancy that may further influence birth weight positively. Moderate physical activity has been shown to be associated with lower stress and depression, better body image, and greater psychological well-being (Cannella, Lobel, & Monheit, 2008; Da Costa, Rippen, Dritsa, & Ring, 2003). Low-intensity activities like walking are also associated with improved psychological well-being in pregnancy (Da Costa et al., 2003; Sorensen, Williams, Lee, Dashow, Thompson, & Luthy, 2003). Conversely, in a variety of studies, reductions in physical activity during pregnancy have been linked to more negative mood (Poudevigne & O'Connor, 2006). However, the direction of these associations has not been definitively established. Associations between physical activity and prenatal psychological states may be bidirectional, thus requiring further prospective investigation.

It is of note that the prevalence of physical activity among women in the United States during pregnancy is low, consistent with larger trends of inactivity in women in general and particularly women of color (U.S. Department of Health and Human Services, 2008). It is estimated that 45% to 60% of pregnant women are sedentary (Leiferman & Evenson, 2003; Poudevigne & O'Connor, 2006; Zhang & Savitz, 1996). There has been little attention to the reasons for inactivity during pregnancy, but research in nonpregnant populations implicates the role of such stress-related factors as fatigue, lack of time, low income, lack of access to facilities, and insufficient tangible and emotional support (Albright, Maddock, & Nigg, 2005; King et al., 2000; Salmon, Owen, Crawford, Bauman, & Sallis, 2003). There may also be myths about exercising in pregnancy that motivate some women to rest more and reduce activity (Cannella, Lobel, & Monheit, 2010).

Stress, Poor Nutrition, and Birth Weight

There has been very little research examining the association between psychosocial factors and nutrition during pregnancy. However, it is well established that lower caloric consumption in pregnancy reduces fetal growth and contributes to lower birth weight (e.g., Scholl, 2008). Insufficient prenatal levels of such micronutrients as iron and folic acid also adversely influence birth weight (Haider & Bhutta 2006; Shah & Ohlosson, 2009). Shah and Ohlosson (2009) suggest a number of mechanisms that may explain how micronutrients affect fetal growth, including improved energy metabolism and better responses to stress. Other mechanisms, including enhanced immune function

and decreased susceptibility to infection, are posited to explain the beneficial association between micronutrient levels and reduced incidence of PTB.

An insightful review by Hobel and Culhane (2003) documents that both stress and poor nutrition independently contribute to poorer birth outcomes, and these authors suggest that there may also be interactive effects. For example, a combination of high stress and poor diet may compound adverse effects on growth. Furthermore, Hobel and Culhane indicate that prenatal stress reduces maternal weight gain during pregnancy by means of metabolic changes that affect the conversion of dietary calories. This link suggests that nutrition may mediate the impact of prenatal stress on birth weight not merely through changes in highly stressed women's eating behaviors but also through physiological processes that are directly affected by nutrition (Hermann, Siega-Ritz, Hobel, Aurora, & Dunkel Schetter, 2001).

Stress may also have a direct effect on reduced appetite, leading to poor prenatal nutrition. Fasting and restricting calories is a known risk factor for fetal growth retardation (Lin & Santolaya-Forgas, 1998). Although there is some ability of the maternal system to adapt to minimal or even moderate changes in diet and calorie intake, severe restriction of calories because of loss of appetite would be a likely pathway to fetal growth retardation.

Stress, Healthy Lifestyle, and Birth Weight

Several studies have investigated associations between prenatal stress and combinations of various health behaviors during pregnancy, referred to as *healthy or unhealthy lifestyle*. This is a sensible approach, given that various health behaviors tend to be intercorrelated. For example, women who are physically active prenatally also tend to engage in other healthy behaviors in pregnancy, including better dietary and sleep habits (Cannella et al., 2008).

One study of more than 3,000 socioeconomically disadvantaged, mostly African American women examined the predictive validity of women's "psychosocial profile" and health practices in relation to LBW (Neggers, Goldenberg, Cliver, & Hauth, 2006). The psychosocial profile combined 28 items assessing low negative affect, worry and stress, and high positive affect, self-esteem, and mastery. Health practices were assessed with 11 items pertaining to vitamin use, exercise, alcohol and tobacco use, and preventive medical and dental care, all combined into one score. The psychosocial profile was significantly correlated with the health practices composite score such that lower psychosocial profiles co-occurred with poorer health practices. Further, LBW was significantly more common in women below the median on the psychosocial composite score compared to women above the median, but LBW was not associated with health practices. The latter finding is not surprising, given the variety of health practices combined in this measure and the unlikely association of some of these practices (e.g., flossing and pap smear use) with birth weight. Furthermore, the authors did not examine mediation by such specific health practices as cigarette smoking, despite their finding that cigarette smoking was significantly more common in women with below-median psychosocial scores.

In sum, much is known about the importance of health behaviors as contributors to optimal growth of the fetus, and there is a small but growing number of studies that examine how stress during pregnancy affects these health behaviors. Yet health psychologists are not as active in this domain as they might be. The expertise of health psychologists is highly valuable because of their theoretical models, established findings in other populations, and understanding of the motivational processes and possible interventions for changing unhealthy habits. We return to this topic at the end to suggest some research agendas.

Formulating Integrative Models

Research on stress in pregnancy is beginning to integrate the multitude of operative factors into more comprehensive biopsychosocial models that can guide hypothesis generation, study design, and intervention. However, most of the models concentrate on PTB, and most focus on only a few of

the levels or subsets of processes shown in Table 19.1 such as the biomedical level or the psychosocial level (Coussons-Read et al., 2003; Hobel et al., 2008; Hogue, Hoffman, & Hatch, 2001; Kramer et al., 2001; Lu & Halfon, 2003; Rutter & Quine, 1990; Wadhwa et al., 2001; Wang et al., 2001). This narrow focus probably reflects the fact that the research training of authors has been in specific disciplines rather than in interdisciplinary research.

A few researchers have provided conceptual models to guide research on LBW or a broader set of outcomes, each with a specific focus or theme. Such models provide useful broad or general frameworks that can be a starting point for identifying more specific mechanisms. For example, Misra, Guyer, and Allston (2003) propose a multiple-determinants model that takes a lifespan approach and incorporates preconception and interconception (between births) factors to improve perinatal health. Their model includes social, psychological, behavioral, environmental, and biological forces operating during pregnancy and influencing short- and long-term maternal and infant disease and complications, health, functioning, and well-being. As such, it is one of the most comprehensive models we have seen. Even larger in scope, Halbreich's model (2005) includes in one comprehensive framework, not only birth weight but also postpartum outcomes, offspring development, offspring long-term disorders, and next-generation vulnerability.

Culhane and Elo's (2005) conceptual framework includes neighborhood factors (e.g., social environment, physical characteristics) as well as individual-level factors (e.g., SES, other demographics), both hypothesized to predict another set of individual-level factors (e.g., psychosocial, support, behavior), stress physiology, and ultimately, birth outcomes, including LBW. The inclusion of neighborhood factors is an attractive feature; yet a weakness of the model is that different types of individual stressors are lumped together into one category labeled "psychosocial factors." As we have tried to illustrate in this chapter, differentiation of specific psychological concepts is needed to determine unique pathways to such specific outcomes as LBW.

These models all offer pieces of a valuable foundation to test multilevel, biopsychosocial hypotheses about the etiology of LBW in interdisciplinary teams. Nonetheless, there remains a need to integrate existing approaches into comprehensive biopsychosocial frameworks that represent existing data on LBW with equal emphasis on psychosocial, sociodemographic, sociocultural, and biological factors.

Figure 19.3 is a simple schematic of the multilevel factors hypothesized to influence stress processes in pregnancy and of the pathways most often thought to mediate effects of stress on birth weight. Both acute and chronic stress exposures are shown to influence infant birth weight through effects on emotional, cognitive, behavioral, and physiological responses to stress (Pathway B). These varied responses to stress are interrelated in complex and dynamic ways based on theory and research on stress in general. Stress responses influence the growth of the infant and resultant birth weight directly (Pathway C), but also by influencing the timing of delivery (Pathways D and E), consistent with the interrelationship of the two major birth outcomes and their overlapping and distinct etiologies. Medical risk conditions influence birth outcomes as well (Pathways H and I). Not shown is the possible effect of stress and stress responses on medical conditions or complications that could be hypothesized. In addition, this meditational model shows that such contextual effects as socioeconomic risk factors and social network resources influence both exposures to stress (Pathway A) and birth outcomes (Pathways F and G). However, possible pathways from these contextual factors to stress responses are not included in this diagram, nor are moderation effects depicted. Thus, this is a simplified model, though it conforms to existing data in many ways and it may be a useful guide for hypothesis generation and research design.

Many of the predictors of infant birth weight shown in Figure 19.3 may operate as modifiers of the effects of other predictors. In particular, interactions of stress, ethnicity, medical risk, and social support deserve attention, as discussed earlier. Although behavioral scientists often view interactions as more interesting and important than direct (main) effects, epidemiologists and health scientists in other disciplines are typically more interested in direct effects and in effect sizes. Given the predominant interest in identifying predictors and direct causes in research on

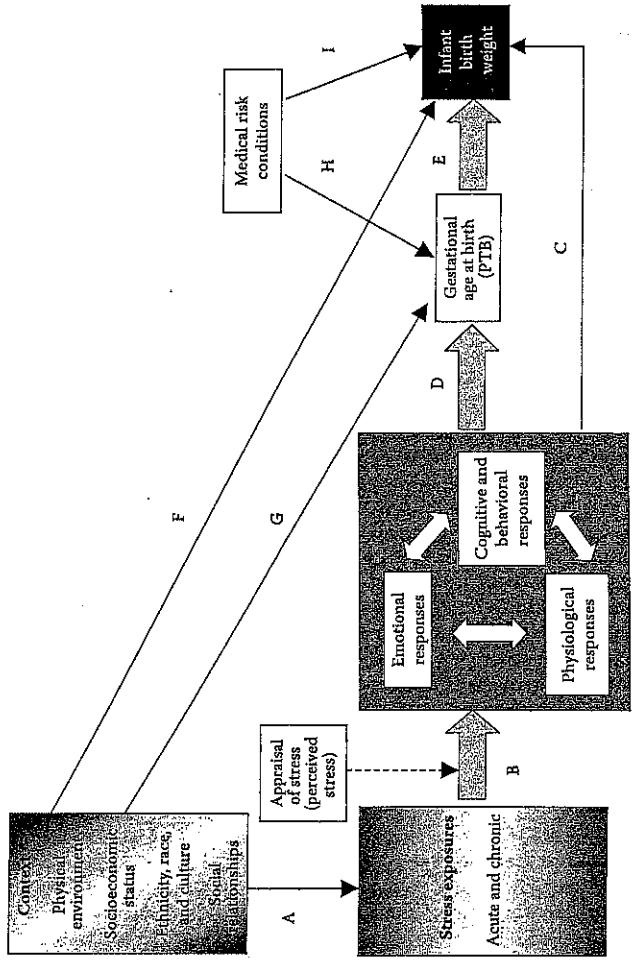

FIGURE 19.3 Multilevel biopsychosocial model of stress and birth weight.

pregnancy and birth, the study of moderation is not well developed, especially within interdisciplinary, multilevel analyses. Many studies do not test for interactions, and very few are designed to do this.

CONCLUSIONS AND THEMATIC ISSUES

We know much more today than we knew in 2001 when our chapter appeared in the first edition of this handbook. However, some of the observations we had then are still applicable today. For example, we suggested then that the psychosocial mechanisms linking stress to LBW might be different from those for PTB. This seems even more likely today since the two birth outcome literatures have grown immensely, and especially given the rapid growth of published research on PTB in recent years. This chapter's emphasis on LBW is intended to focus attention on the underdevelopment of the role of stress in fetal growth. Along with others, Brooks-Gunn (1991) pointed us in that direction long ago but, for various reasons, stress and birth weight research has not kept pace with research on stress and PTB, with a few important exceptions (Field, Diego, & Hernandez-Reif, 2006).

We highlighted in 2001 the need for multilevel analyses, a theme we continue as a focal point in this chapter. Without multilevel analysis, our understanding of LBW or any other birth outcome will not be advanced. We noted previously that behavioral mechanisms were important to examine, and there is now greater evidence that they are likely pathways linking stress to LBW. We emphasized the importance of social support and other potential moderators in our earlier chapter. Although there is only a little more research on support moderation of stress effects today, it remains a theoretically plausible and important topic for further investigation. Finally, almost 10 years ago we mentioned the importance of studying fathers as well as mothers, a call that has been echoed in the intervening years and is now gaining greater attention (Lu et al., 2010; Rini et al., 2006). In the remainder of this chapter, we highlight some conclusions on stress and birth weight in general and especially regarding African Americans. We further discuss health behavior mechanisms linking stress to birth weight. And, finally, we discuss why we may not be ready to intervene.

Conclusions Regarding Stress and LBW

First, do we at present understand the role of stress in fetal growth or the etiology of LBW? In fact, conclusions are very difficult to draw. Researchers must continue to determine what forms of stress in pregnancy are the most significant risk factors for LBW and for whom. We hypothesize that chronic stress is especially harmful because it is most likely to have a sustained influence on biological mediating processes and on health behaviors or a healthy lifestyle. We expect that pregnant women who have chronically stressful jobs, troubled relationships or marriages, chaotic households, or adverse neighborhood environments are most likely to have aberrations in fetal growth, and there is some evidence to support this. Women who experience sudden, acute events that are not accompanied by a chronic aftermath may not be at risk of LBW because they are better able to manage the consequences of these short-lived events and because the human body may be better able to readjust to briefer stressors. There is some evidence suggesting that moderate and intermittent stress may even program the fetus for more optimal development (DiPietro et al., 2010).

Such major stressors as life events, community-level catastrophes, and chronic strain may all operate to influence birth outcomes through specific and distinguishable emotional effects. For example, depression appears to be a risk factor for LBW, whereas anxiety is linked more to PTB (Dunkel Schetter, 2010; Dunkel Schetter & Glynn, 2010). The effects of depression and anxiety may differ because of potentially distinct physiological processes. Evolving models of emotions and stress emphasize the functional properties of specific emotions and their distinct physiological correlates, at least under conditions of acute stress (Kemeny & Shestyuk, 2008; Moons, Eisenberger, & Taylor, 2010). This research has implications for studies of pregnancy that are untapped as yet.

Furthermore, such emotional states as depression or anxiety are rarely conceptualized as mediators of the effects of stress exposures on physiology or outcomes in pregnancy research, although this is a potentially valuable direction for research. In fact, state-of-the-art psychological theory and evidence on emotions generally is rarely integrated into the study of pregnancy. Yet, advances in emotion theory and research are highly relevant to the pregnant state and its influence on birth outcomes. In particular, we posit that the task of conceptualizing pathways from prenatal stress to such birth outcomes as LBW can benefit from advances in our understanding of the effects of specific emotions on physiology.

Stressors may be cumulative or interacting in their effects on birth outcome, and we can begin to understand them only if we find ways to conduct large-scale studies with multiple types and levels of stressors. For example, neighborhood and family stressors experienced together are likely to pose greater risk of adverse outcome than either alone; their interaction may be especially harmful. Health psychologists and psychological scientists who possess sophisticated measurement and statistical skills useful in formulating and testing multivariate models can help to improve the contributions of research on stress and birth outcomes.

Conclusions Regarding Stress and Birth Weight in African Americans

Compared to other groups, African American women experience unique stressors, particularly personal and institutional racism. Although the findings are not completely consistent, evidence suggests that racism over one's lifetime, including childhood vicarious exposure to it, influences health outcomes in general and birth outcomes specifically. However, racial differences in LBW appear to persist even after accounting for the contribution of discrimination. Thus, racism is not a sufficient explanation for these group differences. Sexism and racism together have been hypothesized to create a "double jeopardy" in African American women (Woods-Giscombé & Lobel, 2008; Rosenthal & Lobel, 2011). Also, African American women are often the caretakers of their social network, creating additional responsibilities and stress for them, especially in communities where chronic strain and major life events are common and resources for coping with them are low.

The effects of racism are also compounded by low SES, possibly in a curvilinear fashion. That is, there is some evidence that both low and high SES appear to contribute additional risk for adverse birth outcomes beyond racial risks. For low-SES African American women, a source of stress may be living in a segregated community that is a target of institutional discrimination; high-SES African American women who work or live in integrated settings also incur stress exposure. It is very important for pregnancy research to disentangle SES from race and racism because these variables have often been confounded in previous studies. Among the studies of racial discrimination and birth weight in African Americans, only one that we identified used a sample of African Americans who were not predominantly low SES (Dominguez et al., 2008).

At present, we do not yet know what types of discrimination pose the greatest risk for pregnant women. We also have little data on the various ways that women of color cope with the threat of racism or its occurrence. Such behavioral responses as overeating, smoking, substance use, and alcohol consumption have been noted (Woods-Giscombé & Lobel, 2008). In general, responses to racism have been grouped into avoidant or direct action, but a more complete picture of the various behaviors and patterns of behavior that are used to manage the effects of exposure to personal racism and personal and institutional discrimination is needed to clarify their effects on pregnant women, as well as to identify the modifiers that may reduce or exacerbate these effects.

Conclusions Regarding Stress, Health Behaviors, and Birth Weight

As the studies reviewed in the foregoing sections indicate, there is ample evidence that stress and some related psychosocial factors are associated with poorer health behaviors during pregnancy, and separate evidence confirms that both stress and such health behaviors as cigarette

smoking and inadequate nutrition predict LBW. This research provides a strong foundation for studies that simultaneously examine prenatal stress and the health behaviors that are likely mediators of birth weight (e.g., cigarette smoking and poor nutrition). Studies that examine stress and health behaviors should also investigate the interactive impact of stress and health behaviors. Detecting mediation through health behaviors does not preclude the possibility that stress also has direct effects on birth outcomes or other indirect effects. As we have noted throughout, effects of stress on birth weight, or any birth outcome, are likely to be explained by multiple mechanisms.

As with research on health behaviors in any population, an important challenge in pregnancy is identifying the best method of measurement. Most of the existing research on prenatal health behaviors uses self-report measures, yet there is a valid concern that women may be unusually reluctant to reveal to researchers that they are engaging in behaviors known to be harmful in pregnancy. However, there is some evidence that self-report measures of cigarette smoking during pregnancy are reliable and highly correlated with serum cotinine levels (McDonald, Perkins, & Walker, 2005; Okah, Cai, Dew, & Hoff, 2005). Similarly, physical activity recall measures show strong correlations with objective measures of activity in pregnant women (Lindseth & Vari, 2005; Timperio, Salmon, & Crawford, 2003). Further, there is some evidence that pregnant women accurately report their eating behaviors (Verbeke & De Bourdeaudhuij, 2007). Moreover, if such behaviors as cigarette smoking and poor eating habits are underreported by pregnant women, then studies using self-report measures that find effects may be underestimating the strength of those effects.

Another important challenge involves the difficulty in establishing causality among stress, health behaviors, and birth weight. There appear to be bidirectional associations between stress and some health behaviors. For example, negative correlations in cross-sectional research designs may mean that stress reduces the level of physical activity, but physical activity may also reduce stress (Cannella et al., 2008; Lobel, Hamilton, et al., 2008). Also, pregnant women are usually aware of the dangers of such behaviors as alcohol consumption, substance use, and cigarette smoking; therefore, those women who engage in harmful behaviors may have increased stress or worry as a result of their behavioral risk factors (Weaver et al., 2008). The frequency, pattern, and degree of engagement in specific health behaviors and the possibility of bidirectional associations with stress can be determined only through prospective, repeated and detailed assessments in observational studies or through experience-sampling methods.

Why We Are Not Ready to Intervene

Strong interventions are based on strong research findings and theory. In the study of stress and birth weight, researchers are not in a good position to design interventions because they lack strong theoretical and empirical bases for designing intervention at present. Results of observational research are just now accumulating a base of scientific knowledge to develop models of the pathways to LBW as distinct from those to PTB and that warrant interventions that target the modifiable components of these pathways. However, such interventions must be based in multilevel analysis taking into account sociocultural contexts, psychosocial processes, and biomedical and behavioral pathways. Until the science of prenatal risk and birth outcomes improves further, large-scale intervention studies are not, in our view, warranted. However, small-scale pilot investigations based on specific mechanisms influencing mediators may be valuable. For example, experimental studies might target one or more processes among the neuroendocrine, behavioral, and psychological processes that are hypothesized to affect birth weight. Then, having demonstrated effects on one or more of these mediating processes, a larger scale study targeting a birth outcome could follow. For example, small-scale randomized studies of the effects of social support enhancement or stress reduction on hypothesized behavioral or biological mediators may provide a basis for predicting larger scale effects on outcomes.

CONCLUSION

Our goal in this chapter has been to elucidate the state of research on psychosocial factors in pregnancy while focusing on processes leading to fetal growth and birth weight as distinct from the processes involved in other outcomes such as preterm birth. In doing so, a broader goal was to emphasize the value of a multilevel analysis of pregnancy and birth that incorporates more sophisticated and in-depth consideration of psychological factors. It is an exciting time for researchers working in this area because of advances on all fronts and transdisciplinary collaboration. With rigorous theory and research directed to the study of pregnancy and its outcomes, there should be important developments in our understanding of birth outcomes, especially low birth weight, to report when a third edition of this handbook appears.

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