

MATERNAL NUTRITION AND INFANT MORTALITY IN THE CONTEXT OF RELATIONALITY



THE COURAGE TO LOVE:
INFANT MORTALITY COMMISSION

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THE COURAGE TO LOVE: INFANT MORTALITY COMMISSION
IMPLICATIONS FOR CARE, RESEARCH, AND PUBLIC POLICY TO
REDUCE INFANT MORTALITY RATES

MATERNAL NUTRITION AND INFANT MORTALITY
IN THE CONTEXT OF RELATIONALITY

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PREFACE

Solutions to the problem of higher infant death rates among black families have eluded medical, health policy, and research communities for decades. African American women continue to face a disproportionately higher risk for delivering premature and low birthweight babies, many of whom die within their first year of life.

Although infant mortality in the United States decreased among all races between 1980 and 2000, the overall black-white gap for infant mortality widened—and this pattern has continued. A 2002 Centers for Disease Control and Prevention analysis of infant mortality rates in 1995-1998 in the 60 largest U.S. cities revealed that the median infant mortality rate for blacks was 13.9 per 1,000 live births, compared to 6.4 and 5.9 for whites and Hispanics, respectively. Nationwide, the most recent data (2003) show that the infant mortality rate for blacks is 13.5 per 1,000 live births, compared to 5.7 for non-Hispanic whites and for Hispanics. The lack of progress in closing the black-white gap is largely due to a persistent two- to threefold higher risk for low birthweight and very low birthweight among black infants compared to white infants.

Healthy People 2010 is this nation's health promotion and disease prevention initiative. It includes a national objective to reduce deaths among infants (aged less than one year) to fewer than 4.5 per 1,000 live births within all racial and ethnic groups. If current infant mortality rates among African Americans persist, however, such national health objectives to reduce infant mortality and to eliminate related racial and ethnic disparities will not be met.

The root causes of persistent racial disparities in infant mortality are not thoroughly understood. Many theories have been proposed. The high incidence of infant deaths among African Americans has been attributed to high teen pregnancy rates, single motherhood, lower education levels, poverty, and—most recently suggested—genetic causes. These theories fade in the light of robust research, however; alarmingly high levels of infant mortality persist, even when most factors are controlled. African Americans have higher infant mortality rates in every age category; maternal characteristics, such as marital or employment status, do not alter disparities; nor do education or income levels. The genetic theory is weakened by research that shows better birth outcomes among foreign-born black women; regardless of their socioeconomic status, native-born African American women fare worse in birth outcomes compared to white women at every income and education level. Most recently, the Institute of Medicine's 2006 Report on Preterm Birth concluded that

racial/ethnic differences in socioeconomic condition, maternal behaviors, stress infection, and genetics cannot fully account for disparities. The report called for research that continues to prioritize efforts to understand factors contributing to the high rates of preterm birth among African American infants.

If age, marital status, education, income, and/or genetics cannot be seen as a singular root cause for racial and ethnic disparities in infant mortality, what variables or set of variables might be seen as common among African American women and others who experience poor birth outcomes? Are these variables or set of variables responsive to intervention? The search for answers to these perplexing questions led the Health Policy Institute of the Joint Center for Political and Economic Studies to establish a national commission to study infant mortality within a new context of "relationality"—the notion that relationships are constitutive of what it means to be human. The central role of relationships and their associated effects upon maternal and infant well-being have generated a new understanding of the infant mortality challenge. This new approach is grounded in social determinants of health theory; women and their babies must be viewed not only as individuals, but as members of families, communities, and larger systems that have either positive or negative impacts upon their psychological and physical states. The economies, opportunities, environmental influences, as well as risk and protective factors within their places of work, life, and play must be considered.

The Courage to Love: Infant Mortality Commission, co-chaired by Ronald David, MD, MDiv, and Barbara Nelson, PhD, was formed by the Joint Center Health Policy Institute, in collaboration with the University of California, Los Angeles (UCLA) School of Public Affairs, to review the history of infant mortality rate analysis and interpretation, examine basic assumptions, redefine the problem, and imagine new possibilities for action. The Commission's intentional focus on relationality has potential implications for improved pregnancy outcomes, economic prosperity, and meaningful civic participation for all women and for African American women in particular.

To better understand the issues and to inform its deliberation in formulating recommendations for policy, research, and practice, the Commission asked experts in various fields related to maternal and child health and infant mortality to prepare background papers on specific issues. This background paper explores the relationship between maternal nutrition and infant mortality, with an emphasis on the context of relationality. It provides



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an analysis of the relationship between maternal nutrition and leading causes of infant mortality, as well as maternal, infant, and child health; an overview of the nutritional status and behaviors of pregnant women in the U.S.; and a comprehensive review of the effectiveness of nutritional supplementation programs in pregnancy. The final chapters reframe the relationship between maternal nutrition and infant mortality within the context of relationality over the life course and offer related recommendations for research, policy, and practice. This analysis complements and reinforces the recommendations of other Courage to Love: Infant Mortality Commission background and framing papers on infant mortality and resilience; the role of breastfeeding in maternal and infant health; the historical framework of policies and practices to reduce infant mortality; the authentic voices of those affected by infant mortality; and infant mortality in a global context.

The work of the Courage to Love: Infant Mortality Commission is part of the larger effort by the Joint Center Health Policy Institute (HPI), whose mission is to ignite a “Fair Health” movement that gives people of color the inalienable right to equal opportunity for healthy lives. Funded by the W. K. Kellogg Foundation, HPI seeks to help communities of color identify short- and long-term policy objectives and related activities that:

- Address the economic, social, environmental, and behavioral determinants of health;
- Allocate resources for the prevention and effective treatment of chronic illness;

- Reduce infant mortality and improve child and maternal health;
- Reduce risk factors and support healthy behaviors among children and youth;
- Improve mental health and reduce factors that promote violence;
- Optimize access to quality health care; and
- Create conditions for healthy aging and the improvement of the quality of life for seniors.

We are grateful to Dr. Michael C. Lu and Jessica S. Lu for preparing this paper and to those Joint Center staff members who have contributed to the preparation, editing, design, and publication of this paper and the Commission’s other papers. Most of all, we are grateful to Drs. David and Nelson, the members of the Commission, and Dr. Gail C. Christopher, Joint Center vice president for health, women and families, for their dedication and commitment to improving birth outcomes for African Americans and reducing racial and ethnic disparities in infant mortality rates.

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EXECUTIVE SUMMARY

The United States has one of the highest rates of infant mortality among developed nations. Disparities in infant mortality by race and class continue to be a national disgrace. Insofar as poor maternal nutrition may be linked to several leading causes of infant mortality, such as preterm birth and fetal growth restriction, a focus on maternal nutrition may help shed light on the causes and prevention of infant mortality.

The purpose of this report is to examine the relationship between maternal nutrition and infant mortality. Our specific aims are as follows:

1. To describe the relationship between maternal nutrition and several leading causes of infant mortality (e.g., preterm birth, fetal growth restriction, birth defects, and maternal complications such as anemia, infections, or preeclampsia), as well as the contributions of maternal nutrition to maternal, infant, and child health and developmental programming of adult diseases;
2. To describe the nutritional status and behaviors of pregnant women in the U.S., with a focus on racial-ethnic disparities where data are available, in terms of anthropometric, biochemical, and clinical measures, and dietary content and patterns;
3. To review evidence of the effectiveness of nutritional supplementation programs in pregnancy, including randomized controlled trials on single- and multinutrient supplementation in pregnancy and evaluations of the Women, Infants, and Children (WIC) programs; and
4. To reframe the relationship between maternal nutrition and infant mortality in the context of relationality over the life course.

MATERNAL NUTRITION AND INFANT MORTALITY

We found good evidence linking poor maternal nutrition to several leading causes of infant mortality, including birth defects, preterm birth, fetal growth restriction, and maternal complications of pregnancy (preeclampsia, anemia, infections/inflammation). Maternal folate and B12 deficiencies have been associated with neural tube defects, while deficiencies

in B vitamins, vitamin K, magnesium, copper, and zinc have also been linked to other birth defects. Low prepregnancy body mass index (BMI) and poor gestational weight gain are associated with greater risk for preterm birth and fetal growth restriction. Maternal nutrition can also mediate or modulate several of the major pathways (e.g., inflammatory) leading to spontaneous preterm birth. While the contribution of specific nutrient deficiencies to preeclampsia remains unclear, maternal nutrition can potentially play an important role in the pathogenesis of preeclampsia by affecting endothelial function, ameliorating oxidative stress, modulating inflammatory response, and improving insulin action. In light of the importance of abnormal implantation and placentation in the pathogenesis of preeclampsia, periconceptional nutrition may be of paramount importance. Nutritional deficiencies of iron, folate, and vitamins A, B6, and B12 can cause anemia. Vitamin A and other micronutrient deficiencies have been implicated in maternal infections, and antioxidants can potentially play a major role in modulating inflammation and oxidative stress from maternal infections. The growing body of research on fetal programming of adult diseases further elevates the clinical and public health significance of maternal nutrition.

NUTRITIONAL STATUS AND BEHAVIORS OF PREGNANT WOMEN IN THE UNITED STATES

Most pregnant women in the U.S. start off pregnancy overweight or underweight, and had inappropriate weight gain during pregnancy. For low-income African American women, only 40 percent enter pregnancy with normal BMI and less than 30 percent achieve ideal weight gain during pregnancy. Approximately one of every three low-income women is anemic in the third trimester of pregnancy; the prevalence of anemia is substantially higher (44 percent) among African American women than among all other racial-ethnic groups. The prevalence of diabetes during pregnancy is 3.3 percent. The prevalence, however, ranged from three percent for non-Hispanic black mothers to 5.4 percent for Asian and Pacific Islander mothers and 5.7 percent for Native American mothers. With respect to dietary intakes among pregnant women, soft drinks, fruit juices, biscuits, muffins, white bread, and other refined carbohydrates are the leading sources of energy from carbohydrates, while mayonnaise, salad dressings, whole milk, French fries, and fried potatoes are the leading sources of energy from fats. In general, white women consume a higher nutrient-dense diet, and black women consume more calories but fewer nutrients after energy adjustment. Available data suggest that pregnant women in the U.S.



consume more protein, fat and trans-fat, and carbohydrates than recommended. A substantial proportion of pregnant women do not meet their recommended daily allowances (RDA) for iodine, calcium, magnesium, iron, zinc, vitamins A, B1, B2, B3, B6, B12, and vitamin C from food sources. Dietary intake of folate is inadequate for over 95 percent of women, and that of vitamin E is inadequate for 25 percent of pregnant women, which perhaps reflects low intakes of fruits and vegetables. When multivitamins are accounted for, one in four still does not consume adequate amounts of folate and vitamin E. For African American women, only about one in four pregnant women meets the RDA for calcium, magnesium, zinc, and vitamin E, and about one in three does not meet the RDA for iron and folate. Fasting, pica, and fast-food consumption are common among pregnant women, particularly among African American women.

PRENATAL NUTRITIONAL INTERVENTIONS: EVIDENCE OF EFFECTIVENESS

The evidence of the effectiveness of any single macro- or micronutrient supplement for preventing fetal growth restriction, preterm birth, birth defects, and maternal complications (preeclampsia, anemia, and infections) is far from conclusive, with the possible exceptions of periconceptional folic acid supplementation for prevention of neural tube defects and iron and folate supplementation for prevention of maternal anemia. Fish oil for prevention of recurrent preterm birth, balanced protein-energy supplementation for prevention of fetal growth restriction, and calcium supplementation for prevention of preeclampsia in high-risk women also appear promising. No other maternal supplementation interventions have been shown to be effective. However, it is premature to conclude that maternal nutritional interventions do not work, as most trials have focused on supplementing one single nutrient during pregnancy; few studies have examined the impact of multinutrient supplementation that starts before pregnancy. Most evaluations of Women, Infant, and Children (WIC) programs have found evidence of their effectiveness for preventing low birthweight (LBW); however, their effectiveness may be overstated due to problems of selection bias, simultaneity bias, and lack of generalizability.

RETHINKING MATERNAL NUTRITION AND INFANT MORTALITY — THE CONTEXT OF RELATIONALITY OVER THE LIFE COURSE

There is no quick fix or silver bullet for our nation's infant mortality problem. Nutrition can play a key role in preventing several leading causes of infant mortality, but only as part of a longitudinally and contextually integrated strategy for improving maternal and family health. In this report, we examine the problems of infant mortality and poor maternal nutrition in the context of relationality. We contend that both are, in essence, problems of broken relationships at many levels. These broken relationships are manifested in the lack of support for breastfeeding, the decline in family meals concomitant to the rise of fast food, the marketing of junk foods to children, and the prevalence of food insecurity in a land of plenty. These broken relationships create lifelong conditions of high stress and low support, which in turn pattern physiological, psychological, and behavioral responses that put the mother at risk for poor nutrition during pregnancy, and her baby at risk for fetal and infant death. African American families are disproportionately affected by these broken relationships, which contribute to disparities in maternal nutrition and infant mortality. We concur with the recommendation of the Joint Center Health Policy Institute's *Courage to Love: Infant Mortality Commission* that efforts to reduce maternal and infant mortality and morbidity must focus on the repair and support of relationships at all levels and across the life course.

RECOMMENDATIONS

The report concludes with recommendations for future directions and priority areas in research, practice, and policy related to maternal nutrition and infant mortality.

RESEARCH

- **Preconceptional and Interconceptional Nutrition**
Researchers should conduct more intervention studies of maternal nutritional supplementation that begins before (preconceptional) and between (interconceptional) pregnancies. The National Institutes of Health and other funding agencies should support these studies.
- **Content of Nutritional Supplementation**
Researchers should conduct more intervention studies of multinutrient maternal nutritional supplementation. The National Institutes of Health and other funding agencies should support these studies.



- **Relational Context of Maternal Nutrition**

Researchers should conduct more research studies of the influences of partner support, provider encouragement, social network, social capital, and other relational contexts on maternal nutritional status and behaviors. Furthermore, researchers should conduct more intervention studies that build upon these relational contexts for improving maternal nutritional status and behaviors before and during pregnancy. The National Institutes of Health and other funding agencies should support this research.

- **Psychosocial Factors and Maternal Nutrition**

Researchers should conduct more research studies of the influences of psychosocial factors on maternal nutritional status and behaviors before and during pregnancy. Such research efforts should be guided by an integrative framework that takes into account the multilevel influences of stress and nutrition. Furthermore, researchers should conduct more intervention studies that test different social support strategies for improving maternal nutritional status and behaviors before and during pregnancy. Development of such strategies should be guided by the principles and methods of community participatory action research. The National Institutes of Health and other funding agencies should support these studies.

- **Life-Course Influences on Maternal Nutrition**

Researchers should conduct more research studies that identify critical influences over the life course on maternal nutritional status and behaviors. Furthermore, researchers should conduct more intervention studies that address these life-course influences to improve maternal nutritional status and behaviors before and during pregnancy and over the life course. The National Institutes of Health and other funding agencies should support such research.

PRACTICE

- **Increase Surveillance for Food Insecurity**

The American College of Obstetricians and Gynecologists (ACOG), the American Academy of Pediatrics, and other professional organizations should recommend routine assessments of food insecurity during perinatal care as practice guidelines, and the Centers for Disease Control and Prevention, the Maternal and Child Health Bureau, and state and local Title V agencies should

routinely monitor food insecurity in maternal and child health (MCH) populations. Furthermore, prenatal care providers must ensure that women and families who screen positive for food insecurity receive appropriate nutritional services, and state and local Title V agencies must ensure that such services are available and accessible to women and families who need them. All eligible women and families who are at nutritional risk must be referred to WIC.

- **Address Psychosocial Factors in Pregnancy**

Perinatal care providers, including WIC programs, should routinely assess psychosocial factors, including stress and social support among pregnant women and families. Those who screen positive should be referred to appropriate support services (e.g., psychological counseling for women with depressive or other affective disorders, support services for women and children who are victims of intimate partner violence).

- **Support Healthy Nutrition in Relational Contexts**

Perinatal care providers, including WIC programs, should experiment with the use of a pregnant woman's own personal relationships to support her nutritional behaviors. Such experiments should be supported and encouraged by health plans, Title V programs, and other public agencies or private foundations. The use of *doulas*, group prenatal care (e.g., Centering Pregnancy), and other forms of social support for improving maternal nutrition should also be explored.

POLICY

- **Promote Baby-Friendly Hospitals and Workplace**

The U.S. Department of Health and Human Services, the Centers for Disease Control and Prevention, the Maternal and Child Health Bureau, state and local Title V agencies, and other public and private organizations should promote the adoption of baby-friendly policies in birthing hospitals in the U.S. All public agencies should adopt baby-friendly policies in the workplace, including provision of a breastfeeding room for nursing mothers.

- **Regulate Marketing of Junk Foods to Children**

The Federal Trade Commission should regulate marketing of junk foods during children's television programming. State and local educational authorities should promote healthful diets for children and youth in all aspects of the school environment (e.g., restrictions on commercial



sponsorships of school activities, banning of soda and other junk food vending machines from schools, provision of healthy nutrition through school lunch programs, and development of educational curricula to promote healthy nutrition).

- **Strengthen Women, Infants, and Children (WIC) Programs**

The Department of Agriculture should continue to explore ways to strengthen WIC. The commissioned Institute of Medicine report on repackaging WIC food packages is a good start, but more can be done to enhance the content of WIC food packages to improve maternal health and optimize developmental programming. Additionally, more efforts and resources are needed to improve outreach, health education, service coordination and systems integration, and community building to increase food access and quality, particularly in disadvantaged communities. More attention must be paid to addressing psychosocial barriers to WIC participation. WIC alone is not the answer to infant mortality, however. The Department of Agriculture needs to join forces with the Centers for Disease Control and Prevention, the Maternal and Child Health Bureau, the National Institute of Child Health and Development, and other leaders in maternal and child health to develop a roadmap for addressing this nation's infant mortality problem, using an integrative approach and making improvement of maternal and infant nutrition a priority.

- **Support Fragile Families and Strengthen Partner/Father Involvement**

Federal and state governments should pass legislation to encourage family formation and remove disincentives for partner/father involvement through Temporary Assistance for Needy Families (e.g., eliminating the distinction between single- and two-parent families for eligibility determination), the Earned Income Tax Credit (e.g., allowing a second-earner deduction), and child support programs (e.g., establishing amnesty programs; allowing greater “pass-through” of child-support payments to children; and extending TANF, EITC, and other support services to non-custodial fathers who pay child support). Policymakers should also increase support for educational programs, employment-related services, legal and social services, and marriage counseling for low-income fathers to strengthen their capacity to fulfill the roles and responsibilities of fatherhood.

- **Address Institutionalized Racism in Maternal Nutrition**

State and local governments, civic and business leaders, community- and faith-based organizations, and other community leaders can all play an active role in improving maternal and family access to healthy nutrition. Tax incentives, subsidies, and other incentives can be provided to encourage grocers, restaurants, and farmers' markets to do business in the community, and to induce fast-food restaurants and sundry stores (even liquor stores) to sell healthy produce. Multilevel, multichannel health messaging should be undertaken to stimulate demand for healthy nutrition. Title V and other public health agencies need to routinely monitor differential access to healthy nutrition and study its impact on health disparities.



CHAPTER 1

MATERNAL NUTRITION AND INFANT MORTALITY

I. INTRODUCTION

The United States has one of the highest rates of infant mortality among developed nations. Disparities in infant mortality by race and class continue to be a national disgrace. Insofar as poor maternal nutrition may be linked to several leading causes of infant mortality, such as preterm birth and fetal growth restriction, a focus on maternal nutrition may help shed some light on the causes and prevention of infant mortality.

The purpose of this report is to examine the relationship between maternal nutrition and infant mortality. Our specific aims are as follows:

1. To describe the relationship between maternal nutrition and several leading causes of infant mortality (e.g., preterm birth, fetal growth restriction, birth defects, and maternal complications such as anemia, infections, or preeclampsia), as well as the contribution of maternal nutrition to maternal, infant, and child health and developmental programming of adult diseases (Chapter 1);
2. To describe the nutritional status and behaviors of pregnant women in the U.S., with a focus on racial-ethnic disparities where data are available, in terms of anthropometric, biochemical, and clinical measures, and dietary content and patterns (Chapter 2);
3. To review evidence of the effectiveness of nutritional supplementation programs in pregnancy, including randomized controlled trials on single- and multivitamin supplementation in pregnancy and evaluations of the Women, Infants, and Children (WIC) programs (Chapter 3); and
4. To reframe the relationship between maternal nutrition and infant mortality in the context of relationality over the life course (Chapter 4).

This report serves as a background paper for the Joint Center Health Policy Institute's *Courage to Love: Infant Mortality Commission*. Given the intransigence of the problem of infant mortality, this national commission was charged with examining basic assumptions, redefining the problem, and exploring new

possibilities for action. Specifically, there is new and promising research on the lack of social support and the presence of unmitigated stress as potentially contributing factors to preterm births and infant mortality rates. However unconscious or coincidental, these studies are emerging at a time when philosophers, theologians, and scientists are beginning to consider relationality and complexity as substantive of human nature, or as constitutive of what it means to be human. The Commission was asked to address the following question: *If relationships are primary and all else is derivative, what then are the implications for care, research, and public policy to reduce infant mortality?*

This report takes a similar approach to examining maternal nutrition and infant mortality, with an intentional focus on relationality. We posit that both are, in essence, problems of broken relationships at many levels. These broken relationships create lifelong conditions of high stress and low support, which in turn pattern physiological, psychological, and behavioral responses that put the mother at risk for poor nutrition during pregnancy, and her baby at risk for fetal and infant death. African American families are disproportionately affected by these broken relationships, which contribute to disparities in maternal nutrition and infant mortality. We concur with the recommendation of the Commission that efforts to improve maternal nutrition and reduce infant mortality must focus on the repair and support of relationships at all levels and across the life course. We conclude this report with recommendations for future directions and priority areas in research, practice, and policy related to maternal nutrition and infant mortality (Chapter 5).

II. INFANT MORTALITY IN THE UNITED STATES

In 2003, 28,428 infant deaths were reported in the United States (1). The infant mortality rate (IMR) was 6.9 per 1,000 live births. Despite a steady decline in IMR (except for 2001-2002) over the past several decades, the United States still has one of the highest IMRs among developed nations. In 2003, the U.S. ranked 27th among developed nations with populations of greater than 250,000 (**Table 1.1, next page**). One quarter of the countries shown in Table 1 had IMRs that were half the U.S. rate in 2002. Although some researchers have suggested that the reporting of infant deaths in the U.S. may be more complete than in other developed countries (2, 3), these reporting differences are certainly not the entire story (1). Some reasons for the U.S. position may include the high percentage of low birthweight (LBW) infants, the heterogeneity of the U.S. population relative to many other developed countries, and continuing disparities in health among disadvantaged groups (1).



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TABLE 1.1: NUMBER OF LIVE BIRTHS AND BIRTH RATES FOR 2002 AND IMR FOR 2000, 2001, AND 2002 FOR COUNTRIES OF >250,000 WITH IMR EQUAL OR LESS THAN THE UNITED STATES IN 2002 AND 2001

Country	No. of Births in 2002	Birth Rate, 2002	IMR		
			2002	2001	2000
Hong Kong	48,209	7.1	2.3	2.6	3.0
Singapore	40,760	9.8	3.0	2.4	2.9
Japan	1,153,855	9.1	3.0	3.1	3.2
Finland	55,555	10.7	3.0	3.2	3.8
Sweden	95,815	10.7	3.3	3.7	3.4
Norway	55,434	12.2	3.5	3.9	3.8
Czech Republic	97,878	9.6	3.9	4.0	4.1
Spain	407,135	10.1	4.1	3.4	4.0
France	770,945	13.0	4.1	4.5	4.4
Austria	78,399	9.7	4.1	4.8	4.8
Germany	734,475	8.9	4.2	4.3	4.4
Belgium	114,014	11.1	4.4	4.5	4.8
Denmark	64,149	11.9	4.4	4.9	5.3
Italy	537,070	9.3	4.5	4.7	4.5
Switzerland	72,372	9.9	4.5	5.0	4.9
Portugal	114,383	11.0	5.0	5.0	5.6
Australia	250,988	12.8	5.0	5.3	5.2
Korea	494,625	10.4	5.1	5.4	4.5
Netherlands	202,083	12.5	5.1	5.4	5.1
Greece	102,282	10.2	5.1	5.1	5.2
Ireland	60,521	15.5	5.1	5.8	6.2
United Kingdom	668,777	11.3	5.2	5.5	5.6
Israel	139,535	21.2	5.4	5.1	5.5
Canada	328,802	10.5	5.4	5.2	5.3
New Zealand	54,021	13.7	5.6	5.3	6.1
Cuba	141,276	12.6	6.5	6.2	7.2
United States	4,021,726	13.9	7.0	6.8	6.9

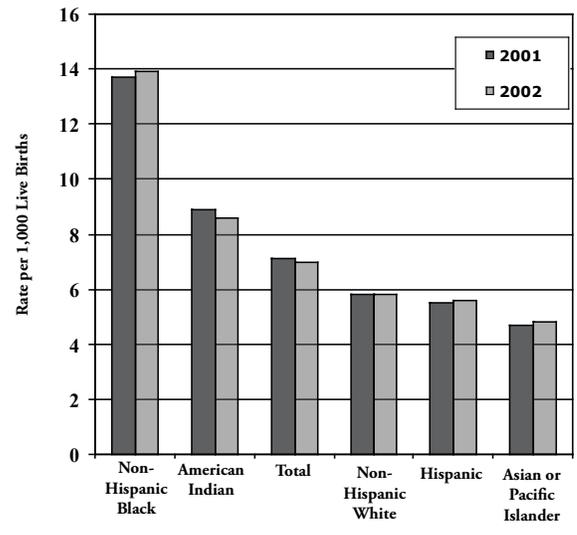
Source: D. L. Hoyert, T. J. Mathews, F. Menacker, D. M. Strobino, B. Guyer, "Annual summary of vital statistics: 2004," *Pediatrics* 117 (2006):168-83.

III. DISPARITIES IN INFANT MORTALITY IN THE UNITED STATES

There are significant racial-ethnic, geographic, and sociodemographic disparities in infant mortality in the United States.

Racial-ethnic disparities in infant mortality remain a national concern. In 2002, the IMR was 13.9 for non-Hispanic black infants and 5.8 for non-Hispanic white infants (Figure 1.1) (1, 4). The relative difference in the IMRs of non-Hispanic black and non-Hispanic white infants expressed as a ratio was 2.4 in 2002, a

FIGURE 1.1: INFANT MORTALITY RATES BY RACE AND ETHNICITY, 2001 AND 2002



Includes persons of Hispanic and non-Hispanic origins. Persons of Hispanic origin may be of any race.

Source: T. J. Mathews, F. Menacker, and M. F. MacDorman, Centers for Disease Control and Prevention, National Center for Health Statistics, "Infant mortality statistics from the 2002 period: linked birth/infant death data set," *Natl Vital Stat Rep.* 53, no. 10 (Nov 24, 2004): 1-29.

ratio that has been steadily increasing since 1980 (Figure 1.2) (1, 4). Within a racial or ethnic group, there are significant disparities in IMRs. For example, in 2002 IMRs for infants born to Asian and Pacific Islander mothers ranged from 3.1 for Chinese Americans to 9.3 for Hawaiians, and IMRs for infants born to Hispanic mothers ranged from 3.7 for Cuban Americans to 8.2 for Puerto Ricans (4).

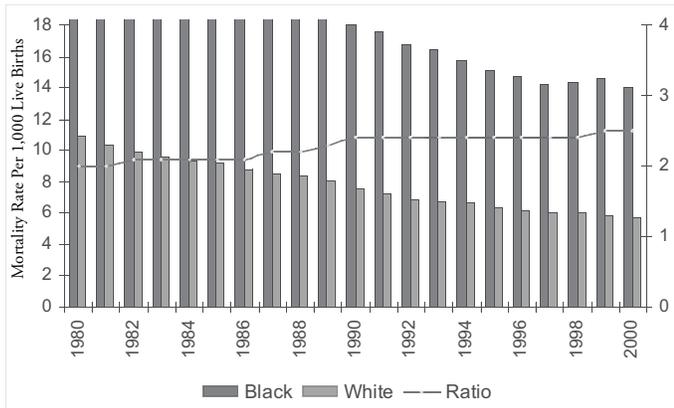
IMRs also varied considerably by region of the country and state, and within states by race and ethnicity. Generally, states in the South had the highest IMRs, and states in the West and Northeast had the lowest. IMRs for states ranged from 4.8 for Massachusetts to 10.5 for Mississippi, with the District of Columbia reporting the highest IMR of 11.4 for 2000-2002. For infants of non-Hispanic black mothers, IMRs ranged from 9.5 in Washington State to 17.9 in Wisconsin. For infants of Hispanic mothers, IMRs ranged from 4.6 in South Carolina to 8.6 in Pennsylvania (Table 1.2) (4).

IMRs also varied significantly by maternal sociodemographic characteristics in 2002. Maternal age is related to IMR in a U-shaped curve; infants born to mothers under the age of 20 or ages 35 and over have higher IMRs (Table 1.3, page 5) (4). However, the association between maternal age and IMR was not consistent across racial and ethnic groups. IMR began to rise at a younger maternal age for non-Hispanic black mothers (ages 30-34) than for non-Hispanic white mothers (ages 35-39).

MATERNAL NUTRITION AND INFANT MORTALITY IN THE CONTEXT OF RELATIONALITY



FIGURE 1.2: RATES AND RATIO OF NON-HISPANIC BLACK AND WHITE INFANT MORTALITY RATES, 1980-2000



Source: T. J. Mathews, F. Menacker, and M. F. MacDorman, Centers for Disease Control and Prevention, National Center for Health Statistics, "Infant mortality statistics from the 2002 period: linked birth/infant death data set," *Natl Vital Stat Rep.* 53, no. 10 (Nov. 24, 2004): 1-29.

Geronimus (5) attributes this differential rise with increasing age to "weathering." According to the "weathering hypothesis," the effects of social inequality on health compound with age, leading to growing gaps in health status between black and white women during young and middle adulthood that can affect their reproductive outcomes. However, evidence supporting the "weathering hypothesis" remains inconclusive, as most studies using cross-sectional data cannot adequately control for potential cohort effects.

Infants born to unmarried mothers had higher IMRs compared to those born to married mothers in 2002 (4). The reasons for the higher IMRs among unmarried mothers are not known but are commonly attributed to their relative lack of social support and resources (6, 7). Note, however, that IMRs are significantly higher for married non-Hispanic black mothers than for unmarried non-Hispanic white mothers.

TABLE 1.2: INFANT MORTALITY RATES BY STATE AND MATERNAL RACE AND ETHNICITY, 2000-2002

State	Total	Race				Hispanic origin		
		White	Black	American Indian	Asian or Pacific Islander	Hispanic	Non-Hispanic White	Non-Hispanic Black
Infant mortality rates per 1,000 live births in specified groups								
United States	6.9	5.7	13.5	8.9	4.8	5.5	5.7	13.6
Alabama	9.3	6.8	14.8	*	*	7.0	6.8	14.7
Alaska	6.8	5.4	*	11.2	*	*	5.1	*
Arizona	6.7	6.3	14.4	9.4	5.3	6.0	6.5	14.4
Arkansas	8.3	7.2	12.8	*	*	4.5	7.5	12.8
California	5.4	5.0	11.4	7.6	4.5	5.1	4.7	11.4
Colorado	6.0	5.5	13.8	11.8	6.2	6.2	5.2	13.7
Connecticut	6.4	5.4	14.2	*	3.7	7.1	4.9	14.3
Delaware	9.6	7.9	14.8	*	*	7.9	7.9	14.9
District of Columbia	11.4	4.8	15.2	*	*	7.5	*	15.3
Florida	7.2	5.6	12.9	5.8	5.1	5.2	5.7	13.0
Georgia	8.7	6.3	13.4	*	6.8	6.0	6.3	13.4
Hawaii	7.2	6.6	*	*	7.3	6.0	6.3	*
Idaho	6.6	6.6	*	*	*	8.8	6.2	*
Illinois	7.8	6.1	15.8	*	6.5	6.4	5.9	15.8
Indiana	7.7	6.9	13.9	*	*	6.4	7.0	13.9
Iowa	5.8	5.6	11.7	*	*	6.7	5.5	11.4
Kansas	7.0	6.5	14.6	*	*	7.1	6.4	14.7
Kentucky	6.7	6.3	10.7	*	*	4.8	6.4	10.8
Louisiana	9.8	6.8	13.8	*	8.1	6.0	6.9	13.7
Maine	5.1	5.1	*	*	*	*	5.0	*
Maryland	7.7	5.3	12.6	*	4.5	5.7	5.3	12.7
Massachusetts	4.8	4.3	9.6	*	3.7	6.0	4.0	10.5
Michigan	8.1	6.3	16.9	*	4.9	6.7	6.0	16.9
Minnesota	5.5	4.9	10.8	10.3	6.1	6.5	4.7	10.8
Mississippi	10.5	7.0	14.8	*	*	*	7.0	14.7

Table continued on next page.



MATERNAL NUTRITION AND INFANT MORTALITY IN THE CONTEXT OF RELATIONALITY

TABLE 1.2: INFANT MORTALITY RATES BY STATE AND MATERNAL RACE AND ETHNICITY, 2000-2002 (CONTINUED)

Missouri	7.7	6.3	15.6	*	4.5	7.2	6.3	15.6
Montana	6.9	6.5	*	9.9	*	*	6.4	*
Nebraska	7.0	6.3	14.8	15.8	*	7.2	6.2	15.0
Nevada	6.0	5.3	13.6	*	4.7	5.1	5.1	13.7
New Hampshire	4.9	4.9	*	*	*	*	4.5	*
New Jersey	6.1	4.8	13.1	*	3.3	6.3	4.0	13.6
New Mexico	6.4	6.2	15.6	6.8	*	6.3	6.0	15.8
New York	6.1	5.0	10.7	*	3.4	5.5	4.8	11.2
North Carolina	8.4	6.3	15.0	10.6	5.9	5.6	6.4	15.1
North Dakota	7.8	7.2	*	13.4	*	*	6.8	*
Ohio	7.7	6.4	15.5	*	4.8	7.6	6.3	15.3
Oklahoma	8.0	7.3	14.6	7.6	*	5.7	7.4	14.5
Oregon	5.5	5.5	10.3	*	3.7	5.1	5.6	10.4
Pennsylvania	7.3	6.2	14.6	*	4.0	8.6	5.9	14.4
Rhode Island	6.7	6.2	11.9	*	*	8.0	5.3	12.6
South Carolina	9.0	5.9	15.0	*	*	4.6	6.0	14.9
South Dakota	6.4	5.5	*	11.6	*	*	5.4	*
Tennessee	9.0	7.0	17.0	*	*	6.2	7.0	17.0
Texas	5.9	5.3	11.1	*	4.0	5.1	5.5	11.1
Utah	5.3	5.2	*	*	8.4	6.5	5.0	*
Vermont	5.5	5.6	*	*	*	*	5.5	*
Virginia	7.2	5.4	13.7	*	4.6	4.8	5.5	13.6
Washington	5.5	5.3	9.5	10.6	4.8	5.1	5.2	9.5
West Virginia	7.9	7.8	12.1	*	*	*	7.7	11.7
Wisconsin	6.9	5.6	17.9	11.5	5.2	6.2	5.6	17.9
Wyoming	6.5	6.6	*	*	*	*	6.3	*
Puerto Rico	9.4	9.4	10.4	—	—	—	—	—
Virgin Islands	7.0	*	6.0	*	*	*	*	*
Guam	7.3	*	*	*	7.7	*	*	*

Source: T. J. Mathews, F. Menacker, and M. F. MacDorman, Centers for Disease Control and Prevention, National Center for Health Statistics, "Infant mortality statistics from the 2002 period: linked birth/infant death data set," *Natl Vital Stat Rep.* 53, no. 10 (Nov. 24, 2004): 1-29.

IMRs also varied by maternal education in 2002 (4). Generally speaking, infants born to mothers with lower educational attainment had higher IMRs compared to those born to mothers with higher educational attainment. Education, however, does not afford equal protection across racial and ethnic groups. In fact, non-Hispanic black mothers with 16 or more years of education still had a higher IMR than non-Hispanic white mothers with less than nine years of education (4). Lu and Halfon (8) attribute this higher IMR among well-educated African American women to their cumulative experience of chronic stress over the life course, which causes wear and tear on their reproductive health over time.

IMRs also varied by nativity (4). In 2002, the IMR was 7.3 for infants of mothers born in the United States and 5.1 for infants of mothers born outside the United States. For every major racial

group, IMRs were substantially higher for U.S.-born mothers than for foreign-born mothers. Even duration of residence matters. A study in California found that long-term Mexican immigrants who have lived in the United States for more than 5 years were more likely to deliver preterm and low birthweight infants than newcomers who have lived in the United States for five years or less (9).

Table 1.3 displays disparities in IMR based on maternal or infant characteristics. Interested readers are referred to the National Center for Health Statistics website (10) for more details.

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TABLE 1.3: INFANT MORTALITY RATES BY MATERNAL & INFANT CHARACTERISTICS, 2002

Characteristics	Race of Mother				
	All Races	White	Black	American Indian	Asian or Pacific Islander
Infant mortality rates per 1,000 live births in specified group					
Total	7.0	5.8	13.8	8.6	4.8
Age at death:					
Total neonatal	4.7	3.9	9.3	4.6	3.4
Early neonatal (less than 7 days)	3.7	3.1	7.6	3.2	2.7
Late neonatal (7-27 days)	0.9	0.8	1.7	1.4	0.7
Postneonatal	2.3	1.9	4.5	4.0	1.4
Sex:					
Male	7.6	6.4	14.8	9.7	5.1
Female	6.3	5.1	12.8	7.6	4.4
Plurality:					
Single births	6.1	5.0	12.3	7.9	4.3
Plural births	32.3	28.0	55.9	38.4	23.5
Birthweight:					
Less than 2,500 grams	59.5	54.7	76.5	64.2	41.0
Less than 1,500 grams	260.8	242.1	272.1	249.1	218.4
1,500-2,499 grams	15.1	15.3	15.4	24.0	10.7
2,500 grams or more	2.4	2.2	3.9	4.3	1.6
Period of gestation:					
Less than 32 weeks	186.4	175.8	212.9	158.6	163.4
32-36 weeks	9.2	8.7	11.1	13.1	7.3
37-41 weeks	2.5	2.2	4.0	4.3	1.7
42 weeks or more	3.1	2.8	4.7	5.9	2.5
Trimester of pregnancy prenatal care began:					
First trimester	6.2	5.2	12.8	7.9	4.4
After first trimester or no care	9.0	7.6	14.3	9.5	5.3
Second trimester	7.3	6.5	10.5	8.9	4.3
Third trimester	6.0	4.9	9.3	*	4.5
No prenatal care	38.4	29.9	58.0	*	30.5
Age of Mother:					
Under 20 years	10.4	8.8	15.2	9.1	9.2
20-24 years	7.8	6.4	13.9	9.4	5.2
25-29 years	6.0	5.1	12.4	7.6	3.9
30-34 years	5.6	4.7	13.4	7.6	4.3
35-39 years	6.5	5.5	14.5	8.5	5.4
40-54 years	8.5	7.3	16.1	*	8.2
Educational attainment of mother:					
0-8 years	6.6	6.1	14.7	*	4.0
9-11 years	9.6	8.0	15.8	8.3	5.9
12 years	7.8	6.5	13.4	9.1	5.6
13-15 years	6.0	4.9	11.7	8.6	4.7
16 years and over	4.2	3.7	9.9	*	3.7
Live-birth order:					
1	7.0	5.9	14.2	9.1	4.7
2	6.1	5.2	12.3	8.4	4.0
3	6.6	5.6	12.2	6.8	5.2
4	8.3	6.7	15.1	7.9	7.8
5 or more	11.1	8.7	18.7	11.2	7.7

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MATERNAL NUTRITION AND INFANT MORTALITY IN THE CONTEXT OF RELATIONALITY

TABLE 1.3: INFANT MORTALITY RATES BY MATERNAL & INFANT CHARACTERISTICS, 2002 (CONTINUED)

TABLE 1.3: INFANT MORTALITY RATES BY MATERNAL & INFANT CHARACTERISTICS, 2002 (CONTINUED)										
Marital status:										
Married	5.4	5.0	11.8	7.2	4.4					
Unmarried	9.9	7.9	14.8	9.6	7.1					
Mother's place of birth:										
Born in the 50 States and DC	7.3	5.9	14.2	8.7	6.6					
Born elsewhere	5.1	4.9	8.8	*	4.3					
Maternal smoking during pregnancy:										
Smoker	11.1	9.8	20.0	12.1	11.6					
Nonsmoker	6.8	5.3	13.1	7.7	4.7					
Characteristics	All Origins	Total	Hispanic					Non-Hispanic		
			Mexican	Puerto Rican	Cuban	Central and South American	Other and Unknown Hispanic	Total	White	Black
Infant mortality rates per 1,000 live births in specified group										
Total	7.0	5.6	5.4	8.2	3.7	5.1	7.1	7.3	5.8	13.9
Age at death:										
Total neonatal	4.7	3.8	3.6	5.8	3.2	3.5	5.1	4.8	3.9	9.3
Early neonatal (less than 7 days)	3.7	3.0	2.9	4.9	2.7	2.7	4.3	3.9	3.0	7.6
Late neonatal (7-27 days)	0.9	0.8	0.8	0.9	*	0.8	0.9	1.0	0.8	1.8
Postneonatal	2.3	1.8	1.8	2.4	*	1.6	2.0	2.4	1.9	4.6
Sex:										
Male	7.6	6.0	5.9	8.7	4.5	4.9	8.0	8.0	6.5	14.9
Female	6.3	5.2	4.9	7.7	2.9	5.3	6.2	6.5	5.1	12.8
Plurality:										
Single births	6.1	5.1	4.9	7.1	3.2	4.5	6.4	6.3	5.0	12.3
Plural births	32.3	31.1	30.0	42.9	*	27.6	37.7	32.3	27.1	55.9
Birthweight:										
Less than 2,500 grams	59.5	56.7	57.0	59.2	46.6	52.0	62.2	59.7	53.4	76.5
Less than 1,500 grams	250.8	241.8	247.7	234.4	188.6	213.7	268.1	250.9	239.5	272.1
1,500 - 2,499 grams	15.1	16.1	16.6	14.1	*	15.2	15.3	14.9	14.9	15.4
2,500 grams or more	2.4	2.0	2.0	2.6	*	1.7	2.3	2.5	2.2	3.9
Period of gestation:										
Less than 32 weeks	186.4	160.9	159.3	182.2	144.5	147.6	176.7	191.1	179.9	212.9
32-36 weeks	9.2	8.0	7.8	8.9	*	7.7	10.2	9.4	8.9	11.1
37-41 weeks	2.5	2.1	2.1	2.7	*	1.9	2.3	2.6	2.3	4.1
42 weeks or more	3.1	2.5	2.6	*	*	*	*	3.3	2.9	4.9
Trimester of pregnancy prenatal care began:										
First trimester	6.2	5.3	5.1	7.5	3.4	4.8	6.1	6.4	5.2	12.9
After first trimester or no care	9.0	6.0	5.7	9.7	*	5.5	7.7	10.2	8.6	14.4
Second trimester	7.3	5.2	5.0	7.9	*	4.6	6.5	8.2	7.4	10.5
Third trimester	6.0	3.4	3.3	*	*	*	*	7.1	6.1	9.5
No prenatal care	38.4	23.0	19.7	49.2	*	29.3	36.5	45.5	36.4	57.9
Age of mother:										
Under 20 years	10.4	7.3	6.8	10.6	*	6.8	10.9	11.6	9.7	15.2
20-24 years	7.8	5.3	5.0	8.2	*	4.8	6.5	8.7	6.9	14.0
25-29 years	6.0	5.1	4.8	7.4	*	4.9	6.8	6.2	5.1	12.5
30-34 years	5.6	5.0	5.1	7.2	*	4.4	4.4	5.6	4.6	13.4
35-39 years	6.5	6.2	6.3	7.6	*	5.1	7.3	6.4	5.3	14.6
40-54 years	8.5	8.9	9.2	*	*	8.2	*	8.3	6.8	16.3

Table continued on next page.

MATERNAL NUTRITION AND INFANT MORTALITY IN THE CONTEXT OF RELATIONALITY



TABLE 1.3: INFANT MORTALITY RATES BY MATERNAL & INFANT CHARACTERISTICS, 2002 (CONTINUED)

TABLE 1.3: INFANT MORTALITY RATES BY MATERNAL & INFANT CHARACTERISTICS, 2002 (CONTINUED)										
Educational attainment of mother:										
0-8 years	6.6	5.3	5.1	11.5	*	5.8	7.6	10.4	9.9	15.2
9-11 years	9.6	6.1	5.7	9.7	*	6.0	7.4	11.7	9.9	15.9
12 years	7.8	5.6	5.3	8.8	*	4.7	7.4	8.4	6.9	13.6
13-15 years	6.0	4.9	5.0	6.0	*	4.3	5.3	6.1	4.8	11.9
16 years and over	4.2	4.0	4.1	3.9	*	4.4	*	4.2	3.7	10.0
Live-birth order:										
1	7.0	5.8	5.7	8.2	3.8	4.9	8.2	7.2	5.8	14.3
2	6.1	5.0	5.0	7.6	*	4.4	5.4	6.3	5.1	12.4
3	6.6	5.3	5.0	7.6	*	5.4	6.2	7.0	5.7	12.2
4	8.3	5.6	5.0	7.8	*	6.4	9.8	9.4	7.3	15.3
5 or more	11.1	7.9	7.4	13.8	*	7.7	*	12.3	9.1	18.8
Marital status:										
Married	5.4	5.0	5.0	6.9	3.0	4.4	5.8	5.5	4.9	11.8
Unmarried	9.9	6.4	6.0	9.1	5.4	5.9	8.9	11.2	8.8	14.8
Mother's place of birth:										
Born in the 50 States and DC	7.3	6.6	6.3	8.2	3.9	5.5	7.5	7.4	5.8	14.2
Born elsewhere	5.1	5.0	4.8	7.9	3.6	5.0	4.7	5.3	4.6	9.1
Maternal smoking during pregnancy:										
Smoker	11.1	10.7	9.8	12.4	*	*	10.7	11.1	9.7	20.1
Nonsmoker	6.6	5.6	5.4	7.9	3.5	4.9	6.8	6.8	5.2	13.2

Source: T. J. Mathews, F. Menacker, and M. F. MacDorman, Centers for Disease Control and Prevention, National Center for Health Statistics, "Infant mortality statistics from the 2002 period: linked birth/infant death data set," Natl Vital Stat Rep. 53, no. 10 (Nov. 24, 2004): 1-29.

TABLE 1.4: LEADING CAUSES OF INFANT MORTALITY IN THE UNITED STATES, 2002

Rank	Cause of death (based on the <i>International Classification of Diseases, Tenth Revision, 1992</i>)	Number	Percent of total deaths	Rate
	All causes	28,034	100.0	697.1
1	Congenital malformations, deformations and chromosomal abnormalities (Q00-Q99)	5,623	20.1	139.8
2	Disorders related to short gestation and low birthweight, not elsewhere classified (P07)	4,637	16.5	115.3
3	Sudden infant death syndrome (R95)	2,295	8.2	57.1
4	Newborn affected by maternal complications of pregnancy (P01)	1,708	6.1	42.5
5	Newborn affected by complications of placenta, cord and membranes (P02)	1,028	3.7	25.6
6	Accidents (unintentional injuries) (V01-X59)	946	3.4	23.5
7	Respiratory distress of newborn (P22)	943	3.4	23.4
8	Bacterial sepsis of newborn (P36)	749	2.7	18.6
9	Diseases of the circulatory system (I00-I99)	667	2.4	16.6
10	Intrauterine hypoxia and birth asphyxia (P20-P21)	583	2.1	14.5
	All other causes (Residual)	8,855	31.6	220.2

Source: T. J. Mathews, F. Menacker, and M. F. MacDorman, Centers for Disease Control and Prevention, National Center for Health Statistics, "Infant mortality statistics from the 2002 period: linked birth/infant death data set," Natl Vital Stat Rep. 53, no. 10 (Nov. 24, 2004): 1-29.

IV. LEADING CAUSES OF INFANT MORTALITY IN THE UNITED STATES

The ten leading causes of infant mortality in the United States are: 1) birth defects; 2) causes related to short gestation or low birthweight (LBW); 3) sudden infant death syndrome (SIDS); 4) maternal complications of pregnancy; 5) complications of placenta, cord, and membranes; 6) accidents; 7) respiratory

distress of newborn; 8) bacterial sepsis of newborn; 9) diseases of the circulatory system; and 10) intrauterine hypoxia and birth asphyxia (Table 1.4) (4). The first four causes account for more than half (50.8 percent) of all infant deaths; therefore, this report focuses on these four leading causes.



MATERNAL NUTRITION AND INFANT MORTALITY IN THE CONTEXT OF RELATIONALITY

**TABLE 1.5: INFANT DEATHS AND MORTALITY RATES FOR THE FIVE LEADING CAUSES OF INFANT DEATH, BY RACE AND HISPANIC ORIGIN OF THE MOTHER, UNITED STATES, 2002
(RATE PER 100,000 LIVE BIRTHS IN SPECIFIED GROUP)**

Cause of death (based on the <i>International Classification of Diseases, Tenth Revision, 1992</i>)	All races			Non-Hispanic white			Non-Hispanic black ¹			American Indian ^{2,3}			Asian or Pacific Islander ⁴		
	Rank	Number	Rate	Rank	Number	Rate	Rank	Number	Rate	Rank	Number	Rate	Rank	Number	Rate
All causes	...	27,970	695.4	...	13,327	579.9	...	8,031	1,388.6	...	366	864.8	...	1,006	477.2
Congenital malformations, deformations and chromosomal abnormalities (Q00-Q99)	1	5,630	140.0	1	2,999	130.5	2	987	170.6	1	80	188.1	1	225	106.8
Disorders related to short gestation and low birthweight, not elsewhere classified (P07)	2	4,636	115.3	2	1,769	77.0	1	1,828	316.0	3	46	108.0	2	161	76.4
Sudden infant death syndrome (R95)	3	2,295	57.1	3	1,269	55.2	3	642	110.9	2	52	123.3	4	51	24.3
Newborn affected by maternal complications of pregnancy	4	1,704	42.4	4	797	34.7	4	548	94.8	4	22	52.6	3	68	32.1
Newborn affected by complications of placenta, cord and membranes (P02)	5	1,013	25.2	5	491	21.3	6	308	53.2	9	7	*	6	32	15.0
Cause of death (based on the <i>International Classification of Diseases, Tenth Revision, 1992</i>)	Total Hispanic			Mexican			Puerto Rican ⁶			Central and South American ⁷					
	Rank	Number	Rate	Rank	Number	Rate	Rank	Number	Rate	Rank	Number	Rate			
All causes	...	4,927	562.0	...	3,399	541.6	...	471	818.9	...	637	505.6			
Congenital malformations, deformations and chromosomal abnormalities (Q00-Q99)	1	1,277	145.6	1	914	145.6	2	96	166.6	1	172	136.4			
Disorders related to short gestation and low birthweight, not elsewhere classified (P07)	2	759	86.6	2	503	80.1	1	97	168.6	2	93	74.1			
Sudden infant death syndrome (R95)	3	260	29.7	3	181	28.8	3	31	54.3	5	26	20.8			
Newborn affected by maternal complications of pregnancy ⁵	4	241	27.5	4	149	23.8	4	28	48.9	4	27	21.1			
Newborn affected by complications of placenta, cord and membranes (P02)	5	158	18.0	5	112	17.8	6	18	*	9	12*	*			

... Category not applicable

* Figure does not meet standard of reliability or precision; based on fewer than 20 deaths in the numerator.

1 For non-Hispanic blacks, respiratory distress of newborn was the fifth leading cause of death, with 319 deaths and a rate of 55.1.

2 Includes Aleuts and Eskimos.

3 For American Indians, accidents (unintentional injuries) was the fifth leading cause of death; however, with only 16 deaths, a reliable infant mortality rate could not be computed.

4 For Asian or Pacific Islanders, disease of the circulatory system was the fifth leading cause of death, with 34 deaths and a rate of 16.2.

5 Cause-of-death coding changes may affect comparability with the previous year's data for this cause.

6 For Puerto Ricans, respiratory distress of newborn was the fifth leading cause of death, with 20 deaths and a rate of 35.1.

7 For Central and South Americans, respiratory distress of newborn was the third leading cause of death, with 32 deaths and a rate of 25.1.

Note: Reliable cause-specific infant mortality rates cannot be computed for Cubans because of the small number of infant deaths (53).

Source: T. J. Mathews, F. Menacker, and M. F. MacDorman, Centers for Disease Control and Prevention, National Center for Health Statistics, "Infant mortality statistics from the 2002 period: linked birth/infant death data set," *Natl Vital Stat Rep.* 53, no. 10 (Nov. 24, 2004): 1-29.



The leading causes of infant mortality varied by race and ethnicity, according to 2002 data (Table 1.5) (4). Birth defects were the leading cause of infant deaths for all racial and ethnic groups, except among infants born to non-Hispanic black mothers and Puerto Rican mothers, for whom diagnoses related to short gestation and LBW were the leading cause of infant deaths. Infants born to non-Hispanic black mothers had the highest IMR due to prematurity and LBW, whereas infants born to American Indians had the highest rates of birth defects and SIDS. In fact, these two causes account for more than one-third of all infant deaths among infants born to American Indian mothers.

V. MATERNAL NUTRITION AND INFANT MORTALITY

Every single cell, organ, and system inside a newborn baby comes mostly from her mother's food intake before or during pregnancy. Maternal nutrition is a critical determinant of infant health; thus, it is not hard to see that poor maternal nutrition can contribute, directly or indirectly, to infant mortality. In this section, we begin to examine the relationship between maternal nutrition and the four leading causes of infant mortality in the United States: birth defects; LBW and its twin constituents, preterm birth and fetal growth restriction; SIDS; and maternal complications of pregnancy.

A. MATERNAL NUTRITION AND BIRTH DEFECTS

Birth defects account for one in five (20.1 percent) infant deaths in the United States. Nutritional deficiencies during organogenesis can cause birth defects in susceptible individuals. Because organogenesis occurs early in pregnancy (e.g., the heart begins to beat at 22 days post-conception, and the neural tube is closed by 28 days post-conception), nutritional deficiencies before and in early pregnancy are most harmful to the developing fetus. It is generally accepted now that inadequate intake of natural folate, or its synthetic form, folic acid, before and during early pregnancy, is associated with an increased risk of spina bifida, anencephaly, and other neural tube defects (11). Case-control studies, randomized clinical trials, and community-based interventions with vitamin supplements have shown that the failure to consume folic acid supplements or folic acid-containing multivitamins increases the risk of having an affected child by twofold to eight-fold (12-19). The mechanism underlying the association between neural tube defects and folate deficiency has not been established, but probably involves disruption of nucleic acid synthesis and a range of methylation reactions

(11). Disruptions in folate metabolism can also result in raised homocysteine concentrations, which are teratogenic to the neural tube in some animal models (11, 20). Nutrient deficiencies other than folate, in particular vitamin B12, might also be associated with neural tube defects (11, 21-23). Deficiencies in B vitamins, vitamin K, magnesium, copper, and zinc have also been linked to other birth defects (24). Certain nutritional excesses, such as vitamin A, can also cause birth defects. In women with pregestational diabetes, poor dietary control of blood sugar during critical periods of organogenesis significantly increases the risk of birth defects in their offspring, particularly cardiac and neural tube defects (25).

B. MATERNAL NUTRITION AND LOW BIRTHWEIGHT

Low birthweight is a leading cause of infant and childhood mortality and morbidity in the United States (4). Its twin constituents are preterm birth and fetal growth restriction. The evidence linking poor maternal nutrition to preterm birth and fetal growth restriction is examined below.

Maternal Nutrition and Preterm Birth

In 2004, the 12.5 percent of infants born at less than 37 weeks accounted for 75 percent of perinatal deaths (1). Almost one-fifth of all infants born at less than 32 weeks' gestation do not survive the first year of life, compared with about one percent of infants born between 32 and 36 weeks and 0.3 percent of infants born 37-41 weeks' gestation. The IMR per 1,000 live births for infants born at less than 32 weeks was 180.9, nearly 70 times the rate for infants born between 37 and 41 weeks (26). Preterm birth was also a leading cause of racial-ethnic disparities in infant mortality. In 2003, infants born to non-Hispanic black mothers were about 1.6 times as likely to be born preterm (less than 37 weeks) and 2.5 times as likely to be born very preterm (less than 32 weeks), compared to non-Hispanic white mothers (1, 4). The two-and-a-half-fold difference in very preterm birth rates accounted for more than two-thirds of excess black infant deaths (4, 27).

Low prepregnancy body mass index (BMI) and poor gestational weight gain have been linked to greater risk for preterm birth, with adjusted relative risks in the range of 1.5 to 2.5 (28-31). A number of nutrient deficiencies have been implicated in the pathogenesis of preterm birth, including zinc, iron, folate, and calcium, although causal relationships are difficult to establish. While it is not clear whether specific nutritional deficiencies can cause preterm birth, a number of nutritional factors could theoretically affect the mechanisms controlling the initiation of



labor. Four major pathways to spontaneous preterm labor have been proposed involving 1) activation of the maternal or fetal hypothalamic-pituitary-adrenal axis, 2) decidual-chorioamniotic or systemic inflammation, 3) decidual hemorrhage (i.e., abruption), and 4) pathological distention of the uterus (32). Nutrition could mediate or modulate several of these four pathways. For example, omega-3 polyunsaturated fatty acids (PUFA) have been found to depress the biosynthesis of 2-series prostaglandins and leukotrienes that are centrally involved in the inflammatory pathway (33), and fish oil supplementation containing high amounts of omega-3 has been shown to reduce the recurrence risk of preterm birth (34-36). A recent study showed that modest periconceptional undernutrition induces preterm delivery in the sheep model (37), apparently mediated via premature activation of the fetal hypothalamic-pituitary-adrenal (HPA) axis and acceleration of the “placental clock” (38-39). Maternal fasting during pregnancy has also been associated with greater risk for preterm birth, mediated via increased placental corticotropin-releasing hormone (CRH) gene expression and premature fetal HPA activation (40). Thus, it appears that nutrient deficiencies can activate one or more pathways to preterm birth and that nutritional supplementation can potentially modulate these pathways. More studies are needed, however, to elucidate the relationship between nutrition and preterm birth.

Nutrition and Fetal Growth Restriction

Fetal growth restriction is one of the twin constituents of LBW; it is indicated by small-for-gestational-age (SGA) at birth, although there are other causes of SGA. In the developing world, maternal malnutrition is an important cause of SGA. Low prepregnancy BMI, commonly used as an indicator of poor maternal nutritional status before pregnancy, is associated with greater risk for SGA (28-31). A recent study in China found that, compared with a normal BMI, being severely underweight before pregnancy was associated with mean reductions of 219 ± 40 g in infant birthweight and an 80 percent increase in the risk of fetal growth restriction (41). Observational studies have reported that gestational weight gain is also strongly and positively associated with fetal growth (29-31, 42-43). Moreover, these associations are stronger in undernourished women—i.e., those with low prepregnancy BMI. Michael Kramer and colleagues at McGill University estimated that low prepregnancy BMI and low gestational weight gain can account for up to 25 percent of cases of fetal growth restriction in a developed country (30). A number of macro- and micronutrient deficiencies have also been implicated in fetal growth restriction, although causal relationships have not been clearly established.

C. MATERNAL NUTRITION AND SUDDEN INFANT DEATH SYNDROME

The relationship between maternal malnutrition and sudden infant death syndrome (SIDS) is not well established. Maternal malnutrition is a risk factor for LBW, which in turn is a risk factor for SIDS (44). Micronutrient deficiencies during pregnancy, such as magnesium deficiency, have been linked to SIDS (45), but supportive evidence is far from conclusive.

D. MATERNAL NUTRITION AND COMPLICATIONS OF PREGNANCY

While a number of severe maternal complications of pregnancy can cause fetal or infant deaths, in this section we focus on the big three: hemorrhage, infection, and preeclampsia. This classic H.I.T. triad (hemorrhage, infection, and toxemia, which is an old term for preeclampsia) accounts for about half of all maternal deaths and a substantial portion of fetal and infant deaths due to maternal complications in developed and developing countries.

Nutrition and Gestational Hypertension/Preeclampsia

Between 1991 and 1999, preeclampsia accounted for 16 percent of all pregnancy-related maternal deaths in the United States (46). The contribution of nutrient deficiencies or excesses to gestational hypertension/preeclampsia is unknown. For many years, diet has been suggested to play a role in preeclampsia; however, rarely were these hypotheses appropriately tested (47). Cross-sectional and case-control studies in the past have shown association with deficiencies in a number of nutrients, including calcium, antioxidants, and fish oil; however, a recent prospective cohort study found an absence of a relationship between 28 nutritional factors analyzed and gestational hypertension/preeclampsia (48). Specifically, the study found no evidence that these hypertensive disorders were related to a diet low in calcium, high in sodium, low in protein, low in antioxidants, or low in zinc during pregnancy. Most of the study participants, however, had received multivitamins during pregnancy and probably did not have severe micronutrient deficiencies.

Based on current knowledge of the pathophysiology of preeclampsia, Roberts et al. (47) identified several new targets for nutritional investigation related to preeclampsia: 1) nutrients potentially affecting endothelial function, such as arginine as the precursor to nitric oxide or oleic acid to decrease endothelial expression of vascular cell adhesion molecule (V-CAM); 2) nutrients such as saturated or trans-fatty acids that contribute to oxidative stress, or deficiencies in nutrients that protect



against oxidative stress (e.g., ascorbate, vitamin E, carotenoids, and zinc); 3) nutrients that modulate inflammatory response (e.g., vitamin E, omega-3 polyunsaturated fatty acids); and 4) nutrients that improve insulin action (e.g., chromium, omega-3 PUFA). Most importantly, special attention should be directed at periconceptional nutrition in light of the importance of abnormal implantation and placentation in the pathogenesis of preeclampsia (49-50). Implantation and placentation begin early in pregnancy (approximately a week after conception) and are thought to be regulated, in part, by immunologic mechanisms (51-53), which in turn may be modulated by macro- and micronutrients (47). Thus, there may be a critical window of opportunity in early pregnancy for the prevention of preeclampsia.

Nutrition and Hemorrhage/Anemia

Between 1991 and 1999, hemorrhage accounted for 17 percent of all pregnancy-related maternal deaths in the United States (46). The contribution of nutrient deficiencies or excesses to maternal hemorrhage is not known. Overweight and obesity are an important risk factor for maternal complications including hemorrhage, infections, and preeclampsia. Some reports have noted low serum zinc levels to be associated with abnormalities of labor, such as prolonged labor and uterine atony which could result in postpartum hemorrhage (54), while others (55, 56) have failed to show any such association.

While it is not known whether nutritional factors play a direct role in the pathogenesis of maternal hemorrhage, anemia can certainly contribute to morbidities associated with hemorrhage. Anemia is quite common in the United States; about one of every three U.S. women is anemic in the third trimester of pregnancy. Nutritional deficiencies of iron, folate, and vitamins A, B6, and B12 can cause anemia (57). Iron deficiency can result from both low intake of heme iron from animal sources and low bioavailability of non-heme iron from cereal/legume-based diets, often exacerbated by dietary intake of phytate, fiber, or other iron absorption inhibitors, including tannins in tea or calcium. Iron and/or folate supplementation during pregnancy has been shown to reduce anemia at birth and six months postpartum (58). Several studies have also shown that iron stores at conception are a strong predictor of maternal iron status and risk of anemia in later pregnancy (59-61). Thus, improving women's iron stores before pregnancy and iron and/or folate intake during pregnancy can prevent morbidities associated with maternal hemorrhage.

Nutrition and Infections/Inflammation

Between 1991 and 1999, maternal infections accounted for 13 percent of all maternal deaths in the United States (46). During the course of an ascending intrauterine infection, microorganisms may reach the decidua, membranes, amniotic fluid, and even the fetus (62). Severe infection can lead to fetal and neonatal sepsis and death. Maternal infection/inflammation may also be responsible for the majority of very preterm births (62). In light of the striking impact of deficiencies of micronutrients such as vitamin A and zinc on immune function, morbidity, and mortality in children, it seems reasonable to suggest that such deficiencies might play a contributing role in maternal infections during pregnancy (63). Surprisingly little has been published on the relationship between maternal nutritional status and susceptibility to infection during pregnancy. A prospective study of over 1,000 pregnant women in Camden, New Jersey, found that iron and folate deficiencies in early pregnancy were associated with high serum ferritin concentration in the third trimester, which in turn was associated with maternal infection and preterm premature rupture of membranes (64). Vitamin A deficiencies have also been implicated in maternal infections, and vitamin A supplementation has been shown to reduce placental parasitemia and infant mortality from maternal malarial infections in populations with subclinical vitamin A deficiency (65), although the evidence is far from conclusive.

Maternal infections typically provoke a local or systemic inflammatory response; excessive inflammation can cause preterm birth and has been implicated in many of the disorders common to preterm infants, including chronic lung disease, intraventricular hemorrhage, periventricular leukomalacia, retinopathy of prematurity, necrotizing enterocolitis, and bronchopulmonary dysplasia (66, 67). As discussed earlier, nutrition can play a major role in modulating inflammation, and fish oil supplementation high in omega-3 polyunsaturated fatty acids (PUFA) has been shown to reduce the recurrence risk of preterm birth in women with prior preterm birth. Inflammation can also cause oxidative stress to the body. Oxidative stress occurs when the production of damaging free radicals and other oxidative molecules exceeds the capacity of the body's antioxidant defenses to detoxify them. Oxidative stress has been implicated in a number of pregnancy complications, including preeclampsia, fetal growth restriction, and preterm rupture of fetal membranes (68-70). Antioxidants can be categorized as either free radical scavengers that trap or decompose existing free radicals, or cellular and extracellular enzymes that inhibit peroxidase reactions involved in the production of free radicals (71). Free radical scavengers include vitamin C (ascorbate), vitamin E



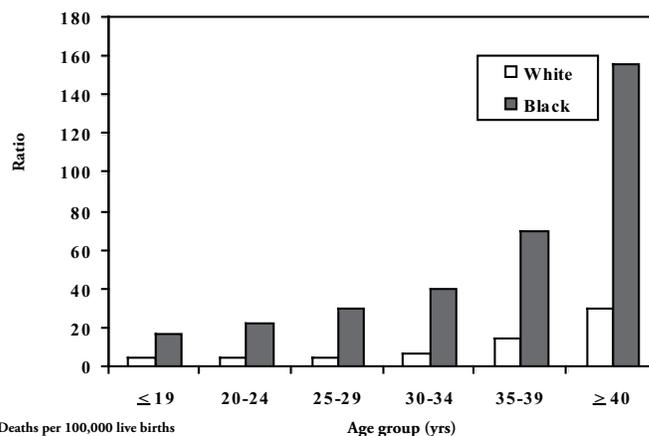
(tocopherols), carotenoids, and glutathione. Antioxidant enzymes include glutathione peroxidase, superoxide dismutase, and catalase, which are dependent on the presence of co-factors such as selenium, zinc, and iron (72). Thus, maternal nutrition can potentially play a major role in modulating inflammation and oxidative stress from maternal infections.

Nutrition and Maternal Mortality

Between 1991 and 1999, a total of 4,200 deaths in the U.S. were determined to be pregnancy-related (46). The overall pregnancy-related mortality ratio (PRMR) was 11.8 deaths per 100,000 live births and ranged from 10.3 in 1991 to 13.2 in 1999. This ratio has not improved in more than two decades. Significant racial-ethnic disparities in maternal mortality persist; PRMR is 3.7 times higher for non-Hispanic black women (PRMR = 30.0) than for non-Hispanic white women (PRMR = 8.1). The disparities are particularly striking by age groups (Figure 1.3), supporting the aforementioned “weathering hypothesis,” which posits that the effects of social inequality on health compound with age, leading to growing gaps in the reproductive health outcomes of black and white women as they transition from young to middle adulthood.

The contributions of poor nutrition to maternal mortality in the U.S. are not known; in developing countries, maternal malnutrition almost certainly plays an important role in several of the leading causes of maternal death, including preeclampsia, puerperal infections, hemorrhage, and obstructed labor (the latter due to short stature and small pelvis from malnutrition in childhood and adolescence) (73). Few nutritional supplementation trials have reported maternal mortality as an outcome. West et al. (74) recently completed an important trial in Nepal. Using a cluster randomized design, 270 communities were randomized to three groups, and all women of reproductive age in each community were given a weekly placebo, 7000 µg of vitamin A, or 42 mg of β-carotene for over three-and-a-half years. Pregnancy-related mortality was lowered by 40 percent ($P < 0.04$) among those who received vitamin A and by 49 percent ($P < 0.01$) among those who received β-carotene (although differences in cause of death could not be reliably distinguished between supplemented and placebo groups). The study is notable for supplementation that begins before pregnancy; the protective impact was established after one-and-a-half years of supplementation, reflecting a potential duration of dosing a population by which a clear mortality reduction could be expected. More studies are needed in the U.S., particularly in the context of addressing racial-ethnic disparities in maternal mortality.

FIGURE 1.3: PREGNANCY-RELATED MORTALITY RATIOS, BY AGE AND RACE UNITED STATES, 1991-1999



* Deaths per 100,000 live births

Source: J. Chang, L. D. Elam-Evans, C. J. Berg, J. Herndon, L. Flowers, K. A. Seed, and C. J. Snyerson, “Pregnancy-related mortality surveillance—United States, 1991–1999,” *MMWR Surveill Summ.* 52, no. 2 (Feb. 21, 2003): 1-8.

VI. MATERNAL NUTRITION AND CHILD HEALTH, GROWTH, AND DEVELOPMENT

Although the focus of this report is on infant mortality, the impact of maternal nutrition goes beyond immediate birth outcomes and infancy. Maternal nutritional status and behaviors before and during pregnancy can have lasting influences on child health, growth, and development. This section briefly examines the evidence for the lasting influences of maternal nutrition.

MATERNAL NUTRITION AND CHILD HEALTH

Maternal nutrition has been linked to child health in several ways. Maternal anemia strongly affects the iron stores of the infant at birth (61). In Indonesia, de Pee et al. observed that, compared to a normal birthweight infant born to a mother without anemia, a similar infant born to an anemic mother had a 1.8 times greater risk of developing anemia by three to five months of age (75). The highest prevalence of anemia at three to five months occurred in LBW infants whose mothers were anemic during pregnancy, with an odds ratio of 3.7 compared to normal birthweight infants born to nonanemic mothers. A substantial number of both observational (76, 77) and iron intervention (78, 79) trials support this relation between maternal and infant iron status. As discussed earlier, poor maternal nutrition has been implicated in the pathogenesis of preterm birth and fetal growth restriction, which in turn are significant predictors of childhood morbidities, including childhood anemia and infection (80, 81). In a review to quantify the risks of mortality and morbidity associated with fetal growth restriction, Ashworth found an increased risk of



diarrhea and pneumonia in term infants weighing less than 2500 grams who were drawn from nine studies (82). These findings are corroborated by Huttly et al., who reviewed six studies on the association between LBW and diarrheal morbidity (83). The direct relationship between maternal nutrition status and childhood morbidity remains obscure; further studies are needed to elucidate this relationship, particularly in the context of racial-ethnic disparities in early childhood diseases in the U.S.

MATERNAL NUTRITION AND CHILD GROWTH

Most studies linking maternal malnutrition to poor child growth have been conducted in the setting of developing countries. A substantial body of literature suggests that maternal malnutrition is one of several factors contributing to postnatal stunting (84-85). Neumann and Harrison found that maternal prepregnancy BMI, lean body mass, and gestational weight gain were strong determinants of the infants' size at birth and during the first six months of life (86). In childhood and adolescence, growth failure continues to prevail, further reducing the chances of girls to reach an optimal body size with adequate nutrient stores before conception, which ultimately leads to birth of LBW infants. This pattern generates an intergenerational cycle of undernutrition (87-88). What this means in the U.S. context, where less than half of all women enter pregnancy with normal BMI and only about one-third of low-income pregnant women achieve ideal weight gain during pregnancy (see Chapter 2), needs further exploration.

MATERNAL NUTRITION AND CHILD DEVELOPMENT

Maternal malnutrition can contribute to poor child development. Undernutrition during pregnancy has been associated with smaller head circumference and brain weight (89, 90). As discussed earlier, poor maternal nutrition is an important risk factor for LBW, fetal growth restriction, and preterm birth, and studies have identified a relationship between these birth outcomes and later cognitive delays. In Hack's review of twelve longitudinal observational studies of children between nine and seventeen years of age who were small-for-gestational-age (SGA) at birth, all the studies indicated that SGA infants had poorer cognition than normal-birthweight children, although some of the differences were not statistically significant (90). Maternal iodine deficiency has also been linked to a wide spectrum of neuropsychological disorders in children, ranging from subclinical deficits in cognitive motor and auditory functions to hypothyroid-induced cognitive impairment in infants. A recent study in China found that infants whose mothers had subclinical

prenatal iodine deficiency had poorer information processing skills at seven months of age and lower scores on the cognitive development index at thirteen months of age (91). A meta-analysis of studies conducted in China found significant cognitive deficits in children exposed to maternal iodine deficiency *in utero*, as demonstrated by a loss of 12.45 IQ points; maternal iodine supplementation before and during pregnancy was associated with a recovery of 8.7 IQ points (92). Maternal iodine deficiency is uncommon in the United States; however, the impact of current public health recommendations for dietary salt restriction, particularly in populations where iodine intake from other food sources is low, needs further examination.

VII. NUTRITION AND DEVELOPMENTAL PROGRAMMING OF CHRONIC ADULT DISEASES

A growing body of evidence suggests that predisposition to a host of chronic adult diseases may be acquired very early in development through inappropriate fetal or neonatal nutrition (93). Most of these investigations have been stimulated by the "fetal origins" hypothesis proposed by Barker and colleagues, which showed a relationship between birthweight and adulthood hypertension (94), insulin resistance (95), vascular dysfunction (96), obesity (97), and dyslipidemia (98). The thrifty phenotype hypothesis has evolved as a result of further investigation, and "developmental programming" is now more commonly ascribed to any situation where a stimulus or an insult, at a sensitive or critical period of development, has lasting or lifelong impact on health or function (93, 99). For example, maternal malnutrition during the critical period of fetal pancreatic development can lead to poor development of pancreatic β -cell mass and function, which can increase the fetus' susceptibility to diabetes mellitus in later life. The importance of maternal nutrition, as well as the effect that poor nutrition may have on birthweight and subsequent adult diseases, is suggested by studies of exposure to famine, most notably the Dutch Hunger Winter (100-102). However, for the most part, little information is available about the specific role of maternal nutritional status on fetal programming, since nutritional status has often been inferred from birthweight rather than directly assessed (103). Moreover, these epidemiological studies have attracted increasing criticism largely because of their retrospective nature and inability to control for potential confounders.

Further evidence for developmental programming of chronic adult diseases has now been suggested by a growing body of animal studies in which the fetal environment has been manipulated through altered maternal dietary intake (93). For



example, hypertension has been reliably reproduced in the offspring of laboratory animals deprived of protein, calories, and iron during pregnancy (104-107). The mechanisms underlying these changes are thought to include programmed alterations in the hypothalamic-pituitary-adrenal (HPA) and renin-angiotensin axes, and/or diminished nephron number with a subsequent decrease in renal mass (108-111). Additionally, these offspring have impaired vascular endothelial cell dilatation (112-113), which may be related to elevated endothelin-1, alterations in aortic elastin/collagen, and reduced expression of angiogenic proteins (114-115). In a similar fashion, glucose intolerance has been evoked after *in utero* deprivation, the etiology of which includes diminished pancreatic β -cell mass and subsequent abnormalities in glucose secretion. In addition, hepatic glucokinase activity is decreased while phosphoenolpyruvate carboxykinase (PEPCK) activity is increased, contributing to an increase in hepatic gluconeogenesis noted in the offspring of protein-deprived mothers (116). Ross and colleagues performed studies among pregnant rats and sheep in which drought conditions were simulated via maternal dehydration, and famine conditions via nutrient restriction. Maternal dehydration resulted in LBW offspring, which demonstrated gender-specific plasma hypernatremia and hypertonicity and arterial hypertension. Nutrient restriction during pregnancy also resulted in LBW offspring. If permitted rapid catch-up growth by nutrient availability, these offspring demonstrated evidence of increased body weight and body fat and leptin resistance as adults, consistent with the “thrifty phenotype” hypothesis. Conversely, if the catch-up growth is delayed by nutrition restriction, the offspring exhibit normal body weight, body fat, and plasma leptin levels as adults (117). While most studies have examined the impact of maternal undernutrition, more recently Armitage and colleagues provided intriguing evidence in animal models that maternal overnutrition, in the form of a fat-rich diet, may be deleterious to the health of offspring and can result in a phenotype of the offspring that is characteristic of metabolic syndrome. These animal studies are beginning to elucidate the mechanisms by which poor maternal nutrition could “program” fetal risk for subsequent adult diseases; however, their relevance to the human context must be interpreted with caution (93).

SUMMARY

We found good evidence linking poor maternal nutrition to several leading causes of infant mortality, including birth defects, preterm birth, fetal growth restriction, and maternal complications of pregnancy (preeclampsia, anemia, infections/inflammation). Maternal folate and vitamin B12 deficiencies have been associated with neural tube defects, while deficiencies in B vitamins, vitamin K, magnesium, copper, and zinc have also been linked to other birth defects. Low prepregnancy body mass index (BMI) and poor gestational weight gain are associated with greater risk for preterm birth and fetal growth restriction. Maternal nutrition can also mediate or modulate several of the major pathways (e.g., inflammatory) leading to spontaneous preterm birth. While the contribution of specific nutrient deficiencies to preeclampsia remains unclear, maternal nutrition can potentially play an important role in the pathogenesis of preeclampsia by affecting endothelial function, exacerbating or ameliorating oxidative stress, modulating inflammatory response, and improving insulin action. In light of the importance of abnormal implantation and placentation in the pathogenesis of preeclampsia, periconceptional nutrition may be of paramount importance. Nutritional deficiencies of iron, folate, and vitamins A, B6, and B12 can cause anemia. Vitamin A and other micronutrient deficiencies have been implicated in maternal infections, and antioxidants can potentially play a major role in modulating inflammation and oxidative stress from maternal infections. The growing body of research on fetal programming of adult diseases further elevates the clinical and public health significance of maternal nutrition.



CHAPTER 2

NUTRITIONAL STATUS AND BEHAVIORS OF PREGNANT WOMEN IN THE UNITED STATES

Chapter 1 examined the relationship between maternal nutrition and infant mortality. We found evidence suggesting that poor maternal nutrition may contribute to several leading causes of infant mortality and may also play a role in poor child health and development, as well as developmental programming of adult diseases. Many of these studies, however, were conducted outside the United States, often in the context of severe maternal malnutrition in developing countries. Their relevance to the U.S. population is unclear.

This chapter examines the nutritional status and behaviors of pregnant women in the United States, with special emphasis on racial-ethnic disparities. We assess the ABCD of nutritional status: anthropometry, biochemical, clinical, and dietary. For dietary assessment, we also describe the content and pattern of dietary intakes for pregnant women in the United States.

I. ANTHROPOMETRIC ASSESSMENT

Anthropometry is the comparative study of body size or measurements. In research it is commonly operationalized as body mass index (BMI), a measurement of body size that takes into account both weight and height. For pregnant women, anthropometry also includes measurements of weight gain during pregnancy.

Both low prepregnancy BMI and poor weight gain during pregnancy have been associated with increased risk for preterm birth and fetal growth restriction (1-3), which are important causes of infant mortality. In the Preterm Prediction Study, low prepregnancy BMI was strongly associated with an increased risk of preterm birth, with relative risk above 2.5 (4).

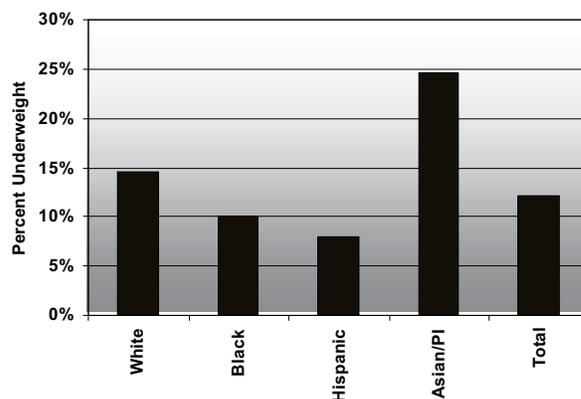
Both high prepregnancy BMI and excessive weight gain during pregnancy have been associated with increased risk for multiple pregnancy complications, including gestational diabetes, preeclampsia, chorioamnionitis, and postpartum hemorrhage (5-7). For the mother, weight gain during pregnancy predicts postpartum weight retention (8), which may have implications for her long-term health. For the fetus, maternal obesity has been linked to neural tube defect (9-10) and fetal death (11), and both high prepregnancy BMI and weight gain during pregnancy have been associated with increased risk for fetal macrosomia and shoulder dystocia (7). While protective against spontaneous preterm deliv-

ery, maternal obesity is a risk factor for indicated preterm delivery because of the increased risk for maternal complications such as preeclampsia (4).

UNDERWEIGHT

Underweight is defined as a BMI below 19.8 prior to pregnancy (1, 12). The lower a woman's BMI, the more likely she is to be undernourished. According to data from the 2003 CDC Pregnancy Nutrition Surveillance System of nearly 728,000 low-income pregnant women, approximately one of every eight (12.1 percent) pregnant women was underweight right before pregnancy (12). This is comparable to a recent report based on the 2000-2002 Pregnancy Risk Assessment Monitoring System (PRAMS) survey of over 6,000 socio-economically diverse mothers in Colorado, which reported a weighted prevalence of maternal underweight of 15.4 percent (13). There are significant racial-ethnic disparities in maternal underweight; one in four Asian and Pacific Islander mothers was underweight before pregnancy, compared to about one in ten African American and one in twelve Latina mothers (Figure 2.1) (12). The prevalence of maternal underweight has been decreasing for all racial and ethnic groups (12).

FIGURE 2.1: MATERNAL UNDERWEIGHT BEFORE PREGNANCY, BY MATERNAL RACE & ETHNICITY, 2003



Source: Centers for Disease Control and Prevention, Pediatric and Nutrition Surveillance System, <http://www.cdc.gov/pednss/index.htm> (accessed August 9, 2006).

OVERWEIGHT

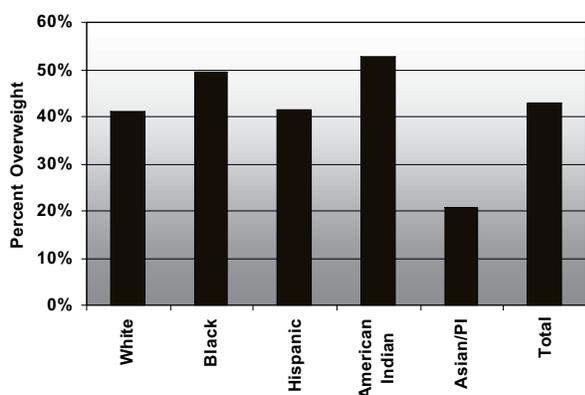
Overweight is defined as a BMI greater than 26.0 up to 29.0 (12), whereas obese is defined as a BMI greater than 29.0 (1, 12). According to data from the 2003 CDC Pregnancy Nutrition Surveillance System, 14.5 percent of low-income pregnant women were overweight and 28.5 percent were obese (12). The prevalence of maternal overweight and obesity has increased



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from 29.8 percent in 1989 to 43 percent in 2003 (12). There are significant racial-ethnic disparities in maternal overweight and obesity. Approximately half of low-income African American and Native American pregnant women, and over 40 percent of white and Latina pregnant women, were overweight or obese before pregnancy, compared to about 20 percent for Asian and Pacific Islanders (Figure 2.2). The prevalence of maternal overweight and obesity has been increasing for all racial-ethnic groups—by more than 10 percent over the past decade for Native Americans and African Americans (12).

FIGURE 2.2: MATERNAL OVERWEIGHT AND OBESITY BEFORE PREGNANCY, BY MATERNAL RACE & ETHNICITY, 2003



Source: Centers for Disease Control and Prevention, Pediatric and Nutrition Surveillance System, <http://www.cdc.gov/pednss/index.htm> (accessed August 9, 2006).

LESS THAN IDEAL WEIGHT GAIN DURING PREGNANCY

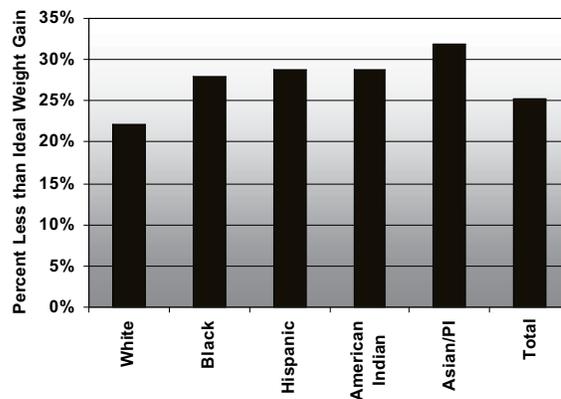
A 1990 Institute of Medicine (IOM) report recommended ideal weight gain for pregnant women based on their prepregnancy BMI (1). The recommendations for weight gain during pregnancy are summarized in Table 2.1.

TABLE 2.1: RECOMMENDED IDEAL WEIGHT GAIN FOR PREGNANT WOMEN, BY PREPREGNANCY BMI (INSTITUTE OF MEDICINE, 1990)		
Weight	Prepregnancy BMI	Total Weight Gain (lb)
Underweight	<19.8	28–40
Normal weight	19.8–26.0	25–35
Overweight	>26.0–29.0	15–25
Obese	>29	At least 15

Source: Centers for Disease Control and Prevention, Pediatric and Nutrition Surveillance System, <http://www.cdc.gov/pednss/index.htm> (accessed August 9, 2006).

Less than ideal weight gain is defined as a total weight gain below the lower limits of that recommended by IOM for each prepregnancy BMI classification. For example, less than ideal weight gain for women who were underweight before pregnancy is less than 28 lbs, whereas that for women who were overweight or obese is less than 15 lbs for the entire pregnancy. According to data from the 2003 CDC Pregnancy Nutrition Surveillance System, approximately one of every four (25.2 percent) low-income pregnant women in the U.S. had less than ideal weight gain during pregnancy (12). There are significant racial-ethnic disparities in less than ideal weight gain during pregnancy, with about 22 percent of non-Hispanic whites and nearly 32 percent of Asian and Pacific Islander women achieving less than ideal weight gain during pregnancy (Figure 2.3). Overall, there has been a substantial decline in the prevalence of less than ideal weight gain during pregnancy, from 35.6 percent in 1989 to 25.2 percent in 2003. For non-Hispanic black women and Hispanic women, the prevalence of less than ideal weight gain was higher than 40 percent in 1997; the prevalence has since declined to 28 percent and 29 percent, respectively, by 2003 (12).

FIGURE 2.3: LESS THAN IDEAL WEIGHT GAIN DURING PREGNANCY, BY MATERNAL RACE & ETHNICITY, 2003



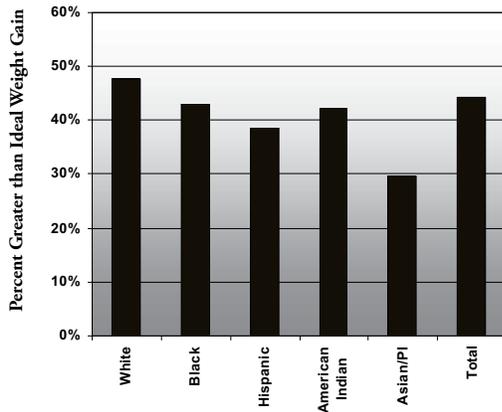
Source: Centers for Disease Control and Prevention, Pediatric and Nutrition Surveillance System, <http://www.cdc.gov/pednss/index.htm> (accessed August 9, 2006).

GREATER THAN IDEAL WEIGHT GAIN

Greater than ideal weight gain is defined as a total weight gain that exceeds the upper limit of that recommended by IOM for each prepregnancy BMI classification (1, 12). For example, greater than ideal weight gain for women who were underweight before pregnancy is greater than 40 lbs, whereas that for women who were overweight is more than 25 lbs for the entire pregnancy. According to data from the 2003 CDC Pregnancy Nutrition



FIGURE 2.4: GREATER THAN IDEAL WEIGHT GAIN DURING PREGNANCY, BY MATERNAL RACE & ETHNICITY, 2003



Source: Centers for Disease Control and Prevention, Pediatric and Nutrition Surveillance System, <http://www.cdc.gov/pednss/index.htm> (accessed August 9, 2006).

Surveillance System, 44.2 percent of low-income pregnant women in the U.S. had greater than ideal weight gain during pregnancy (12). The prevalence of greater than ideal weight gain during pregnancy has increased substantially in the U.S., from 32 percent in 1989 to 44.2 percent in 2003. There are significant racial-ethnic disparities in greater than ideal weight gain during pregnancy; nearly half (47.7 percent) of non-Hispanic white women and over 40 percent of non-Hispanic black and Native American women gain more than the recommended weight gain during pregnancy, compared to about 30 percent for Asian and Pacific Islander women (Figure 2.4) (12).

SUMMARY: PREPREGNANCY WEIGHT AND WEIGHT GAIN DURING PREGNANCY

In 2003, one in eight low-income U.S. women were underweight before pregnancy, and one in four had less than ideal weight gain. Both low prepregnancy BMI and poor weight gain during pregnancy have been associated with increased risk for preterm birth and intrauterine growth restriction, which are important causes of infant mortality. Nearly half of low-income U.S. women were overweight or obese before pregnancy (43 percent), or gained excessive weight during pregnancy (44 percent). Both high prepregnancy BMI and excessive weight gain during pregnancy are associated with a number of important maternal and fetal complications. While the trend of maternal underweight and low weight gain during pregnancy has been declining in the past decade, that for maternal overweight and excessive weight gain during pregnancy has been increasing. There are significant racial-ethnic disparities in maternal anthropometry. Asian Pacific Islander women have the highest prevalence of low prepregnancy BMI and inadequate weight gain during pregnancy; women of color in general are substantially more likely to have less than ideal weight

TABLE 2.2. PREPREGNANCY BMI AND WEIGHT GAIN DURING PREGNANCY AMONG LOW-INCOME WOMEN IN THE UNITED STATES, BY MATERNAL RACE-ETHNICITY, AGE, AND EDUCATION, 2003

Race/Ethnicity	Prepregnancy BMI (3)			Weight Gain (4)		
	Number	Under-weight	Over-weight	Number	< Ideal	> Ideal
		%	%		%	%
White, Not Hispanic	332,590	14.5	41.2	287,821	22.1	47.7
Black, Not Hispanic	164,001	10.0	49.6	138,220	28.0	42.9
Hispanic	143,724	7.9	41.4	121,443	28.7	38.4
American Indian/Alaskan Native	8,747	8.9	52.6	6,451	28.8	42.2
Asian/Pacific Islander	12,601	24.6	20.9	10,843	31.8	29.6
All Other/Unknown	17,336	13.7	38.1	14,493	23.8	44.6
Total	678,999	12.1	43.0	579,271	25.2	44.2
Age						
< 15 Years	2,940	20.3	20.0	2,468	25.5	45.9
15 - 17 Years	47,505	20.9	23.9	40,057	23.8	46.6
18 - 19 Years	91,971	17.8	31.0	77,831	22.2	48.7
20 - 29 Years	407,482	11.5	44.5	348,990	24.9	44.3
30 - 39 Years	120,748	6.6	54.2	102,999	28.7	39.7
>= 40 Years	8,319	5.8	56.1	6,903	32.7	36.1
Unknown	34	*	*	23	*	*
Total	678,999	12.1	43.0	579,271	25.2	44.2
Education						
< High School	224,085	13.6	39.4	190,284	27.8	41.6
High School	274,794	11.8	44.6	234,722	24.3	45.3
> High School	133,914	10.1	45.7	115,430	23.1	45.4
Unknown	46,206	12.5	42.3	38,835	24.3	45.9
Total	678,999	12.1	43.0	579,271	25.2	44.2

Source: Centers for Disease Control and Prevention, Pediatric and Nutrition Surveillance System, <http://www.cdc.gov/pednss/index.htm> (accessed August 9, 2006).

gain during pregnancy than non-Hispanic white women. For African American women, only 40 percent had normal BMI before pregnancy and less than 30 percent had ideal weight gain during pregnancy. These racial-ethnic and sociodemographic disparities are summarized in Table 2.2.

II. BIOCHEMICAL ASSESSMENT

Nutritional status can be determined using biochemical testing. One biochemical indicator of possible nutritional deficiency is anemia.



ANEMIA

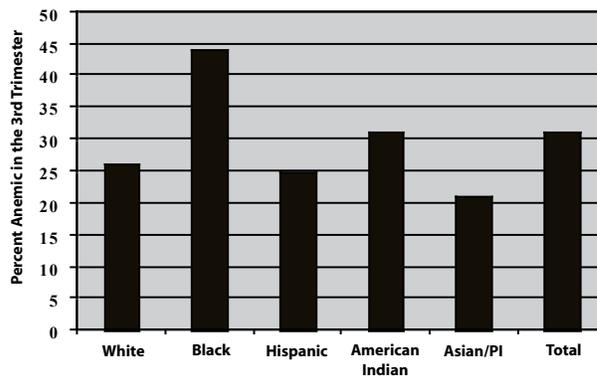
Maternal anemia is defined as less than the 5th percentile of the distribution of hemoglobin (Hb) or hematocrit (Hct) (12). The criteria for establishing anemia in pregnant and postpartum women are summarized in **Table 2.3**. Strong evidence exists for an association between maternal hemoglobin concentration and birthweight, as well as between maternal hemoglobin concentration and preterm birth (14). As discussed in Chapter 1, maternal anemia can also contribute to maternal and fetal/infant morbidities and mortality associated with obstetrical hemorrhage.

TABLE 2.3: CUT-OFF VALUES FOR DIAGNOSING ANEMIA IN PREGNANT WOMEN BY TRIMESTER OF PREGNANCY, AND IN POSTPARTUM WOMEN BY MATERNAL AGE, 2003		
Pregnancy Trimester	Hemoglobin	Hematocrit
First	11.0	33.0
Second	10.5	32.0
Third	11.0	33.0
Postpartum Age	Hemoglobin	Hematocrit
12 - < 15 yrs	11.8	35.7
15 - < 18 yrs	12.0	35.9
≥ 18 yrs	12.0	37.7

Source: Centers for Disease Control and Prevention, Pediatric and Nutrition Surveillance System, <http://www.cdc.gov/pednss/index.htm> (accessed August 9, 2006).

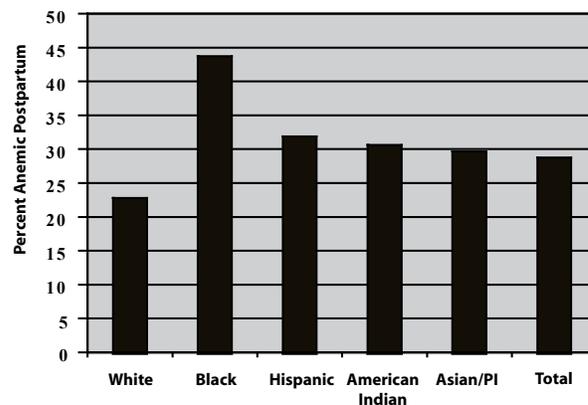
According to data from the 2003 CDC Pregnancy Nutrition Surveillance System, approximately one of every three (30.3 percent) low-income U.S. women was anemic in the third trimester of pregnancy, and one of every three (29.5 percent) was anemic during postpartum (12) (**Figures 2.5a and 2.5b**). There were significant racial-ethnic disparities in anemia. Whereas one of every five Asian and Pacific Islander women and one of every four non-Hispanic white and Hispanic women were anemic in the third trimester, 44 percent of non-Hispanic black women were anemic. From third trimester to postpartum, the prevalence of anemia increased substantially for Hispanic women—from 25 percent to 32 percent—and for Asian and Pacific Islander women—from 21 percent to 29 percent (12). The reason for this increase from third trimester to postpartum is unknown. As shown in **Table 2.4**, there were significant disparities in maternal anemia by age and education as well.

FIGURE 2.5A: ANEMIA IN THE 3RD TRIMESTER OF PREGNANCY, BY MATERNAL RACE & ETHNICITY, 2003



Source: Centers for Disease Control and Prevention, Pediatric and Nutrition Surveillance System, <http://www.cdc.gov/pednss/index.htm> (accessed August 9, 2006).

FIGURE 2.5B: ANEMIA DURING POSTPARTUM, BY MATERNAL RACE & ETHNICITY, 2003



Source: Centers for Disease Control and Prevention, Pediatric and Nutrition Surveillance System, <http://www.cdc.gov/pednss/index.htm> (accessed August 9, 2006).

TABLE 2.4: ANEMIA DURING POSTPARTUM, BY MATERNAL AGE AND EDUCATION, 2003		
Maternal Age	Anemia, 3rd Trimester	Anemia Postpartum
< 15 Years	35.4%	34.1%
15 – 17 Years	34.7%	35.1%
18 – 19 Years	32.5%	33.2%
20 – 29 Years	30.0%	28.7%
30 – 39 Years	28.0%	27.7%
> = 40 Years	26.8%	28.9%
Maternal Education		
< High School	32.9%	31.3%
High School	30.3%	28.9%
> High School	26.2%	24.4%

Source: Centers for Disease Control and Prevention, Pediatric and Nutrition Surveillance System, <http://www.cdc.gov/pednss/index.htm> (accessed August 9, 2006).



III. CLINICAL ASSESSMENT

A comprehensive review of clinical problems that could affect nutritional status during pregnancy is beyond the scope of this report. We highlight one common clinical problem that has an important impact on maternal nutrition and infant health: diabetes during pregnancy.

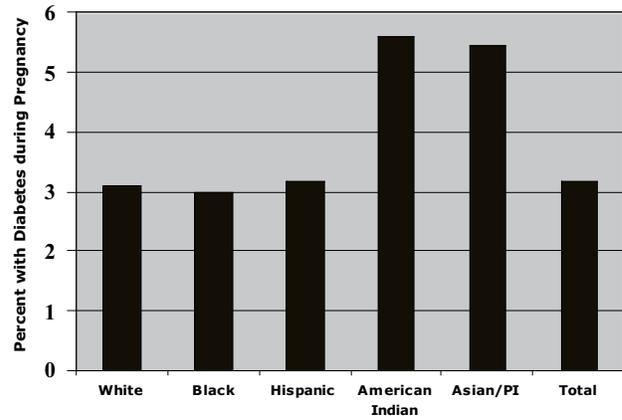
DIABETES DURING PREGNANCY

Diabetes complicating pregnancy is the second most frequently reported medical risk factor during pregnancy. Diabetes can pre-exist before pregnancy (pregestational diabetes) and is worsened by pregnancy, or its onset can occur during pregnancy (gestational diabetes) as a result of hormonally-induced (particularly human placental lactogen) relative insulin resistance. Pregestational diabetes has greater relevance to this report because it can cause fetal death and congenital anomalies, especially if the diabetes is poorly controlled (15). Preconceptional glycemic control among women with pregestational diabetes has been shown to reduce the risk of congenital anomalies (16). Gestational diabetes can increase the risk of fetal macrosomia, birth trauma, newborn hypoglycemia, and hyperbilirubinemia (15). Evidence from animal models suggests that gestational diabetes, especially if poorly controlled, may also program insulin and leptin resistance in the fetus, leading to greater susceptibility to diabetes and obesity in the offspring (17). From a maternal health perspective, long-standing and poorly controlled pregestational diabetes can cause microvascular, neuropathic, and cardiovascular disease, and gestational diabetes has been associated with increased future risk for Type II diabetes in the mother.

In 2002, more than 130,000 U.S. women reported having diabetes during pregnancy (18). The prevalence of diabetes during pregnancy was 3.3 percent. The prevalence, however, ranged from three percent for non-Hispanic black mothers to 5.4 percent for Asian and Pacific Islander mothers and 5.7 percent for Native American mothers, as shown in **Figure 2.6**.

These numbers, however, mask a great deal of intra-group variations. For example, the prevalence of diabetes during pregnancy among Asian and Pacific Islander mothers ranged from 4.3 percent for Japanese women to 6.0 percent for Filipino women. Similarly, among Latina mothers, prevalence ranged from 2.7 percent for Cubans to 4.2 percent for Puerto Rican women. Prevalence by Asian and Pacific Islander and Latina sub-groups is shown in **Table 2.5** (18).

FIGURE 2.6: DIABETES DURING PREGNANCY, BY MATERNAL RACE & ETHNICITY, 2002



Source: J. A. Martin, B. E. Hamilton, P. D. Sutton, S. J. Ventura, F. Menacker, M. L. Munson, "Births: final data for 2002," *Natl Vital Stat Rep.* 52, no. 10 (2003):1-114.

TABLE 2.5: DIABETES DURING PREGNANCY, BY API AND LATINA SUBGROUPS, 2002

Asian and Pacific Islander Mothers	Percent with Diabetes during Pregnancy
Chinese	4.79%
Japanese	4.25%
Hawaiian	4.92%
Filipino	5.98%
Other	5.51%
Latina Mothers	
Mexican	3.18%
Puerto Rican	4.21%
Cuban	2.73%
Central and South American	3.13%
Other	3.64%

Source: J. A. Martin, B. E. Hamilton, P. D. Sutton, S. J. Ventura, F. Menacker, M. L. Munson, "Births: final data for 2002," *Natl Vital Stat Rep.* 52, no. 10 (2003):1-114.

Dietary control of blood sugar is a critical determinant of pregnancy outcomes for women with diabetes during pregnancy. For women with pregestational diabetes, periconceptional control is key (16). No population-based data are available regarding glycemic control of U.S. women with diabetes during pregnancy. One small study of 47 pregnant women with pregestational diabetes found that 73 percent reported that their pregnancy was unintended at the time of conception, and 89 percent did not have adequate glucose control in early pregnancy, despite the fact that 80 percent had received medical care within six months of conception (19). If this study is representative of the larger U.S. population, clearly more can be done to improve dietary control of blood sugar before and during pregnancy for U.S. women with diabetes.



IV. DIETARY ASSESSMENT

This section evaluates the content and pattern of food intake by U.S. pregnant women.

A. CONTENT OF FOOD INTAKE: WHAT ARE U.S. PREGNANT WOMEN EATING?

What are pregnant women eating? Surprisingly little data on the foods that U.S. women consume during pregnancy are available because of the small numbers of pregnant women who are included in national nutritional surveys and the limited amount of dietary information that is collected in most large epidemiological studies (20). Furthermore, data on racial and socioeconomic differences in food intake by pregnant women are lacking. Such food intake data could have important clinical and public health applications for improving the nutritional status of pregnant women in the U.S. and for addressing disparities in maternal and infant health outcomes.

Important Foods

Important foods are those that contribute most to a population's nutrient intake (20); they are influenced both by nutrient density and the frequency of consumption. Unlike other studies that present dietary data as a percent of recommended level for one or more nutrients, data on important foods detail the foods that contribute most to nutrient intake. For example, 50 percent of energy intake may be derived from carbohydrates; however, important sources of carbohydrates may be complex carbohydrates like whole grains and vegetables or may be simple sugars like cookies, cake, and candy (20).

We found only one population-based study of important foods in diets of pregnant women, based on a prospective study of 2,505 pregnant women enrolled in the Pregnancy, Infection, and Nutrition (PIN) Study in North Carolina (20). Ninety percent of the women completed the food frequency questionnaire in the second trimester, and the contribution of each food item to the population's intake was calculated. Most women (53.5 percent) in the study sample were low-income (less than 185 percent of the federal poverty level), and over 90 percent were white and black women residing in North Carolina (50.3 percent and 43.2 percent, respectively). Thus, generalizability of the findings to the rest of pregnancy, and to the larger U.S. population, is somewhat limited.

Table 2.6 shows the five food sources that contributed most to the intake of energy and macronutrients among pregnant women

TABLE 2.6: TOP FIVE FOOD SOURCES THAT CONTRIBUTE MOST TO THE INTAKE OF ENERGY AND MACRONUTRIENTS AMONG WOMEN IN THE PIN STUDY, TOTAL SAMPLE AND BY RACE/ETHNICITY

Food	Overall sample (N=2247)	Non-Hispanic black women (N=971)	Non-Hispanic white women (N=1131)
Energy (%)			
Biscuits, muffins	6.1	6.2	6.3
French fries/fried potatoes	5.1	5.9	4.5
Whole milk	5.0	5.1	5.1
White bread, bagels, crackers	4.4	4.4	4.3
Soft drinks	3.9	3.6	4.2
Protein (%)			
Whole milk	7.5	7.6	7.4
Hamburger, beef burrito, meatloaf	7.5	7.7	7.3
Cheese and cheese spread	4.9	4.5	5.5
Beef, steak, roasts	4.2	4.4	3.9
Fried chicken	4.1	5.7	2.2
Carbohydrates (%)			
Soft drinks	7.6	7.1	8.1
Other fruit juices	7.3	8.2	6.2
Biscuits, muffins	6.4	6.6	6.7
White bread, bagels, crackers	5.8	6.0	5.8
French fries, fried potatoes	5.1	5.9	4.5
Fat (%)			
Mayonnaise, salad dressings	7.7	7.9	8.2
Whole milk	7.3	7.2	7.4
French fries, fried potatoes	6.7	7.6	6.0
Biscuits, muffins	6.7	6.6	7.0
Cheese, cheese spread	5.7	5.0	6.5

Source: A. M. Siega-Riz, L. M. Bodnar, and D. A. Savitz, "What are pregnant women eating? Nutrient and food group differences by race," *Am J Obstet Gynecol.* 186, no. 3 (March 2002): 480-6.

in the PIN study. The top 20 foods can be viewed at <http://www.cpc.unc.edu/projects/pin/publications/index.html> (21). Overall, biscuits and muffins, whole milk, and French fries and fried potatoes were among the top 10 sources for energy, carbohydrates, and fat, which illustrates their frequent consumption in this population. For carbohydrates, soft drinks and other fruit juices were the leading sources, accounting for nearly one-sixth (15.3 percent) of total carbohydrate consumption. Carbohydrate consumption in this population's diet was mostly refined and included such foods as soft drinks, biscuits and muffins, and white bread. For fats, mayonnaise, salad dressings, and French fries and fried potatoes were among the leading sources, and much of the fat that was consumed was saturated fat, as illustrated by the frequent consumption of high-fat animal products. Diets high in



TABLE 2.7: TOP FIVE FOOD SOURCES THAT CONTRIBUTE MOST TO THE INTAKE OF IRON, FOLATE, VITAMIN C, AND DIETARY FIBER AMONG WOMEN IN THE PIN STUDY, TOTAL SAMPLE AND BY RACE/ETHNICITY

Food	Overall Sample	Non-Hispanic black women	Non-Hispanic white women
Iron (%)			
Dry cereal, excluding high fiber and fortified	9.0	9.7	9.0
Highly fortified cereals	7.6	5.8	8.5
White bread, bagels, crackers	6.8	7.1	6.7
Biscuits, muffins	6.4	6.7	6.6
Bran, granola cereal	4.8	3.4	6.5
Folate (%)			
White bread, bagels, crackers	7.6	7.8	7.4
Dry cereal, excluding high fiber and fortified	7.4	8.0	7.0
Highly fortified cereals	6.2	4.7	7.4
Orange juice, grapefruit juice	5.7	5.9	5.4
Rice	5.4	5.9	3.9
Vitamin C (%)			
Orange juice, grapefruit juice	25.1	23.7	27.0
Other fruit juices	13.5	14.2	12.8
Oranges, tangerines	12.6	14.7	9.4
Fortified fruit drinks	5.9	7.4	4.1
Broccoli	4.8	4.7	5.2
Dietary fiber (%)			
French fries, fried potatoes	6.8	7.9	6.1
Beans (dried type)	6.5	5.9	7.0
Oranges, tangerines	5.5	6.9	3.7
Apples, pears	5.2	4.9	5.2
Bananas	6.7	4.5	5.0

Source: A. M. Siega-Riz, L. M. Bodnar, and D. A. Savitz, "What are pregnant women eating? Nutrient and food group differences by race," Am J Obstet Gynecol. 186, no.3 (March 2002): 480-6.

fat and empty calories (e.g., soft drinks) may be of concern for pregnant women, given not only their tendency to gain excessive weight during pregnancy but also the problem of postpartum weight retention. Furthermore, diets high in refined carbohydrates with high glycemic index can cause fetal hyperinsulinemia and result in programmed insulin and leptin resistance in the fetus, leading to greater susceptibility to early-onset diabetes and obesity in later life (17). Diets high in saturated and trans fat can promote inflammation and potentially contribute to inflammatory-mediated pregnancy complications, including preeclampsia, fetal growth restriction, and preterm birth.

Table 2.7 shows the top five food sources that contributed most to intake of iron, folate, vitamin C, and dietary fibers among pregnant women in the PIN study (20). Overall, fortified foods contributed substantially to micronutrient intake. For iron, the

top five leading sources consisted of fortified grain products (such as dry cereal, white bread, biscuits, and muffins), accounting for more than one-third of total iron intake. Interestingly, juice (orange/grapefruit) ranked as the sixth-highest contributor to iron intake (data not shown), even though these juices are not rich sources of iron, which illustrates their frequent consumption in this population. Ground beef ranked number seven and is the preferred meat in this population. For folate, ready-to-eat cereals made up one-sixth (16.0 percent) of total folate intake; other folate-fortified grains (e.g., white bread, bagels, crackers, rice, biscuits, and muffins) were also important sources of folate. Orange and grapefruit juice and dried beans were among the top 10 sources of this micronutrient. For Vitamin C, more than half of the population's intake was contributed by three foods: orange and grapefruit juice, other fruit juices, and oranges and tangerines.

Table 2.8 (next page) displays the mean amount (distribution not shown) of nutrients consumed during the second trimester of pregnancy among women in the PIN Study, compared to recommended daily allowances and dietary reference intakes. Overall, the study population consumed higher total energy, protein, and vitamin C than recommended. They also consumed a high-fat diet; more than one-half of the women consumed diets that included greater than 30 percent of kilocalories from fat. The median intake of iron was approximately 76 percent of the recommended level; only 29.7 percent of women met the recommendation through dietary sources alone. Expressed in Dietary Folate Equivalents (DFEs), 59.9 percent of women consumed the recommended amount of folate. Intake of dietary fiber was also below the minimum recommendation for more than half of the total study population.

When a comparison of important foods was made by race-ethnicity, there were no important differences for energy, carbohydrates, and fats (20). For protein, fried chicken (5.7 percent vs. 2.2 percent) and eggs (4.6 percent vs. 2.8 percent) contributed more to the intakes among black women compared to white women. Rice, oranges and tangerines, French fries and fried potatoes, and collards, kale, and greens were major contributors to folate and iron in the diets of black women, whereas highly fortified and bran and granola cereals, spaghetti, lasagna, pasta, and dried beans contributed more iron and folate for white women. Although black women consumed significantly higher total energy and higher absolute values of every macro- and micronutrient, white women consumed more protein, iron, folate, and fiber and had lower fat intakes after energy adjustment. In general, white women were consuming a higher nutrient-dense diet, and black women were consuming more calories but fewer nutrients after



MATERNAL NUTRITION AND INFANT MORTALITY IN THE CONTEXT OF RELATIONALITY

TABLE 2.8: MEAN AMOUNT OF NUTRIENTS CONSUMED DURING THE SECOND TRIMESTER OF PREGNANCY AMONG WOMEN IN THE PIN STUDY, TOTAL SAMPLE AND BY RACE/ETHNICITY

Nutrient	Recommendation	Population	Mean (SD)
Energy (kcal)	2500 kcal	Total	2830 (1488)
		Non-Hispanic white women	2530 (1192)
		Non-Hispanic black women	3220 (1727)*
Protein (g)	60 g	Total	103 (54.0)
		Non-Hispanic white women	94.0 (42.1)†
		Non-Hispanic black women	115 (64.1)
Carbohydrate (g)	N/A	Total	372 (197)
		Non-Hispanic white women	332 (157)
		Non-Hispanic black women	419 (231)
Fat (% kcal)	30% total kcal	Total	33.5 (6.4)
		Non-Hispanic white women	33.2 (6.1)
		Non-Hispanic black women	34.4(6.5)†
Iron (mg)	22 mg (≤18y) 23 mg (19-50y)	Total	20.1 (10.8)
		Non-Hispanic white women	18.2 (8.8) †
		Non-Hispanic black women	22.3 (12.5)
Folate (mg, expressed as DFE)‡	520 mg DFE ¹²	Total	668 (366)
		Non-Hispanic white women	604 (284) †
		Non-Hispanic black women	736 (432)
Vitamin C (mg)	66 mg (≤18y) 70 mg (19-50y)	Total	230 (182)
		Non-Hispanic white women	185 (125)
		Non-Hispanic black women	281 (223)†
Fiber (g)	20-30 g	Total	24.1 (14.2)
		Non-Hispanic white women	21.7 (10.7)†
		Non-Hispanic black women	26.6 (17.0)

* $P < .01$, significantly greater than non-Hispanic white women.

† $P < .01$, significantly higher nutrient intake after energy adjustment compared with the other ethnic group.

‡ To estimate DFE, we multiplied the folate intake of each individual by the percent of folate contributed by fortified foods (33%). This proportion of intake was multiplied by 1.7 to account for increased bioavailability of synthetic folic acid in fortified foods. Finally, this value was added to the remaining micrograms of folate from natural sources. Without this calculation, the mean folate intake and SD in the total sample and for white and black women was $543 \pm 298 \mu\text{g}$, $491 \pm 231 \mu\text{g}$, and $598 \pm 351 \mu\text{g}$, respectively.

Source: A. M. Siega-Riz, L. M. Bodnar, and D. A. Savitz, "What are pregnant women eating? Nutrient and food group differences by race," *Am J Obstet Gynecol*, 186, no. 3 (March 2002): 480-6.

energy adjustment. These data suggest that black women need to increase their intake of nutrient-dense (particularly micronutrient-dense) foods (20).

Specific Macronutrients and Micronutrients

Another approach to assessing the content of food intake by U.S. women is to examine the adequacy of specific macro- or micronutrients in their diets.

1. Energy

The estimated energy requirement (EER) for pregnant women is calculated based on the EER for nonpregnant women, plus additional energy expended during pregnancy as well as energy deposition. The sum of additional energy expended and energy deposition is deemed negligible in the first trimester, 340 kcal in the second trimester, and 452 kcal in the third trimester (22). These estimates agree with the recent estimates by Butte et al.—the incremental needs during pregnancy were negligible in the first trimester, 350 kcal/d in the second trimester, and 500 kcal/d in the third trimester over nonpregnant values for women in the normal BMI group (23). Using these numbers, we estimate that the energy requirement for an active pregnant woman of average height (162 cm) ranged from 1,950 kcal/d to 2,100 kcal/d (depending on her prepregnancy BMI) in the first trimester, 2,300 to 2,450 kcal/d in the second trimester, and 2,450 to 2,600 kcal in the third trimester.

According to the Continuing Survey of Food Intake by Individuals (CSFII), pregnant women in the U.S. consumed, on average, 1,986 kcals (standard error 153) daily (22). This number is lower than the average daily intake of about 2,100 kcals from several studies (24), and substantially lower than the calculated energy intakes of about 2,350 kcals by 12 nutritionists who weighed and recorded all food intakes for three different weeks during their pregnancies (24). Under-reporting notwithstanding, about half of the pregnant women in CSFII did not meet the estimated energy requirement of pregnancy (22). There is no evidence that energy intake varies with the stage of pregnancy. Generally, intake increases during the first trimester and remains relatively constant thereafter, with a possible decline near term (22). Unfortunately, available published data do not allow us to examine racial-ethnic differences in energy intake during pregnancy.



2. *Macronutrients*

The need for nitrogen and essential amino acids is met by dietary protein. The recommended daily allowance for protein is estimated to be 71 grams per day for a healthy pregnant woman (22). Available data show that U.S. pregnant women generally consume more protein than recommended. The average intake of protein by U.S. pregnant women in CSFII was 78.2 g/d (22) (Table 2.9). However, protein intake varied substantially; about 25 percent of women consumed less than 70 g/d, and 25 percent of women consumed more than 88 g/d. Approximately 16 percent of total energy intake came from protein (22).

There is no recommended daily allowance for fat intake. According to the CSFII, pregnant women, on average, consumed about 75 grams of fat a day (22) (Table 2.9). Fat intake accounted for greater than 30 percent of total energy intake for more than half of pregnant women in CSFII (22). Moreover, more than a third of the total fat intake, on average, consisted of saturated fatty acids (22). With respect to polyunsaturated fatty acids, although pregnant women, on average, are consuming α -linolenic acid (an important constituent of omega-3 polyunsaturated fatty acids, or PUFAs) in amounts that are considered adequate intake, about 25 percent of pregnant women did not consume an adequate amount (22). The average intake of docosahexaenoic fatty acid (DHA), another important component of omega-3 PUFAs, by pregnant women in CSFII was 51 mgs per day, far less than the 300 mg/d recommended for pregnant and lactating women by the International Society for the Study of Fatty Acids and Lipids (25). Omega-3 PUFAs can modulate inflammation by acting on the 2-series prostaglandins, whereas omega-6 PUFAs can promote inflammation (26). Consumption of omega-3 and omega-6 PUFAs (especially linoleic to α -linolenic acid) needs to be in proportionate balance; a ratio of omega-6 to omega-3 of 1:1 to 5:1 has been proposed to maintain good cardiovascular health. According to the National Health and Nutrition Examination Survey (NHANES) 1999-2000, that ratio is about 10:1 for U.S. women in their reproductive years (27) (i.e., skewed toward a more pro-inflammatory profile), and among pregnant women in CSFII, the ratio was nearly 10:1 (22).

Consumption of carbohydrates by pregnant women in CSFII far exceeded the recommended daily allowance (22) (Table 2.9). The average intake was about 100 g/d more than the recommended amount of 175 g/d, an allowance that was exceeded by greater than 95 percent of pregnant women in CSFII (22). On average, more than half (53 percent) of total energy intake was derived from carbohydrates. Published reports of CSFII did not break down carbohydrate consumption by types of carbohydrates, but

TABLE 2.9: RECOMMENDED AND MEAN DAILY INTAKE OF MACRONUTRIENTS AMONG PREGNANT WOMEN IN CSFII

Macronutrients	Recommended Daily Intake	Mean Daily Intake (SD)
Protein	71 g/d	78.2 (2.6) g/d
% of total energy	10-35%	15.6 (0.6) %
Total Fat	ND	75.5 (5.2) g/d
% of total energy	20-35%	32.9 (0.9) %
Saturated fatty acids	ND	27.6 (1.4) g/d
Linoleic acid	13 g/d	13.9 (1.1) g/d
α-linolenic acid	1.1 g/d	1.49 (0.11) g/d
Docosahexaenoic acid	300 mg/d*	51 (14) mg/d
Carbohydrate	175 g/d	277 (12) g/d
% of total energy	45-65%	53.0 (1.3) %
Total Fiber	28 g/d	16.2 (1.0) g/d

Source: Institute of Medicine, "Dietary reference intakes for energy, carbohydrate, fiber, fat, fatty acids, cholesterol, protein, and amino acids" (Washington DC: National Academies Press, 2005).

the aforementioned PIN study and other studies suggest a high intake of refined carbohydrates and added sugar (20). According to NHANES III, pregnant women, on average, consumed 21 teaspoons of added sugar (defined as sugars and syrups that are added to foods during processing or preparation, such as high-fructose corn syrup) daily, and 10 percent of pregnant women consumed more than 33 teaspoons daily, although no upper limits on added sugar have been recommended (22).

3. *Elements*

Table 2.10 (next page) displays the mean daily intake of selected elements from food sources by pregnant women in NHANES III and CSFII (28-31). The mean daily intake of calcium, copper, manganese, phosphorus, selenium, and zinc (in the NHANES III sample only) exceeded the recommended daily allowance; however, a substantial proportion of pregnant women still did not meet the recommended daily allowance for several of these elements (25 percent for calcium, 10-25 percent for copper, 5 percent for manganese, and 50 percent for zinc). The mean daily intake of iron, magnesium, and molybdenum from food sources fell far short of the RDA for these elements.

Taking supplements generally improved the average daily intake, but a substantial proportion of pregnant women still did not meet the RDAs for these elements. For example, mean daily intake of iron was 15.3 mg without supplement and 49.0 mg with supplements; however, the proportion of women who consumed less than the RDA fell from over 95 percent to 50 percent. This is significant considering that half of pregnant women in these surveys did not meet the RDA for iron even when multivitamin



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TABLE 2.10: RECOMMENDED AND MEAN DAILY INTAKE OF ELEMENTS BY PREGNANT WOMEN AND PERCENTAGE OF WOMEN WHOSE AVERAGE INTAKE DID NOT MEET RECOMMENDED DAILY ALLOWANCE OR ADEQUATE INTAKE FOR A PARTICULAR ELEMENT, NHANES III AND CSFII

Elements	Recommended Daily Intake	Mean Daily Intake (SD) NHANES III	% < RDA/AI NHANES III	Mean Daily Intake (SD) CSFII	% < RDA/AI CSFII	AMDR
Calcium	1,000 mg/d	NA	NA	1,168 (90.4) mg/d	25%	2,500 mg/d
Copper	1,000 µg/d*	1,280 (50) µg/d	10%	1,170 (60) µg/d	25%	10,000 µg/d*
Iodine	220 µg/d	158 (4) µg/d	95%	NA	NA	1,100 µg/d*
Iron	27 mg/d	15.3 (0.75) mg/d	95%	14.3 (4.3) mg/d	>99%	45 mg/d
Magnesium	350 mg/d*	NA	NA	292.5 (23.4) mg/d	75%	350 mg/d
Manganese	2.0 mg/d	4.72 (0.17) mg/d	5%	NA	NA	11 mg/d*
Molybdenum	50 mg/d	23.8 (0.8) mg/d	99%	NA	NA	2,000 mg/d*
Phosphorus	700 mg/d*	NA	NA	1,572 (292.6) mg/d	<1%	3,500 mg/d*
Selenium	60 µg/d	115.9 (6.4) µg/d	<1%	NA	NA	400 µg/d
Zinc	11 mg/d	11.2 (0.5) mg/d	50%	10.4 (2.0) mg/d	50%	11 mg/d

* From supplements only.

Sources: Institute of Medicine, "Dietary reference intakes for Vitamin A, Vitamin K, Arsenic, Boron, Chromium, Copper, Iodine, Iron, Manganese, Molybdenum, Nickel, Silicon, Vanadium, and Zinc" (Washington DC: National Academies Press, 2000); Institute of Medicine, "Dietary reference intakes Vitamin C, Vitamin E, Selenium, and Carotenoids" (Washington DC: National Academies Press, 2000); Institute of Medicine, "Dietary reference intakes for Thiamin, Riboflavin, Niacin, Vitamin B6, Folate, Vitamin B12, Pantothenic Acid, Biotin, and Choline" (Washington DC: National Academies Press, 1998).

TABLE 2.11: RECOMMENDED AND MEAN DAILY INTAKE OF VITAMINS BY PREGNANT WOMEN AND PERCENTAGE OF WOMEN WHOSE AVERAGE INTAKE DID NOT MEET RECOMMENDED DAILY ALLOWANCE OR ADEQUATE INTAKE FOR A PARTICULAR VITAMIN, NHANES III AND CSFII

Vitamins	Recommended Daily Intake	Mean Daily Intake (SD) NHANES III	% < RDA/AI NHANES III	Mean Daily Intake (SD) CSFII	% < RDA/AI CSFII	AMDR
Vitamin A	770 µg/d*	757 (147) µgRAE/d	50%	NA	NA	3,000 µg/d*
Vitamin B1 (Thiamin)	1.4 mg/d	1.8 (0.05) mg/d	25%	1.5 (0.39) mg/d	25%	ND
Vitamin B2 (Riboflavin)	1.4 mg/d	2.2 (0.08) mg/d	15%	1.8 (0.37) mg/d	25%	ND
Vitamin B3 (Niacin)	18 mg/d	23.5 (0.71) mg/d	15%	19.5 (6.97) mg/d	25%	35 mg/d*
Vitamin B4 (Choline)	450 mg/d	NA	NA	NA	NA	3,500 mg/d
Vitamin B5 (Panththenic Acid)	6 mg/d	NA	NA	NA	NA	ND
Vitamin B6 (Pyridoxine)	1.9 mg/d	1.9 (0.07) mg/d	50%	1.61 (0.42) mg/d	50%	100 mg/d*
Vitamin B7 (Biotin)	450 mg/d	NA	NA	NA	NA	3,500 mg/d*
Vitamin B9 (Folate)	600 µg/d	288 (9.2) µg/d	>95%	241 (29) µg/d	>99%	1,000 µg/d
Vitamin B12 (Cobalamine)	2.6 µg/d	5.3 (0.18) µg/d	<5%	3.8 (1.45) µg/d	10%	ND
Vitamin C	85 mg/d	124.0 (11.3) mg/d	10%	132.9 (13.7) mg/d	25%	2,000 mg/d*
Vitamin D (Calciferol)	5 µg/d	NA	NA	NA	NA	50 µg/d
Vitamin E (α-Tocopherol)	15 mg/d	10.1 (1.2) mg/d	>95%	7.8 (0.7) mg/d	>99%	1,000 mg/d
Vitamin K	90 µg/d	87.8 (12.5) µg/d	50%	46.4 (4.7) µg/d	90%	ND

Sources: Institute of Medicine, "Dietary reference intakes for Vitamin A, Vitamin K, Arsenic, Boron, Chromium, Copper, Iodine, Iron, Manganese, Molybdenum, Nickel, Silicon, Vanadium, and Zinc" (Washington DC: National Academies Press, 2000); Institute of Medicine, "Dietary reference intakes Vitamin C, Vitamin E, Selenium, and Carotenoids" (Washington DC: National Academies Press, 2000); Institute of Medicine, "Dietary reference intakes for Thiamin, Riboflavin, Niacin, Vitamin B6, Folate, Vitamin B12, Pantothenic Acid, Biotin, and Choline" (Washington DC: National Academies Press, 1998).



TABLE 2.12: MEAN DAILY INTAKE OF SELECTED ELEMENTS AND VITAMINS BY PREGNANT WOMEN AND PERCENTAGE OF WOMEN WHOSE AVERAGE INTAKE DID NOT MEET RECOMMENDED DAILY ALLOWANCE, WITH AND WITHOUT SUPPLEMENTS, NHANES III

Vitamins	Mean Daily Intake (SD) NHANES III Without Supplements	% < RDA/AI Without Supplements	Mean Daily Intake (SD) NHANES III With Supplements	% < RDA/AI With Supplements
Copper	1,280 (50) µg/d	10%	1,860 µg/d	5%
Iron	15.3 (0.75) mg/d	95%	49.0 mg/d	50%
Selenium	115.9 (6.4) µg/d	<1%	123.9 µg/d	1%
Zinc	11.2 (0.5) mg/d	50%	20.0 mg/d	25%
Vitamin A	757 (147) µgRAE/d	50%	1,106 (36) µgRAE/d	5%
Vitamin B1 (Thiamin)	1.8 (0.05) mg/d	25%	5.6 (1.38) mg/d	25%
Vitamin B2 (Riboflavin)	2.2 (0.08) mg/d	15%	6.2 (1.38) mg/d	25%
Vitamin B3 (Niacin)	23.5 (0.71) mg/d	15%	38.7 (2.13) mg/d	25%
Vitamin B6 (Pyridoxine)	1.9 (0.07) mg/d	50%	8.77 (1.45) mg/d	50%
Vitamin B9 (Folate)	288 (9.2) µg/d	95%	858 (40.9) µg/d	25%
Vitamin B12 (Cobalamine)	5.3 (0.18) µg/d	<5%	13.2 (1.23) µg/d	<5%
Vitamin C	124.0 (11.3) mg/d	10%	192.7 mg/d	5%
Vitamin E (α-Tocopherol)	10.1 (1.2) mg/d	95%	31.6 mg/d	25%
Vitamin K	87.8 (12.5) µg/d	50%	87.4 µg/d	50%

Sources: Institute of Medicine, "Dietary reference intakes for Vitamin A, Vitamin K, Arsenic, Boron, Chromium, Copper, Iodine, Iron, Manganese, Molybdenum, Nickel, Silicon, Vanadium, and Zinc" (Washington DC: National Academies Press, 2000); Institute of Medicine, "Dietary reference intakes Vitamin C, Vitamin E, Selenium, and Carotenoids" (Washington DC: National Academies Press, 2000); Institute of Medicine, "Dietary reference intakes for Thiamin, Riboflavin, Niacin, Vitamin B6, Folate, Vitamin B12, Pantothenic Acid, Biotin, and Choline" (Washington DC: National Academies Press, 1998).

and/or iron supplementation were accounted for, suggesting perhaps low intake or poor compliance with supplementation. Similarly, without supplement, more than half of pregnant women consumed less than the RDA for zinc; with supplement, about 25 percent still failed to meet the RDA.

4. Vitamins

Table 2.11 displays the mean daily intake of vitamins from food sources by pregnant women in NHANES III and CSFII (28-31). Mean daily intake met or nearly met the RDAs for all vitamins other than folate and vitamin E. Despite this, a substantial proportion of women still consumed less than the recommended daily amount (50 percent for vitamin A, 25 percent for vitamin B1, 15-25 percent for B2, 15-25 percent for B3, 50 percent for B6, about 5-10 percent for B12, 10-25 percent for vitamin C, and 50-90 percent for vitamin K). Note the large discrepancy between NHANES III and CSFII in terms of mean daily intake of vitamin K.

For folate, pregnant women, on average, consumed less than half of the recommended amount (30); for vitamin E, only about half to two-thirds of the RDA (29). These deficiencies may reflect low consumption of fruits and vegetables. Supplements appeared to ameliorate the deficiencies, but a sizable proportion of women still had inadequate intake. Mean daily intake of folate improved from 288 to 858 µg/d with supplementation, but 25 percent of

pregnant women still had inadequate intake. Similarly, mean daily intake of vitamin A improved from 10.1 to 31.6 mg/d with supplementation, but one in four women still had inadequate intake (**Table 2.12**).

Racial and Ethnic Differences in Dietary Intake

Racial differences are often seen in nutrition, but surprisingly few population-based studies have documented racial-ethnic differences in dietary intake among pregnant women. The aforementioned PIN study found that non-Hispanic white women were consuming a higher nutrient-dense diet, and non-Hispanic black women were consuming more calories but fewer nutrients after energy adjustment (20). In the Calcium for Preeclampsia Prevention Trial (32), a multicenter, prospective study of 4,589 healthy nulliparous women who were enrolled from 1992 to 1995, Cohen et al. found substantial variations in dietary intake by race-ethnicity. Spanish-speaking Hispanic women had lower daily intake of total energy, carbohydrate, fat, and sodium, and higher intake of iron, compared to other groups in general and to English-speaking Hispanic women in particular. Compared to non-Hispanic white women, non-Hispanic black women had lower daily intake of carbohydrate but higher daily intake of protein and fat, and substantially lower intake of calcium, vitamin E, and folate. **Table 2.11** displays the percent of pregnant women, by ethnic group, with intakes greater than or equal to the RDA for a particular nutrient. Overall, a sizable portion of pregnant



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women were deficient in their intakes of calcium (without supplementation), magnesium, zinc, and vitamins A and E. For African American women in particular, only about one in four pregnant women met the RDA for calcium, magnesium, zinc, and vitamin E, and about one in three did not meet the RDA for iron and folate (Table 2.13).

With respect to calcium intake, Harville et al. (33) recently conducted a study of calcium intake during pregnancy among a cohort of 385 black and white women. Median total calcium intake was lower among black women (1,421 mg/d) than white women (1,556 mg/d). Black women were more likely to have whole milk as the largest source of calcium (54 percent vs. 46 percent) and less likely to have skim milk as their primary source (2 percent vs. 11 percent). Black women were also more likely to have soft cheese as their primary source (15 percent vs. 7 percent). Consumption of whole milk and soft cheese increases the risk of exposures to dioxins and listeria, respectively. For the secondary sources, black women were more likely to have soft cheeses (45 percent vs. 24 percent) and less likely to have hard cheeses (17

percent vs. 30 percent). Ten percent of black women and 6 percent of white women reported being lactose intolerant or allergic to milk; women who were lactose intolerant were proportionately more likely to report daily calcium intake of less than 600mg.

With respect to multivitamin-mineral supplements, Yu et al. (34) examined racial differences in supplement use among 9,953 mothers in the National Maternal and Infant Health Survey who delivered live infants in 1988. Sixty-seven percent of black mothers took supplements during pregnancy, compared with 84 percent of white mothers. Multivariate analysis revealed the following: black mothers; mothers who are less educated, younger, unmarried, and non-smokers; and mothers who participate in Women, Infants, and Children programs were at elevated risk for non-use. Vahratian et al. (35) also found in the PIN study that proportionately fewer black women took multivitamins before and during early pregnancy, and proportionately more black women were non-users before or during pregnancy, compared to white women. A more recent study (36) examined adherence to multivitamin supplements using pill-count among a clinic-based sample of low-income pregnant women. The study found that, overall, women took 74 percent of supplements as prescribed. Adherence was higher among non-Hispanic white women than among non-Hispanic black women (79 percent vs. 72 percent, $P \leq 0.01$). Interactions of ethnicity with age group, smoking status, and prior supplement use were significant. Multivariate regression analysis stratified by ethnicity revealed that, among the white women, education beyond high school, unmarried status, nulligravidity, and smoking were positively associated with adherence. In contrast, among the black women, supplement use three months prior to current pregnancy and no loss of appetite were positively associated with adherence.

Given the scarcity of data on racial-ethnic differences in dietary intakes among pregnant women, we also identified studies that have examined such differences in the general population. One of the best studies was conducted by Popkin et al. (37), who compared dietary trends among blacks and whites of varying socioeconomic status (SES) using data for 6,061 participants in the 1965 Nationwide Food Consumption Surveys, 16,425 in the 1977-1978 Nationwide Food Consumption Surveys, and 9,920 in the 1989-1991 Continuing Survey of Food Intake by Individuals (CSFII). They found an overall improvement in dietary quality over time; however, there were still notable excesses and deficiencies. All groups consumed more than 30 percent of total energy in fat, and no group came close to consuming five servings of fruits and vegetables daily. Moreover, significant racial-ethnic and socioeconomic disparities persisted. Low SES individuals still consumed more fat and saturated fat and less fruits and vegetables

TABLE 2.13: PERCENT OF WOMEN, BY ETHNIC GROUP, WITH INTAKES GREATER THAN OR EQUAL TO THE RDA, CALCIUM FOR PREECLAMPSIA PREVENTION TRIAL, 1992-1995

	White	Black	Hispanic/ English	Hispanic/ Spanish
Protein	80	79	77	74
Calcium	48	26*	39*	37**
Magnesium	33	19*	32***	38
Iron	75	68	87	86
Zinc	22	25	24	22
Vitamin A	57	40**	51*	51
Vitamin B6	80	73	89	89
Vitamin C	89	88*	95	95
Vitamin E	39	28*	37	38
Folacin	78	69	8	88
Niacin	97	96	99	95***
Riboflavin	92	83	95	95
Thiamine	88	85	94	90

Nutrient intake from 24-hour dietary recall included. When taken, supplements that included Calcium for Preeclampsia Prevention prenatal supplements (Mission Prenatal; Mission Pharmacal Co, San Antonio, Texas) containing 30 mg iron, 100 mg ascorbic acid, 5 mg thiamine, 3 mg pyridoxine, 2 mg riboflavin, 10 mg niacinamide, 1 mg d-calcium pantothenate, 2 mg vitamin B12, 0.4 mg folic acid, 4000 USP vitamin A, 400 USP vitamin D2, and 50 mg elemental calcium.

The RDA includes protein, 60g; calcium, 1200 mg; magnesium, 320 mg; iron, 30 mg; zinc, 15 mg; vitamin A, 800 mg reuol equivalents; vitamin B6, 2.2 mg; vitamin C, 70 mg; vitamin E, 10 mg; folacin, 400 mg; niacin, 17 mg; riboflavin, 1.6 mg; thiamine, 1.5 mg.

* $P < .001$; ** $P < .01$; *** $P < .05$, significance of differences from white ethnic group, adjusted for center.

Source: G. R. Cohen, L. B. Curet, R. J. Levine, M. G. Ewell, C. D. Morris, P. M. Catalano, D. Clokey, and M. A. Klebanoff, "Ethnicity, nutrition, and birth outcomes in nulliparous women," *AM J Obstet Gynecol* 185 (2001): 660-7.



and calcium than high SES individuals, and low SES blacks consumed the highest amount of fat and the least amount of fruits and vegetables (on average only 2.1 servings per day) and calcium (Table 2.14).

B. PATTERN OF FOOD INTAKE

We also examined the pattern of food intake among pregnant women in the U.S. We were particularly interested in the frequency of eating (meal/snack pattern) and fasting, food locations, and pica.

Frequency of Eating and Fasting

Frequency of eating or meal pattern during pregnancy is a component of maternal nutrition and may be related to pregnancy outcomes. The Institute of Medicine recommends that pregnant

women “eat small to moderate-sized meals at regular intervals, and eat nutritious snacks” consisting of three meals and two or more snacks per day (38). Siega Riz et al. (39) found that women in the aforementioned PIN study who consumed meals/snacks less frequently had a higher risk of delivering preterm (adjusted odds ratio = 1.30, 95 percent confidence interval: 0.96, 1.76). In this population, more than one in four (28 percent) pregnant women did not meet this recommendation. There were no racial differences in meal/snack patterns among pregnant women in this population.

Herrmann et al. (40) also examined the effects of prolonged periods without food intake during pregnancy on preterm delivery. In a prospective cohort study of 237 women enrolled in the Behavior in Pregnancy Study, prolonged periods without food lasting 13 hours or longer were associated with elevated maternal corticotropin-releasing hormone (CRH) concentrations

TABLE 2.14: TRENDS IN INTAKE OF DIETARY COMPONENTS ACCORDING TO RACE/ETHNICITY AND SES

Race and Component	Socioeconomic Status								
	LOW			MEDIUM			HIGH		
	1965	1977-1978	1989-1991	1965	1977-1978	1989-1991	1965	1977-1978	1989-1991
Whites									
No. of respondents	2146	3144	3195	2845	8619	3994	381	2716	1415
Energy from fat (%)	38.8±0.2	37.4±0.1	34.1±0.1	39.5±0.15	38.1±0.09	34.8±0.14	39.1±0.4	38.2±0.1	34.2±0.2
Energy from saturated fat (%)	14.2±0.1	13.4±0.1	11.9±0.1	14.7±0.07	13.7±0.04	12.2±0.06	14.4±0.2	13.8±0.1	11.6±0.1
Cholesterol (mg)	426±6.2	334±4.2	272±4.3	462±5.9	347±2.7	276±3.5	452±13.7	362±5.4	261±5.4
Fruits and vegetables (no. of servings)	2.8±0.04	3.0±0.04	2.7±0.04	3.5±0.04	3.4±0.02	3.0±0.03	4.2±0.11	3.9±0.09	3.5±0.07
Grains and legumes (no. of servings)	4.5±0.06	4.0±0.04	4.4±0.04	4.0±0.05	4.0±0.02	4.5±0.04	3.7±0.11	3.7±0.04	4.6±0.06
Protein (% of RDA)	147±1.6	130±1.1	125±1.2	165.7±1.5	145.1±0.72	138.8±1.0	170±4.0	149±1.3	138±1.5
Sodium (mg)	3519±42	3015±28	3137±30	3673±36.9	3249±17.7	3467±30.1	3601±94	3298±30	3399±46
Calcium (% of RDA)	82±1.1	74±0.8	80±0.9	85.9±1.0	80.4±0.57	87.9±0.94	86±2.7	88±1.0	93±1.5
Blacks									
No. of respondents	504	1065	948	179	763	328	6†	118	40
Energy from fat (%)	37.2±0.4	36.7±0.2	34.9±0.3	38.9±0.66	37.3±0.32	34.7±0.49	—	39.5±0.8	32.6±1.2
Energy from saturated fat (%)	12.8±0.2	12.5±0.1	11.7±0.1	13.9±0.29	12.7±0.14	12.1±0.22	—	13.8±0.3	10.1±0.4
Cholesterol (mg)	441±12.4	392±8.2	305±7.9	502±24.6	399±10.2	311±15.1	—	379±23	281±37
Fruits and vegetables (no. of servings)	2.3±0.09	2.6±0.07	2.1±0.06	3.1±0.18	3.1±0.09	2.9±0.14	—	2.8±0.18	3.6±0.44
Grains and legumes (no. of servings)	5.4±0.14	4.5±0.08	4.3±0.08	4.2±0.23	4.3±0.09	4.4±0.13	—	3.5±0.21	3.8±0.45
Protein (% of RDA)	153±3.5	142±2.2	132±2.3	178.8±6.7	144.2±2.5	142.3±4.3	—	145±6.0	132±9.9
Sodium (mg)	3643±84	3136±52	3086±60	3630±151.8	3110±55.3	3470±110.8	—	2981±172	3095±274
Calcium (% of RDA)	73±2.2	62±1.3	60±1.3	73.1±3.2	62.8±1.6	77.2±3.2	—	66±4.7	76±9.0

* The results were weighted to permit inferences applicable to the total noninstitutionalized U.S. population. Plus-minus values are means ± SE. RDA denotes Recommended Daily Allowance.

† The sample was too small to present meaningful results.

Source: B. M. Popkin, A. M. Siega-Riz, and P. S. Haines, “A comparison of dietary trends among racial and socioeconomic groups in the United States,” *N Engl J Med.* 335, no. 10 (Sep. 5, 1996): 716-20.



compared with prolonged periods without food lasting less than 13 hours at two time points during pregnancy, controlling for pregravid body mass index, energy intake, income, race, smoking, and maternal age (18-20 weeks: adjusted odds ratio, 2.5; 95%CI: 0.9-7.1; 28-30 weeks: adjusted odds ratio, 1.7; 95%CI: 0.7-4.2). There was an inverse, linear relationship between maternal corticotropin-releasing hormone concentrations and gestational age at delivery. Ninety percent of pregnant women in the study reported going 10 or more hours without food; nearly half (48 percent) of the women reported greater than or equal to 13 hours without food at 18 to 20 weeks' gestation. Proportionately more black women reported greater than or equal to 13 hours without food and had high levels of CRH compared to white women in the study. The study did not explore why these women went for a prolonged period without food intake. Delaying first meal of the day after an overnight fast or skipping breakfast altogether may be responsible for prolonged periods without food intake. Proportionately more women who reported greater than or equal to 13 hours without food also reported high stress levels compared to those who reported less than 13 hours without food; however, the difference did not reach statistical significance.

Food Locations

Food locations may influence the content of nutritional intake (41). The consumption of fast food was of particular interest, given the rapid growth of its popularity and its contribution to poor dietary quality. In 1997, fast food comprised 34 percent of total sales of food away from home, compared with only four percent in 1953 (42). Eating at fast-food restaurants (defined as food purchased in self-service or carry-out eating places without wait service) can contribute to obesity because fast-food meals are generally high in total fat, saturated fat, and total energy (43-47), thereby potentially contributing to energy imbalance. Furthermore, fast-food restaurant use can adversely affect diet quality; for example, studies have found that fast-food options tend to be low in calcium, folate, vitamins A and C, and dietary fiber (48-50). Using data from 1,120 women ages 19-50 who were surveyed over a one-year period as part of the 1985 Continuing Survey of Food Intake by Individuals, Haines and colleagues reported that women who ate frequently at fast-food establishments had the highest intakes of total energy, total fat, saturated fat, cholesterol, and sodium, and low nutrient densities for dietary fiber, calcium, vitamin C, and folate (48).

Unfortunately, we were unable to identify any published studies on food locations (i.e., where food is consumed) for pregnant women; thus, we searched for such studies in non-pregnant populations. The best study was conducted by Nielsen et al.

(51), who examined trends in food locations among adolescents and young adults, using a nationally representative sample of 16,810 individuals ages 12-29 from the 1977-1978 Nationwide Food Consumption Survey and the 1989-1991 and 1994-1996 Continuing Surveys of Food Intake by Individuals. Two important trends were identified. First, for both adolescents and young adults, there has been a decrease in the percentage of energy obtained from foods consumed at home. In 1994-96, about half (52.7 percent) of total energy intake for young adults and 60.5 percent of total energy intake for adolescents came from foods consumed at home, in comparison to nearly three-fourths of total energy intake obtained from foods consumed at home in 1977-78 (Table 2.15). Second, for both adolescents and young adults, the proportion of total energy coming from the restaurant/fast-food category has been increasing over time. Whereas in 1977-78 restaurants and fast food accounted for only 6.5 percent of total energy intake for adolescents and 14.3 percent of that for young adults, by 1994-96 restaurants and fast food contributed to nearly one-fifth (19.3 percent) of total energy intake for adolescents and nearly one-third (31.5 percent) of that for young adults. The changes in food locations probably contributed to significantly increased consumption of pizza, cheeseburgers, and salty snacks, and decreased consumption of desserts and certain milk and meat products observed over the years. The study did not report differences in food locations by race-ethnicity or SES.

More recently, Satia et al. (41) reported on the frequency of eating at fast-food restaurants in a population survey of 658 non-pregnant African Americans in North Carolina. Seventy-six percent reported eating at fast-food restaurants during the previous three months: four percent usually, twenty-two percent often, and fifty percent sometimes. Frequency of eating at fast-food restaurants was positively associated with total fat and saturated fat intakes and fat-related dietary behaviors ($P < 0.0001$) and inversely associated with vegetable intake ($P < 0.05$). Participants who reported usual/often eating at fast-food restaurants were younger, never married, obese, physically inactive, and multivitamin non-users (all $P < 0.01$). Frequency of eating at fast-food restaurants was positively associated with fair/poor self-rated health, weak belief in a diet-cancer relationship, low self-efficacy for healthy eating, weight dissatisfaction, and perceived difficulties of preparing healthy meals and ordering healthy foods in restaurants (all $P < 0.05$). Frequency of eating at fast-food restaurants was not associated with financial ability to purchase healthy foods, need for information on how to prepare healthy foods and meals, or knowledge of the Food Guide Pyramid. To our knowledge, this is the only published study of the psychosocial and behavioral correlates of fast-food consumption among African Americans.



TABLE 2.15: TRENDS IN ENERGY INTAKE, BY MEAL PATTERN TYPE AND SOURCE (PERCENTAGE OF ENERGY)*

	Years	Vending	At home	Store eaten out	Restaurant/ fast food	School	Other	Meal pattern type	Total energy (Kcal)
A. Adolescents aged 12-18									
Total energy	1977-1978	0.5 ^{AB}	74.1 ^{AB}	5.3 ^{AB}	6.5 ^{AB}	10.9 ^{AB}	2.6 ^{AB}	100	2,060 ^B
	1989-1991	0.3 ^{AC}	68.3 ^A	2.7 ^{AC}	16.7 ^{AC}	9.5 ^A	2.4 ^{AC}	100	2,030 ^C
	1994-1996	0.9 ^{BC}	60.5 ^B	5.2 ^{BC}	19.3 ^{BC}	7.9 ^B	6.2 ^{BC}	100	2,268 ^{BC}
Meals	1977-1978	0.3 ^{AB}	73.8 ^{AB}	4.9 ^{AB}	6.2 ^{AB}	12.4 ^{AB}	2.4 ^{AB}	100	1,780 ^A
	1989-1991	0.2 ^{AC}	67.5 ^{AC}	1.9 ^{AC}	17.4 ^{AC}	10.9 ^A	2.1 ^{AC}	100	1,723 ^{AC}
	1994-1996	0.5 ^{BC}	59.3 ^{BC}	3.9 ^{BC}	21.5 ^{BC}	9.5 ^B	5.3 ^{BC}	100	1,797 ^C
Snacks	1977-1978	1.7 ^{AB}	76.4 ^{AB}	8.0 ^B	8.2 ^{AB}	1.6 ^{AB}	4.0 ^{AB}	100	280 ^{AB}
	1989-1991	1.1 ^{AC}	72.9 ^{AC}	7.2 ^C	12.7 ^{AC}	1.7 ^{AC}	4.3 ^{AC}	100	307 ^{AC}
	1994-1996	2.3 ^{BC}	64.8 ^{BC}	10.5 ^{BC}	10.9 ^{BC}	1.8 ^{BC}	9.7 ^{BC}	100	471 ^{BC}
B. Young adults aged 19-29									
Total energy	1977-1978	1.2 ^B	71.4 ^B	7.9 ^{AB}	14.3 ^{AB}	0.6 ^{AB}	4.7 ^{AB}	100	1,899 ^{AB}
	1989-1991	1.1 ^C	67.3 ^C	3.3 ^{AC}	25.0 ^{AC}	0.4 ^{AC}	3.0 ^{AC}	100	1,999 ^{AC}
	1994-1996	1.2 ^{BC}	52.7 ^{BC}	7.5 ^{BC}	31.5 ^{BC}	0.7 ^{BC}	6.4 ^{BC}	100	2,274 ^{BC}
Meals	1977-1978	0.7 ^{AB}	72.2 ^{AB}	7.5 ^{AB}	14.5 ^{AB}	0.7 ^{AB}	4.4 ^{AB}	100	1,659 ^{AB}
	1989-1991	0.6 ^A	67.7 ^{AC}	2.5 ^{AC}	26.4 ^{AC}	0.4 ^{AC}	2.5 ^{AC}	100	1,720 ^{AC}
	1994-1996	0.6 ^B	52.4 ^{BC}	6.4 ^{BC}	33.8 ^{BC}	0.7 ^{BC}	6.1 ^{BC}	100	1,849 ^{BC}
Snacks	1977-1978	4.7 ^B	65.8 ^{AB}	10.3 ^B	12.5 ^{AB}	0.3 ^{AB}	6.4 ^B	100	240 ^{AB}
	1989-1991	4.5 ^C	64.6 ^{AC}	8.8 ^C	15.9 ^{AC}	0.5 ^A	5.7 ^C	100	270 ^{AC}
	1994-1996	4.0 ^{BC}	54.0 ^{BC}	12.3 ^{BC}	21.3 ^{BC}	0.4 ^B	7.9 ^{BC}	100	425 ^{BC}

Note. P<0.01: A1977-1978 and 1989-1991, significant; B1977-1978 and 1994-1996, significant; C1989-1991 and 1994-1996, significant.

* Adjusted for age, gender, education level, ethnicity, region, urban classification, household size, and percentage of poverty.

Source: S. J. Nielsen, A. M. Siega-Riz, and B. M. Popkin, "Trends in food locations and sources among adolescents and young adults," *Prev Med.* 35, no. 2 (August 2002): 107-13.

Pica

Pica is the craving for and ingestion of non-nutritive substances or food substances (52-53). Its prevalence among pregnant women has not been conclusively established. Horner et al. (54) conducted a systematic review of the literature from 1950-1990 and concluded that pica affected about one-fifth of high-risk pregnant women. Women at high risk of pica are more likely to be black, to live in rural areas, and to have a positive childhood and family history of pica. Edwards et al. (55) reported on the prevalence of pica in a convenience sample of 553 African American women who were admitted to prenatal clinics in Washington, D.C. In this sample, geophagia, compulsive eating of clay or dirt, was not observed. However, pagophagia, or the ingestion of large quantities of ice and freezer frost, was self-reported in 8.1 percent of the women, who consumed one-half to two cups a day from one to seven days per week. The authors hypothesized that pica in African American women may be a mediator of stress, acting through the immune system. Smulien et al. (56) reported a prevalence of 14.4 percent among a convenience prenatal sample of 125 rural women in Georgia; substances ingested included white and red dirt, ice, cornstarch, laundry starch, soap, ashes, chalk, paint, and burnt matches. Simpson et al. (57) found in a

convenience prenatal sample of 150 Mexican-American women residing in Santa Ana, Bakersfield, and Los Angeles a prevalence of 31 percent. Those who reported pica behavior more commonly had a relative who also practiced pica. In a convenience prenatal sample of 128 rural pregnant women in North Carolina, Corbett et al. (58) found 48 women (38 percent) who practiced pica. Of these, pagophagia was reported by 36 women and polypica was reported by 11 women; substances ingested consisted mostly of ice and freezer frost, but also laundry starch, cornstarch, clay dirt, and baked clay dirt. African American women reported practicing pica more often than other ethnicities. One report in the literature documents how women who practice pica feel about it. Cooksey (59) interviewed 300 pica-practicing African American women in the Midwest during their pregnancies. That qualitative study found that although the women kept the practice secret, pica was a large part of their lives. Much of their time had to be spent obtaining the substance they craved; they feared the effect of the substance on their fetus; they tried to overcome their cravings; some used the substances as "medications"; some altered their food intake because of the pica; and most enjoyed not only the taste of the substance, but also the odor (women in that study described a heightened sense of smell during their pregnancies). To date, the cause of pica remains unknown.



SUMMARY

According to 2003 national data, most pregnant women in the U.S. started off pregnancy overweight or underweight and had inappropriate weight gain during pregnancy. For low-income African American women, only 40 percent had normal BMI before pregnancy and less than 30 percent had ideal weight gain during pregnancy. Approximately one of every three low-income women was anemic in the third trimester of pregnancy; the prevalence of anemia was substantially higher (44 percent) among African American women than all other racial-ethnic groups. The prevalence of diabetes during pregnancy was 3.3 percent. The prevalence, however, ranged from 3 percent for non-Hispanic black mothers to 5.4 percent for Asian and Pacific Islander mothers and 5.7 percent for Native American mothers. With respect to dietary intakes among pregnant women, soft drinks, fruit juices, biscuits, muffins, white bread, and other refined carbohydrates were the leading sources of energy from carbohydrates, while mayonnaise, salad dressings, whole milk, French fries, and fried potatoes were the leading sources of energy from fats, according to several nutritional surveys. In general, white women consumed a higher nutrient-dense diet, and black women consumed more calories but fewer nutrients after energy adjustment. Available data suggest that pregnant women in the U.S. consumed more protein, fat and trans-fat, and carbohydrates than recommended. A substantial proportion of pregnant women did not meet their recommended daily allowances (RDA) for iodine, calcium, magnesium, iron, zinc, vitamins A, B1, B2, B3, B6, and B12, and vitamin C from food sources. Dietary intake of folate was inadequate for over 95 percent of women, and that of vitamin E was inadequate for 25 percent of pregnant women, perhaps reflecting low intakes of fruits and vegetables. When multivitamins were accounted for, one of every four still did not consume adequate amounts of folate and vitamin E. For African American women, only about one in four pregnant women met the RDA for calcium, magnesium, zinc, and vitamin E, and about one in three did not meet the RDA for iron and folate. Fasting, pica, and fast-food consumption were common among pregnant women, particularly among African American women.

CHAPTER 3

PRENATAL NUTRITIONAL INTERVENTIONS: EVIDENCE OF EFFECTIVENESS

Chapter 1 examined the relationship between nutrition and infant mortality. We found evidence linking poor maternal nutrition to several leading causes of infant mortality (although causality cannot be conclusively established). We also discussed how poor maternal nutrition may contribute to poor child health and development, as well as developmental programming of adult diseases.

Chapter 2 examined the nutritional status of pregnant women in the United States. We found that most pregnant women in the U.S. start off pregnancy overweight or underweight and have inappropriate weight gain during pregnancy. Approximately one of every three low-income women is anemic in the third trimester of pregnancy. Most pregnant women consume a diet high in refined carbohydrates and trans fats; in general, white women consume a higher nutrient-dense diet, and black women consume more calories but fewer nutrients after energy adjustment. We identified multiple micronutrient deficiencies in the dietary intake of pregnant women. Fasting, pica, and fast-food consumption are common, particularly among pregnant African American women.

If poor maternal nutrition is directly or indirectly related to several leading causes of infant mortality, and if the nutritional status of many pregnant women in the U.S. is poor in many respects, it follows that improving maternal nutritional status should be a key strategy for reducing infant mortality in the United States. In this chapter, we review the evidence of effectiveness of prenatal nutritional interventions for preventing the leading causes of infant mortality: preterm birth, fetal growth restriction, birth defects, and maternal complications of pregnancy. We also evaluate evidence of the effectiveness of the Special Supplemental Nutrition Program for Women, Infants, and Children (WIC) for preventing leading causes of infant mortality and promoting maternal and infant health. Lastly, we critique current prenatal nutritional interventions in terms of their timing, contents, and contexts.

I. PREVENTION OF PRETERM BIRTH

The effectiveness of prenatal nutritional interventions for preventing preterm birth was evaluated by Villar et al. (1) in their recent systematic review of the literature. Their findings are summarized below.



Nutritional Advice

Kramer (2) reviewed four trials on nutritional advice, only one of which reported preterm birth as an outcome. Although the trial showed a protective effect of the nutritional advice on preterm birth, the conclusions are limited by a number of methodological weaknesses, including post-randomization exclusions, disregard of cluster effect, and non-adherence to intention-to-treat rule.

Balanced Protein-Energy Supplementation

Kramer (3) reviewed thirteen trials in which the protein content of the energy supplementation was balanced (less than 25 percent of the total energy content from protein), five of which reported preterm birth as an outcome. Most of the trials were conducted in developing countries and inner-city populations. When the data were pooled in the meta-analysis, preterm delivery rates did not differ between the supplemented and control groups.

Isocaloric Protein-Energy Supplementation

Kramer (4) reviewed three trials on isocaloric protein-energy supplementation; only one, of poor methodological quality, reported preterm birth as an outcome. No differences were found between the supplemented and control groups.

High Protein Supplementation

Kramer (5) reviewed two trials on high-protein supplementation, defined as greater than 25 percent of the total energy supplementation from protein. Only one trial, of good methodological quality, reported the effect on preterm birth and showed that high protein supplementation was not associated with a clinically or statistically significant difference in the rates of preterm birth.

Protein and Energy Restriction

Kramer (6) reviewed three trials on protein and energy restriction in women who are overweight or gained excess weight during pregnancy. Only one small trial, of poor methodological quality, reported preterm birth as an outcome and found no statistically significant difference in the rates of preterm birth between treatment and control groups.

Salt Restriction

Duley et al. (7) reviewed two randomized controlled trials on prenatal salt restriction; only one reported on preterm birth and it found no statistically significant difference between treatment and

control groups. The small sample size of this trial, however, does not allow reliable answers to be extracted.

Calcium Supplementation

Villar et al. (1) reviewed nine randomized controlled trials on calcium supplementation that reported preterm birth as an outcome. Overall, no protective effect of calcium supplementation on preterm delivery could be demonstrated. Among women at high risk for high blood pressure during pregnancy, there was a statistically significant 57 percent reduction in the risk of preterm birth associated with calcium supplementation. No protective effect of calcium supplementation on preterm delivery was observed among women at low risk of pregnancy hypertension or with inadequate dietary calcium intake. These results are in agreement with the most recent update of the Cochrane Library systematic review by Hofmeyr et al. (8).

Iron Supplementation

Mahomed reviewed twenty randomized controlled trials on iron supplementation during pregnancy (9), only one of which reported preterm birth as an outcome. This large trial was conducted in a well-nourished Finish population; no statistically significant difference was found in preterm birth rates between routine iron supplementation compared with selective supplementation, although there appeared to be a trend toward an increased risk of prematurity with iron supplementation.

Folate Supplementation

Mahomed reviewed twenty-one randomized controlled trials on folate supplementation during pregnancy (10), four of which reported the effect on preterm birth. No statistically significant difference in preterm birth rates was found between the treatment and control groups.

Iron and Folate Supplementation

Mahomed (11) reviewed eight randomized controlled trials on combined iron and folate supplementation during pregnancy; only one small trial reported preterm birth as an outcome and it found no statistically significant difference in preterm birth rates between the treatment and control groups.

Magnesium Supplementation

Makrides et al. (12) reviewed seven randomized controlled trials on magnesium supplementation during pregnancy, five of which



reported preterm birth as an outcome. Oral magnesium supplementation started before 25 weeks of gestation was associated with a statistically significant 27 percent reduction in preterm birth rates. However, these results should be interpreted cautiously because most of these trials had methodological weaknesses and significant heterogeneity was noted in the meta-analysis, with a less protective effect observed in large trials or those with higher quality. In fact, the trial with the highest methodological quality showed no effect on reducing preterm birth.

Fish Oil Supplementation

Duley (13) reviewed three randomized controlled trials on fish oil supplementation, two of which reported preterm birth as an outcome. The first trial dominated the review because of its large sample size (N = 5,017); its results indicate a statistically significant 20 percent reduction in preterm birth rates with fish oil supplementation. However, the conclusions were limited by inadequate randomization and allocation concealment. Three studies have been published subsequent to the review by Dudley. The first two were small and found no statistically significant difference in preterm birth rates between treatment and control groups. The third is an international multicenter program that included six trials, one of which used fish oil prophylactically among women who had previously experienced preterm delivery and showed a statistically significant 46 percent reduction in the recurrence of preterm delivery compared with olive oil. Fish oil reduced recurrence risk of preterm delivery from 33 percent to 21 percent among women with prior preterm birth. The results of these trials are not conclusive, but fish oil appears to be a promising intervention for preventing preterm delivery.

Zinc Supplementation

Mahomed reviewed seven randomized controlled trials on prenatal zinc supplementation (14), five of which reported preterm birth as an outcome. Overall, zinc supplementation was protective and showed a statistically significant 26 percent reduction in the risk of preterm birth. However, none of the three trials published subsequent to Mahomed's systematic review observed an effect of zinc supplementation on gestational age or rate of preterm delivery.

Summary

Villar et al. (1) concluded from their systematic review of the literature that “[n]o specific nutrient supplementation was identified for reducing preterm delivery.” Their findings are summarized

in **Table 3.1**. Magnesium supplementation remains controversial due to methodological weaknesses of the included trials. Fish oil appears promising in light of recent evidence for prevention of recurrent preterm birth. Zinc supplementation also appears promising but needs further testing.

II. PREVENTION OF FETAL GROWTH RESTRICTION OR LOW BIRTHWEIGHT

The effectiveness of prenatal nutritional interventions for preventing fetal growth restriction or low birthweight (LBW) was evaluated by Meriardi et al. (15) in their recent systematic review of the literature. LBW is comprised of preterm birth and fetal growth restriction, both of which are important causes of infant mortality. For the purpose of this review, fetal growth restriction and small-for-gestational-age (SGA) are used interchangeably, both defined as less than the 10th percentile in weight for a given gestation unless otherwise stated. The findings of Meriardi et al. are summarized below.

Nutritional Advice

Kramer (2) reviewed four randomized controlled trials on nutritional advice during pregnancy; only one trial, of poor methodological quality, reported SGA as an outcome. No effect of the intervention was observed.

TABLE 3.1: SUMMARY OF SYSTEMATIC REVIEWS OF NUTRITIONAL INTERVENTION TRIALS IN PREGNANCY FOR THE PREVENTION OF PRETERM BIRTH

Type of nutrient supplementation	No. of trials (Ref)	No. trials w/ outcome	EXPT	CTRL	RR	95% CI
Balanced protein/energy	13 (3)	6	97/1225	118/1211	0.83	0.65-1.06
Isocaloric balanced protein	3 (4)	1	40/391	38/391	1.05	0.69-1.60
High protein supplementation	2 (5)	1	62/249	56/256	1.14	0.83-1.56
Energy/protein restriction	3 (6)	1	2/91	4/91	0.50	0.09-2.66
Salt restriction	2 (7)	1	9/110	10/132	1.08	0.46-2.56
Calcium	11 (8)	9	305/3309	327/3362	0.95	0.82-1.10
Iron	20 (9)	1	57/1358	40/1336	1.40	0.94-2.09
Folate	21 (10)	4	51/715	49/710	1.03	0.71-1.49
Iron and folate	8 (11)	1	3/24	0/24	7.00	0.38-128.6
Magnesium	7 (12)	5	86/1125	121/1150	0.73	0.57-0.94
Fish oil	3 (13)	2	505/2509	603/2508	0.82	0.75-0.92
Zinc	7 (14)	5	80/1259	109/1280	0.74	0.56-0.98

Source: J. Villar, M. Meriardi, A. M. Gulmezoglu, E. Abalos, G. Carroli, R. Kulier, and M. de Onis, “Nutritional interventions during pregnancy for the prevention or treatment of maternal morbidity and preterm delivery: an overview of randomized controlled trials,” *J Nutr.* 133, no. 5, Suppl 2 (May 2003): 1606S-1625S.



Balanced Protein-Energy Supplementation

Kramer (3) reviewed thirteen randomized controlled trials on balanced protein-energy supplementation, of which six reported SGA as an outcome and all reported a protective effect, although only one trial showed statistically significant results. Pooled data showed a statistically significant 32 percent reduction in SGA, which is of clinical and public health relevance.

Eleven trials reported birthweight as an outcome. Infants born to mothers who received supplementation tended to be heavier than infants born to non-supplemented mothers. However, the overall difference in mean birthweight was small and not significant. One high-quality study was conducted in rural Gambia among undernourished women (135). In the intervention villages, pregnant women received two supplement biscuits containing roasted groundnuts, rice flour, sugar, and groundnut oil with 1,017 kcal of energy and 22 g of protein, beginning at 20 weeks' gestation. Balanced protein-energy supplementation was associated with a statistically significant 39 percent reduction in LBW, a 53 percent reduction in fetal mortality, and a 46 percent reduction in neonatal mortality. The reduction in LBW was achieved primarily through reduced incidence of SGA and not preterm birth.

Isocaloric Balanced Protein Supplementation

Kramer (4) reviewed three randomized controlled trials on isocaloric balanced protein supplementation in pregnancy, only one of which reported SGA as an outcome. This trial showed an adverse effect of supplementation on the risk of SGA. Three trials reported data on birthweight. Isocaloric balanced protein supplementation was associated with a statistically significant 63.5 gram reduction in mean birthweight.

High Protein Supplementation

Kramer (5) reviewed two randomized controlled trials on high protein supplementation during pregnancy. High protein supplementation was associated with increased risk of preterm SGA. The same trial—conducted among women of low socioeconomic status in the United States—showed a statistically nonsignificant 58.4 gram (95%CI: -146.2, 29.5) decrease in birthweight. The other trial included 25 Indian women and found no difference in the birthweight of infants born to mothers who received high protein supplements compared with those of mothers who did not. In the U.S. trial, high protein supplementation was also associated with an excess of very early premature births and a statistically nonsignificant increase in neonatal deaths. These results suggest that high protein supplementation during pregnancy may be harmful to the fetus and should be avoided.

Energy Protein Restriction

Kramer (6) reviewed three trials on protein and energy restriction in women who are overweight or gained excess weight during pregnancy. None of the trials reported data on LBW or SGA. Two trials reported data on birthweight; one found no difference, while the other found a statistically significant 450 gram reduction in birthweight of infants born to obese women placed on energy protein restriction during pregnancy.

Salt Restriction

Duley (7) reviewed two trials on reduced salt intake compared with normal dietary salt or high salt intake during pregnancy. Both trials found no differential effect on SGA or LBW, although the results trended in opposite directions.

Calcium Supplementation

Villar et al. (1) reviewed nine randomized controlled trials on calcium supplementation during pregnancy, two of which reported SGA as an outcome. Neither study found an effect on SGA, although both studies had small sample sizes and wide confidence intervals. Seven trials presented data on LBW and tended to show a protective effect of calcium supplementation on fetal growth. The overall result showed a statistically significant 17 percent reduction in the risk of LBW associated with calcium supplementation. All seven trials found higher mean birthweights in neonates from calcium supplemented mothers compared to those of unsupplemented women. The observed difference in mean birthweights was statistically significant in three studies.

Iron Supplementation

Mahomed (9) reviewed twenty randomized controlled trials on iron supplementation during pregnancy. Only one trial, comparing selective versus routine supplementation, reported SGA and LBW as outcomes and found no difference in fetal growth by type of supplementation.

Folate Supplementation

Mahomed (10) reviewed twenty-one randomized controlled trials on folate supplementation during pregnancy, five of which reported data on LBW. Although not significant, the overall result shows a tendency toward a decrease in the risk of LBW associated with folic acid supplementation. However, there was heterogeneity in the results, with one trial reporting an increased risk of LBW and reduction in mean birthweight.



Iron and Folate Supplementation

Mahomed (11) reviewed eight randomized controlled trials of combined iron and folate supplementation in pregnancy, only one of which had data on LBW. The small sample size and wide confidence intervals of the study preclude us from drawing any conclusions about the effect of iron and folate supplementation on LBW.

Magnesium Supplementation

Makrides et al. (12) reviewed seven randomized controlled trials on magnesium supplementation during pregnancy (21). Pooled data showed a statistically significant 30 percent reduction in SGA in three trials, and a 33 percent reduction in LBW in four trials. One trial reported a nonsignificant reduction in the risk of very low birthweight (VLBW) following magnesium supplementation. Four trials found that magnesium supplementation was associated with a 50.8 gram increase in mean birthweight. However, as mentioned above, these results should be interpreted cautiously because most of these trials had methodological weaknesses. For example, one trial did not adjust for its cluster design in data analyses; when this trial was excluded from the meta-analysis, the direction of the effect on fetal growth did not change but the effect was no longer statistically significant.

Fish Oil Supplementation

Duley (13) reviewed three randomized controlled trials on fish oil supplementation and found an overall tendency toward an increased risk of birthweight below the 3rd or 5th percentile associated with fish oil supplementation (odds ratio 1.56; 95%CI: 0.56-4.35). Three additional trials have been published subsequent to this review. In an international multicenter study, fish oil supplementation showed no benefit in preventing SGA when used prophylactically in women with intrauterine growth restriction (IUGR) in a previous pregnancy, or therapeutically in women with suspected intrauterine growth impairment diagnosed by ultrasonically estimated fetal weight below the 10th percentile. Two additional trials in women at high risk for IUGR and/or pregnancy-induced hypertension also found no benefit of fish oil supplementation in preventing fetal growth impairment.

Zinc Supplementation

Mahomed (14) reviewed seven randomized controlled trials on prenatal zinc supplementation and found no effect on SGA in

three trials, and a nonsignificant reduction in risk of LBW (RR 0.77; 95%CI: 0.56-1.06) associated with maternal zinc supplementation in five trials. Three trials reported mean birthweight as an outcome and found no overall effect; the only trial that showed a positive effect of zinc supplementation on birthweight (mean difference: 126 gm; 95%CI: 12.1, 239.9), but not LBW rate (RR 0.62; 95%CI: 0.56-1.06) or gestational age rates (RR 0.86; 95%CI: 0.64-1.28) was conducted among low-income pregnant women in Alabama with low serum zinc concentrations at entry into prenatal care. The results of three studies published after this systematic review and conducted in developing countries did not find an effect of zinc supplementation on birthweight.

Vitamin D

Mahomed (16) reviewed two randomized controlled trials on vitamin D supplementation. One study found no effect of vitamin D on SGA or birthweight; the second found that infants born to women who received 1,000 IU of vitamin A daily and 200,000 IU of vitamin D in a single dose had, on average, a lower birthweight than those born to women who received no supplementation.

Vitamins C and E

One trial (17) of vitamin C and E supplementation among pregnant women at increased risk for preeclampsia reported a nonsignificant reduction in the risk for SGA associated with vitamin supplementation (RR 0.74; 95%CI: 0.50-1.08).

Summary

Merialdi et al. (15) evaluated the effectiveness of nutritional supplementation during pregnancy for preventing LBW and SGA. Their findings are summarized in Table 3.2. Overall, balanced protein-energy supplementation reduced the risk of SGA by 30 percent (120), while calcium supplementation also protected against LBW (124). High protein supplementation was associated with increased risk of SGA and a nonsignificant increased risk of neonatal death and should be avoided in pregnancy. No other micronutrient supplementation was found to significantly affect birthweight except for magnesium supplementation, which reduced the risk of SGA by 30 percent. This finding, however, needs to be interpreted with caution because of methodological issues in the data analysis (Table 3.2).



III. PREVENTION OF BIRTH DEFECTS

Birth defects are a leading cause of infant mortality in the United States. Because organogenesis (formation of major organs) occurs early in pregnancy, about 4-8 weeks after conception, nutritional interventions need to begin before or early in pregnancy in order to be effective for preventing birth defects. For example, the neural tube closes at 28 days after conception. Periconceptional folic acid supplementation has been shown to reduce the occurrence and recurrence of neural tube defects (NTD); however, if the supplementation is begun beyond 28 days after conception, no benefit is observed. Therefore, the few nutritional intervention trials that have been conducted to prevent birth defects all began periconceptionally.

Periconceptional Folate Supplementation

Lumley (18) evaluated the effectiveness of periconceptional folate supplementation on preventing neural tube and other birth defects. Four trials (19-22) involving 6,425 women were included. Periconceptional folate supplementation was associated with a statistically significant 72 percent (RR 0.28; 95%CI: 0.13, 0.58) reduction in the prevalence of neural tube defects. The reduction is similar for occurrent defects (those where the mother has not had a previously affected fetus or infant) and for recurrent defects (where the mother has had a previously affected infant), as shown in Table 3.3. The number needed to treat (NNT) for folate prevention of a neural tube defect is 847; that is, one neural tube defect could be prevented for every 847 women who receive periconceptional folate supplementation. Folate supplementation did not significantly increase miscarriage, ectopic pregnancy, stillbirth, LBW, or preterm birth. Pooled data do not suggest a benefit of periconceptional folate supplementation on limb reduction, conotruncal heart, orofacial, or other birth defects, although the study by Czeizel (22) found a 46 percent reduction in other birth defects (Table 3.3).

TABLE 3.2: SUMMARY OF SYSTEMATIC REVIEWS OF NUTRITIONAL INTERVENTION TRIALS IN PREGNANCY FOR THE PREVENTION OF SMALL-FOR-GESTATIONAL AGE (SGA) AND LOW BIRTHWEIGHT (LBW)

Type of nutrient supplementation	No. of trials (Ref)	No. of trials w/ outcome	EXPT	CTRL	RR	95% CI
SGA						
Balanced protein/energy	13 (3)	6	185/2147	275/2128	0.68	0.57-0.80
Isocaloric balanced protein	3 (4)	1	171/391	127/391	1.35	1.12-1.61
High protein supplementation	2 (5)	1	46/249	80/256	1.58	1.03-2.41
Salt restriction	2 (6)	1	15/110	12/132	1.50	0.73-3.07
Calcium	11 (1)	2	6/97	8/93	0.72	0.26-1.99
Iron, selective versus routine	20 (9)	1	81/1355	73/1335	1.09	0.80-1.49
Intravenous versus regular iron	5 (9)	1	8/50	5/50	1.60	0.56-4.56
Magnesium	7 (12)	3	72/865	104/876	0.70	0.53-0.93
Zinc	7 (14)	3	55/909	62/931	0.90	0.64-1.28
Vitamin D	2 (16)	1	9/67	19/59	0.54	0.26-1.10
LBW						
Salt restriction	2 (7)	1	14/184	16/177	0.84	0.42-1.67
Calcium	11 (1)	7	234/3230	283/3261	0.83	0.71-0.98
Iron, selective versus routine	20 (9)	1	42/1358	37/1336	1.12	0.72-1.73
Folate	21 (10)	5	38/754	50/734	0.75	0.50-1.12
Iron and folate	8 (11)	1	2/24	0/24	5.0	0.25-98.97
Magnesium	7 (12)	4	44/968	67/986	0.67	0.46-0.96
Zinc	7 (14)	5	62/750	75/722	0.77	0.56-1.06

Source: M. Meriardi, G. Carroli, J. Villar, E. Abalos, A. M. Gulmezoglu, R. Kulier, and M. de Onis, "Nutritional interventions during pregnancy for the prevention or treatment of impaired fetal growth: an overview of randomized controlled trials," *J Nutr.* 133, no. 5, Suppl 2 (May 2003): 1626S-1631S.

TABLE 3.3: SUMMARY OF PERICONCEPTIONAL FOLATE SUPPLEMENTATION TRIALS FOR THE PREVENTION OF NEURAL TUBE DEFECTS

Author (Reference)	Dosage	NTD	Folate	No Folate	RR	95% CI
Czeizel (1994)	0.8 mg	Occurrence	0/2471	6/2391	0.07	0.00-1.32
Kirk (1992)	0.36 mg	Recurrence	0/169	1/88	0.17	0.01-4.24
Laurence (1981)	4 mg	Recurrence	2/60	4/51	0.43	0.08-2.23
MRC (1991)	4 mg	Recurrence	6/593	21/602	0.29	0.12-0.71
Meta-analysis		All NTD	8/3293	32/3132	0.28	0.13-0.58

Source: J. Lumley, L. Watson, M. Watson, and C. Bower, "Periconceptional supplementation with folate and/or multivitamins for preventing neural tube defects," *The Cochrane Database of Systematic Rev.*, Iss. 3, Art. No.: CD001056. DOI: 10.1002/14651858.CD001056 (2001).



Blood Glucose Control for Pregestational Diabetes Mellitus

Women with pregestational diabetes are at greater risk for having babies with birth defects, particularly heart and neural tube defects (23). The risk appears to depend on blood glucose control in early pregnancy. Korenbrot et al. (24) reviewed seven trials of preconception care for women with pregestational diabetes; interventions included screening, counseling, and monitoring of blood sugar control. All studies that presented results of blood sugar control found preconception care to be associated with improved control early in pregnancy. Overall, preconceptional care was associated with reduced prevalence of major congenital malformations, although not all of the studies had statistically significant results. The prevalence of congenital malformation ranged from 1.2 to 5.0 percent within the preconception care groups, compared to a range of 10.9 to 14.0 percent within the prenatal groups.

Dietary Restrictions for Hyperphenylalaninemia

Hyperphenylalaninemia is a group of inherited disorders of phenylalanine metabolisms that includes phenylketonuria (PKU). High maternal levels of phenylalanine during pregnancy can cause fetal microcephaly (small head size), birth defects (particularly congenital heart defects), and mental retardation. It is recommended that women with PKU restrict their dietary intake of phenylalanine (an essential amino acid) at least 3 months prior to pregnancy and continue the diet throughout the pregnancy. Korenbrot et al. (24) reviewed four studies of preconception and prenatal dietary restrictions; interventions included preconceptional screening, education, and counseling regarding dietary control. Preconception dietary restrictions that resulted in lowered levels of maternal phenylalanine during the earliest weeks of pregnancy were associated with reduced incidences of congenital malformations. In these studies, preconceptional or very early prenatal dietary control was associated with better outcomes than was later prenatal dietary control alone.

Summary

Periconceptional folate supplementation reduces the risk of neural tube defects by 72 percent. Blood glucose control in women with pregestational diabetes, as well as dietary restrictions in women with PKU early in pregnancy, have been shown to reduce the risk of congenital malformations.

IV. PREVENTION OF MATERNAL COMPLICATIONS OF PREGNANCY

Maternal complications of pregnancy can result in fetal or infant deaths. In this review we will focus on preeclampsia, anemia, and infections. The effectiveness of prenatal nutritional interventions for preventing these maternal complications of pregnancy was evaluated by Villar et al. (1) in their recent systematic review of the literature. Their findings are summarized below.

A. GESTATIONAL HYPERTENSION OR PREECLAMPSIA

Balanced Protein-Energy Supplementation

Kramer (3) reviewed thirteen trials in which the protein content of the energy supplementation was balanced (less than 25 percent of the total energy content from protein). Three trials, of poor methodological quality, reported gestational hypertension or preeclampsia as an outcome. No significant beneficial effect was noted.

Isocaloric Balanced Protein Supplementation

Kramer (4) reviewed three trials on isocaloric protein-energy supplementation; only one trial evaluated preeclampsia prevention and found no effect among underweight pregnant women.

Protein and Energy Restriction for Obese Women

Kramer (6) reviewed three trials on protein and energy restriction in women who are overweight or gained excess weight during pregnancy; two reported preeclampsia and three reported gestational hypertension as an outcome. The trials found no effect. This limited evidence suggests that protein-energy restriction for pregnant women who are overweight or exhibit high weight gain during pregnancy is unlikely to be beneficial and may be harmful to the developing fetus (i.e., result in intrauterine growth retardation).

Salt Restriction

Duley (7) reviewed two trials on reduced salt intake compared with normal dietary salt or high salt intake during pregnancy. No effect was found in preventing preeclampsia or gestational hypertension.



Calcium Supplementation

Villar et al. (1) reviewed eleven trials of calcium supplementation during pregnancy. The authors' pre-specified stratified analysis took into account women's risk of hypertensive disorders of pregnancy (low vs. increased) and baseline dietary calcium intake (low: <900 mg/d vs. adequate: 900 mg/d) (86). Overall, calcium supplementation reduced the risk of gestational hypertension by 19 percent (typical RR 0.81; 95%CI: 0.74-0.89) and the risk of preeclampsia by 30 percent (typical RR 0.70; 95%CI: 0.58-0.83), but there was heterogeneity in the magnitude of the effects across the subgroups. The effect was considerably greater for women at high risk of developing hypertension than for those at low risk, and it was greater for those with inadequate calcium intake than for those with adequate calcium intake. The results from the largest trial conducted by the National Institutes of Health, which studied only low-risk women with adequate calcium in their baseline diet (all of whom received low-dose calcium supplementation as part of their routine antenatal care), showed no significant effect on hypertension or preeclampsia (25). Based on these results, populations with adequate dietary calcium intake are presently not encouraged to take routine calcium supplementation during pregnancy. However, evidence from this review supports the view that calcium supplementation might benefit women at high risk or women with low dietary calcium intake.

Folate Supplementation

Mahomed (10) reviewed twenty-one randomized controlled trials on folate supplementation during pregnancy, two of which reported gestational hypertension as an outcome and found no effect of folate supplementation on the occurrence of gestational hypertension.

Iron and Folate Supplementation

Mahomed (11) reviewed eight randomized controlled trials on combined iron and folate supplementation during pregnancy, two of which reported gestational hypertension as an outcome. In women with normal hemoglobin levels, combined iron and folate supplementation showed no effect on the occurrence of gestational hypertension.

Magnesium Supplementation

Makrides et al. (12) reviewed seven randomized controlled trials on magnesium supplementation during pregnancy, two of which reported preeclampsia as an outcome and showed no apparent effect of magnesium supplementation on the prevention of

preeclampsia. The methodological quality of these trials was poor, especially related to concealment of treatment allocation, and the results may have been confounded by the fact that, in the largest trial, all women (including those assigned to the control group) received a multivitamin containing low doses of magnesium.

Fish Oil Supplementation

Duley (13) reviewed three randomized controlled trials on fish oil supplementation, two of which reported gestational hypertension and preeclampsia as an outcome. Fish oil supplementation was found to have a statistically significant 30 percent reduction in the rate of preeclampsia, but this reduction was strongly influenced by a large nonrandomized trial conducted in 1942 in which vitamins and minerals were given to women in addition to fish oil. None of the four trials published subsequent to the Cochrane systematic review demonstrated any differences between groups in the incidence of hypertension during pregnancy or preeclampsia, including the aforementioned international multicenter study. Fish oil showed no effect in preventing gestational hypertension or preeclampsia in women with hypertension in a prior pregnancy or those with uncomplicated twin pregnancies.

Zinc Supplementation

Mahomed (14) reviewed seven randomized controlled trials on prenatal zinc supplementation and found no effect on gestational hypertension in four trials. This was expected because no biological link between zinc and hypertension in adults is known.

Vitamins E and C Supplementation

One randomized controlled trial (17) on prophylactic vitamin E and C supplementation involved women at high risk of developing preeclampsia, defined as those having abnormal Doppler waveform in either uterine artery at 18-22 weeks' gestation or a history of preeclampsia in the previous pregnancy. Prophylactic vitamin E and vitamin C supplements were associated with a statistically significant 54 percent reduction in the risk of developing preeclampsia. Although these findings are promising, they were from one small trial and further studies are needed to evaluate the effectiveness of vitamin E and C supplementation for the prevention of preeclampsia.

Vitamin A Supplementation

No trials have been published that assess the effect of vitamin A supplementation on gestational hypertension or preeclampsia. A double-blind, cluster-randomized trial of low-dose vitamin A or



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TABLE 3.4: SUMMARY OF SYSTEMATIC REVIEWS OF NUTRITIONAL INTERVENTION TRIALS ON PREGNANCY FOR THE PREVENTION OF PREECLAMPSIA OR GESTATIONAL HYPERTENSION

Maternal outcome & type of nutrient supplementation	No. of trials (Ref)	No. of trials w/ outcome	EXPT	CTRL	RR	95% CI
Preeclampsia						
Balanced protein/energy	13 (3)	3	34/258	28/258	1.20	0.77-1.89
Iso-caloric balanced protein	3 (4)	1	23/391	23/391	1.00	0.57-1.75
Energy/protein restriction	3 (6)	2	17/142	15/142	1.13	0.59-2.18
Salt restriction	2 (7)	2	10/294	9/309	1.11	0.46-2.66
Calcium						
Low risk of hypertension	6 (1)	6	188/3146	240/3161	0.79	0.65-0.94
High risk of hypertension	5 (1)	5	9/281	54/306	0.21	0.11-0.39
Adequate intake	4 (1)	4	169/2505	197/2517	0.86	0.71-1.05
Inadequate intake	6 (1)	6	27/907	90/935	0.32	0.21-0.49
Magnesium	7 (12)	2	34/235	40/239	0.87	0.57-1.32
Fish Oil	3 (13)	2	100/2510	143/2511	0.70	0.55-0.90
Vitamins E and C	1 (17)	1	11/141	24/142	0.46	0.24-0.91
Gestational hypertension (with or without proteinuria)						
Energy/protein restriction	3 (6)	3	70/192	72/192	0.97	0.75-1.26
Salt restriction	2 (7)	1	13/110	16/132	0.97	0.49-1.94
Calcium						
Low risk of hypertension	6 (1)	6	611/3146	732/3161	0.84	0.76-0.92
High risk of hypertension	5 (1)	4	25/156	65/171	0.45	0.31-0.66
Adequate intake	4 (1)	4	547/2505	614/2517	0.90	0.81-0.99
Inadequate intake	6 (1)	5	79/782	172/800	0.49	0.38-0.62
Iron and folate	8 (11)	2	5/40	6/47	1.15	0.41-3.81
Folate	21 (10)	2	64/348	51/348	1.26	0.90-1.76
Fish oil	3 (13)	2	516/2553	537/2555	0.96	0.86-1.07
Zinc	7 (14)	4	77/967	89/995	0.87	0.65-1.15
Vitamins E and C	1 (17)	1	16/141	13/142	1.24	0.62-2.48
<i>Source: J. Villar, M. Meriardi, A. M. Gulmezoglu, E. Abalos, G. Carroli, R. Kulier, and M. de Onis, "Nutritional interventions during pregnancy for the prevention or treatment of maternal morbidity and preterm delivery: an overview of randomized controlled trials," J Nutr. 133, no. 5, Suppl 2 (May 2003): 1606S-1625S.</i>						

β -carotene supplementation carried out in Nepal showed a 40 percent reduction in overall maternal mortality among women who received a vitamin A supplement (26). When differences in cause of death, including preeclampsia and eclampsia, were evaluated, the rates of these conditions were not different between supplemented and placebo groups.

Summary

Villar et al. (1) evaluated the effectiveness of nutritional supplementation during pregnancy for preventing gestational hypertension and preeclampsia. Only calcium supplementation reduced the incidence of both preeclampsia and hypertension, especially among women at high risk of hypertension during pregnancy or low calcium intake. Calcium supplementation reduced the odds of preeclampsia by 79 percent among high-risk women and by 68 percent among women with inadequate intake. Fish oil and

vitamins E and C also appeared promising for preventing preeclampsia and need further testing. The findings are summarized in **Table 3.4**.

B. MATERNAL ANEMIA

Iron Supplementation

Mahomed (9) reviewed twenty randomized controlled trials on iron supplementation during pregnancy, twelve of which reported maternal anemia as an outcome. All twelve studies showed a significant reduction in maternal anemia (hemoglobin < 10 g/dL) in late pregnancy; routine iron supplementation reduced maternal anemia by 85 percent (RR 0.15; 95%CI: 0.11, 0.20).



Folate Supplementation

Mahomed (10) reviewed twenty-one randomized controlled trials on folate supplementation during pregnancy, six of which reported maternal anemia as an outcome and found a statistically significant 28 percent reduction in maternal anemia in late pregnancy.

Iron and Folate Supplementation

Mahomed (11) reviewed eight randomized controlled trials on combined iron and folate supplementation during pregnancy, six of which reported maternal anemia as an outcome. Iron and folate supplementation was associated with a statistically significant 78 percent reduction in maternal anemia in late pregnancy.

Magnesium Supplementation

Makrides et al. (12) reviewed seven randomized controlled trials on magnesium supplementation during pregnancy, two of which reported antepartum hemorrhage as an outcome. The systematic review showed a statistically significant large reduction in antepartum hemorrhage with magnesium supplementation. However, because this result is based on two trials of poor methodological quality, particularly with concealment of treatment allocation, and given that there is no likely biological explanation for this effect, the authors attribute this relationship to the poor quality of one trial and the high likelihood of bias.

Vitamin A Supplementation

In the systematic review by Villar et al. (1), three randomized controlled trials of vitamin A supplementation that presented anemia-related outcomes were identified. One trial suggested that a combination of iron and vitamin A resulted in a greater rise in hemoglobin in Indonesia (27), while the other two trials (28, 1), both conducted in Malawi among women receiving iron and

folate supplements, did not find any improvement in the number of women with anemia.

Calcium Supplementation

Calcium may inhibit both heme and nonheme iron absorption (29). Villar et al. (1) examined the prevalence of maternal anemia in a trial of calcium supplementation. Among pregnant women taking iron supplementation, the addition of calcium did not reduce hemoglobin values compared to placebo. Villar et al. concluded that these data support the concept that the calcium-iron interaction under regular antenatal care does not appear to be clinically significant.

Summary

The systematic review by Villar et al. (1) found evidence of effectiveness for prevention of anemia with iron supplementation, folate supplementation, and iron and folate supplementation. Magnesium supplementation remains controversial due to methodological weaknesses of included studies. The effect of vitamin A and calcium supplementation on maternal anemia remains unclear. The findings are summarized in **Table 3.5**.

C. MATERNAL INFECTIONS

Zinc Supplementation

Mahomed (14) reviewed seven randomized controlled trials on prenatal zinc supplementation; only one trial, of high methodological quality, reported on maternal infection. In an adequately nourished population in England, zinc supplementation showed no effect in preventing maternal infections (30). None of the studies published after the last update of the systematic review reported maternal infection as an outcome (1).

Type of nutrient supplementation	No. of trials (Ref)	No. of trials w/ outcome	EXPT	CTRL	RR	95% CI
Iron	20 (9)	12	38/881	212/921	0.15	0.11-0.20
Folate	21 (10)	6	463/1494	653/1620	0.72	0.66-0.80
Iron and folate	8 (11)	6	27/673	98/426	0.22	0.15-0.33
Magnesium (hemorrhage)	7 (12)	2	7/463	19/479	0.38	0.16-0.90
Vitamin A	5 (1)	3	274/513	157/300	0.91	0.80-1.04

Source: J. Villar, M. Meriardi, A. M. Gulmezoglu, E. Abalos, G. Carroli, R. Kulier, and M. de Onis, "Nutritional interventions during pregnancy for the prevention or treatment of maternal morbidity and preterm delivery: an overview of randomized controlled trials," *J Nutr.* 133, no. 5, Suppl 2 (May 2003): 1606S-1625S.



Vitamin A Supplementation

Only one trial has reported the effect of vitamin A supplementation on maternal infections. The aforementioned Nepal cluster-randomized trial (26) of low-dose vitamin A or β -carotene supplementation found a significant reduction in all-cause maternal mortality (40 percent reduction with vitamin A and 50 percent reduction with β -carotene). Maternal deaths due to infection were similar in the vitamin A and placebo groups but fewer in the β -carotene group.

Summary

There is no conclusive evidence supporting the benefit of zinc or vitamin A supplementation for preventing maternal infections. The systematic reviews on nutritional advice and protein-energy, calcium, iron and folate, magnesium, and fish oil supplementation did not report maternal infection as an outcome.

V. PROMOTION OF CHILD HEALTH AND DEVELOPMENT

Although our main focus is on the leading causes of infant mortality, we also searched for studies on the effect of prenatal nutritional supplementation on long-term child health and development, such as childhood anemia and infections, growth, development, and developmental programming of adult diseases.

Childhood Anemia and Infections

We identified very few studies of nutritional supplementation during pregnancy that followed the children longitudinally for sufficient duration to report on child health outcomes, such as childhood anemia and infections. For example, of the twenty studies included in a Cochrane systematic review of iron supplementation during pregnancy (9), only one reported on infant hemoglobin and ferritin levels at three months of age—maternal iron supplementation was associated with higher serum ferritin level in the infant (31). Similarly, of the twenty studies on iron supplementation (9), the seven trials on zinc supplementation (31), and the five trials on vitamin A supplementation (1) during pregnancy, only one trial reported on childhood infections and it found no difference between selective versus routine iron supplementation in pregnancy (32).

Child Growth

We identified very few studies of nutritional supplementation during pregnancy that reported infant and child growth as an

outcome. Schmidt (33) showed that vitamin A supplementation in conjunction with iron supplementation of pregnant Indonesian women did not improve growth of their infants during the first year of life. A study in Guatemala (34) found that prenatal supplementation with Atole, a dietary supplement providing protein, micronutrients, and 3.80 MJ (900 kcal/L), increased the length of three-year-old children by 2.5 cm and reduced prevalence of severe stunting by half.

Child Development

Similarly, we identified very few studies of nutritional supplementation during pregnancy that reported infant and child development as an outcome. In the Bacon Chow Study (35), women who received high protein and energy supplementation during pregnancy and lactation had infants with significantly higher motor but not mental scores at eight months of age, compared to controls. Schmidt (36) did not find an impact of weekly supplementation of 4,800 RE vitamin A in addition to iron during gestation on functional development of Indonesian infants. Tamura et al. (37) also did not find a benefit of zinc supplementation among women during the latter half of pregnancy on the neurological development of their children at five years of age. A recent randomized, placebo-controlled, double-blinded trial (38) found that children born to mothers who had taken cod liver oil during pregnancy and lactation scored higher on the Mental Processing Composite of the Kaufman Assessment Battery for Children (K-ABC) at four years of age, compared with children whose mothers had taken corn oil.

Developmental Programming of Adult Diseases

Because trials of nutrition supplementation have not typically followed the infants into adulthood, the impact of prenatal nutritional supplementation on adult health and disease is not known. Conlisk (34) recently reported results from a nutritional supplementation study during pregnancy and early childhood, conducted from 1969 to 1977 in Guatemala. Among adult female offspring of mothers who received Atole, prenatal supplementation was inversely associated with fasting plasma glucose, consistent with the notion of gestational programming.

Summary

Presently, there are too few trials that have longitudinally followed children for a sufficient duration to evaluate the effects of prenatal nutritional supplementation on child health and development or developmental programming of adult diseases.



VI. THE CASE FOR PRE- AND PERICONCEPTION NUTRITIONAL SUPPLEMENTATION TRIALS

An important criticism of intervention trials on nutritional supplementation during pregnancy is that they may do too little too late. Other than the trials on periconceptional folate supplementation for the prevention of neural tube defects, most prenatal nutritional interventions began in mid-pregnancy—largely due to the logistical difficulties of recruiting mothers into the study in early pregnancy (before initiation of prenatal care). A growing body of evidence now suggests that complications that become apparent relatively late in pregnancy may actually have their origins much earlier in pregnancy. For example, a recent study showed that modest undernutrition commencing before conception and continuing for only 30 days thereafter induces premature delivery in the sheep model. The undernourished ewes had higher adrenocorticotropic hormone (ACTH) levels throughout gestation, with a precocious rise in cortisol in half of them. This raises the possibility that the maternal/fetal clock can be reset around the time of conception by undernutrition, resulting in accelerated maturation of the fetal HPA axis leading to preterm birth. Similarly, the pathogenesis of preeclampsia or fetal growth restriction has now been traced to abnormal trophoblastic invasion during implantation. Implantation is thought to be regulated, in part, by immunologic mechanisms, which in turn may be modulated by macro- and micronutrients. Thus, there may be a critical window of opportunity in early pregnancy to prevent preeclampsia or fetal growth restriction via nutritional immune modulation of the implantation process. By the time placentation is complete, however, there may be little that any added nutrients can do to significantly alter the course or outcome of the disease processes. Furthermore, maternal nutritional status before pregnancy may strongly influence a mother's vulnerability to pregnancy complications. She may be more susceptible to infections and less able to clear the infections that could lead to preterm delivery. She may have less nutritional "reserves" to withstand the stress of prenatal undernutrition. The epidemiological association between a short interpregnancy interval and increased risk for SGA and preterm birth in a subsequent pregnancy has commonly been attributed to the effects of nutritional depletion. Similarly, the association between low prepregnancy BMI and increased risk for SGA and preterm birth has been explained on the basis of increased biological vulnerability from poor prepregnancy nutritional status. Thus, it may be premature to conclude from extant trials that maternal nutritional supplementation does not work. Future studies should be designed to take into account the importance of preconceptional and periconceptional nutrition.

This presents a design challenge, as women need to be enrolled into the study before conception, but it is a challenge that can be overcome.

VII. EVALUATION OF WOMEN, INFANTS, AND CHILDREN (WIC) PROGRAMS

In this section, we evaluate the evidence of the effectiveness of WIC for preventing two of the leading causes of infant mortality in the U.S.—low birthweight and preterm birth.

A. BACKGROUND

WIC was created in 1972 as a two-year pilot program in response to the 1969 White House Conference on Food, Nutrition, and Health (39, 40). The conference report concluded that "substantial numbers of pregnant, postpartum, and breastfeeding women, infants, and young children from families with inadequate income (were) at special risk with respect to their physical and mental health by reason of inadequate nutrition or health care or both." WIC seeks to improve the diets and, therefore, the health of low-income pregnant, breastfeeding, and postpartum women and their infants and children (up to age five). The program was permanently authorized in 1974 under the Special Supplemental Food Program for Women, Infants, and Children (WIC) ("Food" was later changed to "Nutrition"). Funding has increased from \$10.4 million in 1974 to \$5.16 billion in 2005; total participation has increased from 88,000 in 1974 to over 8 million women and children in 2005 (40).

Although WIC is a program of the U.S. Department of Agriculture (USDA), it is administered mostly through state health departments. It provides a supplemental "food package," nutrition and health counseling, and referral for other services to low-income women and children at nutritional risk. Income eligibility is set at family incomes up to 185 percent of the federal poverty level; recipients of Temporary Assistance for Needy Families (TANF), food stamps, and Medicaid benefits are automatically deemed eligible. Pregnant women comprise about 11 percent of WIC participants. In recent years, about 900,000 pregnant women, or nearly one in four pregnant women in the U.S., participated in WIC annually.

In 1997, 69 percent of WIC-eligible pregnant women participated in WIC. The "food package" for pregnant women, worth \$38 per person per month in 1997, included milk, eggs, iron-fortified dry cereal, juice rich in vitamin C, and dry beans or peanut butter. These foods were specifically selected as sources of protein, iron, calcium, and vitamins A and C, all of which are likely to



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TABLE 3.6: PRENATAL WIC PARTICIPATION BENEFICIAL? CONCLUSIONS OF 19 EVALUATION STUDIES

Study	Year	Place	LBW	VLBW	Preterm Delivery	Fetal/ Neonatal Mortality	Maternal Nutrition	Medical Care Costs
Kennedy	73-78	MA	✓					
Kennedy	73-78	MA	✓			✓		
Kotelchuck	84	MA	✓	✓	(✓)	✓		
Metcoff	80-82	OK	X ^a				(✓)	
Rush (Long)	83-84	USA	X ^a	X ^a	(✓)	(✓)	(✓)	
Rush (Hist)	83-84	USA	(✓)		✓	✓		
Stockbauer	79-81	MO	✓		✓	X ^a		
Stockbauer	82	MO	✓	✓	✓	(✓)		
Schramm	80-81	MO	✓	(✓)				✓
Schramm	82	MO	✓			(✓)		✓
Caan	81	CA	✓				✓	
Devaney	87	FL	✓		✓			✓
Devaney	87	MN	✓		✓			✓
Devaney	87	NC	✓		✓			✓
Devaney	87	SC	✓		✓			✓
Devaney	88	TX	✓		✓			✓
Buescher	88	NC	✓	✓		(✓)		✓
New York	88	NY	✓	✓	✓			✓
GAO	Meta-analysis of previous studies		✓					✓

NOTE: ✓ Statistically significant improvement. (✓) Improved, but not statistically significant. X No improvement. X^a No improvement overall, although improvement noted in subgroups.

Source: B. Abrams, "Preventing low birth weight: does WIC work? A review of evaluations of the special supplemental food program for women, infants, and children," *Ann N Y Acad Sci.* 678 (Mar. 15, 1993): 306-16.

be low in the diets of low-income women. In 2005, the Institute of Medicine (41), under contract with the Food and Nutrition Service of the U.S. Department of Agriculture, issued a report recommending changes to the WIC “food package,” adding \$10 per month for fruits and vegetables for pregnant women, as well as yogurt, soymilk, and tofu as additional high-calcium options. In order to be cost-neutral, the Committee recommended reductions in the amounts of juice, milk, cheese, eggs, and, in certain cases, infant formula provided through the WIC “food package.”

WIC’s popularity and growth stem from the widespread belief that research studies have proven that WIC works. “WIC works, perhaps better than any other government program in existence,” Secretary of Agriculture Dan Glickman declared. Former Secretary of Health and Human Services Louis Sullivan made a similar claim: “The WIC Program results in significant Medicaid savings that far outweigh the program’s costs by a ratio of three to one... That is clearly an overwhelming return on a small national investment.” The following section reviews the evidence of the effectiveness of WIC.

B. REVIEW OF PRENATAL WIC EVALUATIONS

The effectiveness of WIC for preventing low birthweight and preterm birth was evaluated in a systematic review of the literature by Abrams (39), who summarized nineteen published WIC evaluations in **Table 3.6**. The studies of greatest importance and relevance are highlighted here.

The first three studies were conducted in Massachusetts shortly after the inception of WIC. They provided some evidence that WIC participation was associated with reduced incidence of LBW (and possibly VLBW), preterm birth, and perhaps neonatal mortality as well.

Metcoff et al. published the only randomized controlled trial of prenatal WIC participation in 1985. WIC participation was associated with a statistically insignificant increase in LBW (8.7 percent versus 6.9 percent in the non-WIC group). Statistically significant improvement in birthweight was observed among cigarette smokers who participated in WIC, and nonsignificant improvement in birthweight was also noted for black women and those who had previously delivered a LBW infant.



Rush conducted a comprehensive National WIC Evaluation using two different study designs. The Historical Study compared use of health services and perinatal outcomes before and after implementation of the WIC program. The study indicated that WIC participation was associated with earlier and more adequate prenatal care; a longer duration of gestation and a lower rate of preterm birth among less educated women; and reduced fetal death. The Longitudinal Study compared pregnancy outcomes of WIC participants with those of controls who were eligible for WIC, but who received prenatal care in settings where the WIC program was not available. A comparison of WIC participants and controls found the control population to be more affluent, and these differences were adjusted in post-hoc analyses. While there was no overall improvement in LBW or preterm birth associated with WIC participation, a significant reduction in LBW and VLBW within WIC programs that were judged to be of “superior quality” by State WIC Directors, as well as a significant reduction in preterm birth among women with a previous LBW delivery, were observed.

Devaney et al. published a series of WIC evaluations in five states, using linked WIC files, vital statistics files, and Medicaid files. The entire sample was restricted to Medicaid beneficiaries, the eligibility for which was extremely stringent at the time, thereby reducing sociodemographic differences between WIC participants and the comparison group. WIC participation was associated with significant improvement in the adequacy of prenatal care utilization and significant reduction in LBW and preterm birth. Using the prorated cost estimates, Devaney et al. estimated that for every dollar spent on WIC, between \$2.84 and 3.90 was saved in newborn costs alone.

Using linked WIC, birth certificate, and Medicaid records, Buescher et al. reported that WIC non-participation was associated with a significant 115 percent increased risk of VLBW and a 45 percent increased risk of LBW, controlling for race, parity, prenatal care, marital status, education, age, previous pregnancy loss, and smoking. Buescher et al. estimated that for every dollar spent on WIC, \$1.92 was saved on newborn care for white infants and \$3.75 was saved on that for black infants. Similarly, using state-linked WIC-birth certificate data files, the New York Bureau of Nutrition reported a significant 185 percent increased risk of VLBW, an 80 percent increased risk of LBW, and a 66 percent increased risk of preterm delivery associated with WIC non-participation, after controlling for race, parity, prenatal care, marital status, education, age, and smoking. Savings in neonatal costs during the first hospital stay were estimated at \$2.35 for every dollar spent on WIC.

In 1992, the U.S. General Accounting Office conducted a meta-analysis of seventeen selected studies of the effectiveness of WIC and concluded that WIC participation reduced the incidence of LBW by 25 percent and VLBW by 44 percent. They estimated that every federal dollar invested in WIC returned \$3.50 to federal, state, and local governments and private payers over the first 18 years of life; the estimated return in the first year was \$2.89.

The findings from this systematic literature review led Abrams to conclude that WIC does, in fact, work:

Prenatal WIC participation is associated with a reduction in the incidence of low birthweight, very low birthweight, and preterm delivery, especially among women at high risk because of sociodemographic characteristics or medical conditions. Furthermore, because the costs of low birthweight are so high, providing WIC to pregnant women actually saves money by preventing poor pregnancy outcome instead of treating it. There is evidence that the earlier in pregnancy and longer a woman receives WIC benefits, the greater the positive effects.

These conclusions were consistent with those of other critical reviews of WIC evaluations, which also suggested that WIC was associated with improved pregnancy outcomes.

C. CRITIQUE OF WIC EVALUATIONS

More recently, Besharov and Germanis (42) have begun to challenge the validity of previous evaluations of WIC, citing significant methodological limitations in these studies. The three most significant weaknesses, according to Besharov and Germanis, were 1) selection bias, 2) simultaneity bias, and 3) lack of generalizability.

1. Selection Bias

Selection bias could overestimate the effect of WIC participation on birth outcomes if WIC participants possess personal characteristics, behaviors, or motivation that increase their likelihood for favorable birth outcomes compared to controls. Scientifically, the best way to deal with potential selection bias is a randomized controlled trial in which individuals eligible for WIC are randomly assigned to a treatment group (that receives WIC) and a control group (that does not). Ethical and political considerations make such a trial highly unlikely. Thus, researchers deal with the potential problem of selection bias by using multivariate regression analysis, which examines the effect of WIC participation on birth outcomes while controlling for group differences



in these other factors. But the question remains about which factors to control for. Clearly race, parity, prenatal care, marital status, education, age, and smoking are such factors, but could other unmeasured factors account for the observed differences in birth outcomes between WIC and non-WIC participants? Citing studies by Gordon and Nelson (43) and Brien and Swann (44), Besharov and Germanis concluded that WIC's estimated positive birthweight effects disappeared for the population as a whole when attempts were made to control for selection bias, and that failure to control for selection bias overstates WIC's impact on birthweight and probably other related outcomes. They did acknowledge that Brien and Swann's findings also suggested that, in particular subgroups (e.g., black women), statistical adjustments for potential selection bias actually increased estimated birthweight impacts of WIC participation. We agree with Rossi (45) that "it appears likely that estimates uncorrected for selection biases overestimate WIC effectiveness but the degree of overestimation cannot be reliably determined." Given that it is presently impossible to determine the size of overestimation due to selection bias, we conclude that current available evidence neither supports nor refutes the effectiveness of WIC for preventing LBW and other related outcomes.

2. Simultaneity Bias

The longer a woman is pregnant, the more likely it is that her baby will be healthy. At the same time, however, the longer she is pregnant, the more likely it is that she will enroll in WIC because she has more opportunities to learn about the program and more time to enroll. Thus, women who enroll in WIC late in their pregnancy most likely have favorable birth outcomes not because of their participation in WIC (the program has too little time to have substantial impact), but because of the length of their pregnancy. Failure to account for these simultaneous effects may exaggerate WIC's estimated impact. Besharov and Germanis (42) cited studies by Devaney et al. and Gordon and Nelson (43), which observed substantial reductions in WIC's positive birthweight effects when attempts were made to control for simultaneity bias (e.g., by including gestational age as an independent variable, and by excluding women with late entry into WIC). Because different approaches yielded different estimates, it is presently impossible to determine the size of overestimation due to simultaneity bias; thus, available evidence does not allow us to establish or refute the effectiveness of WIC for preventing LBW and other related outcomes.

3. Generalizability

Another limitation of the body of research on the effectiveness of WIC is its generalizability (42). Most evaluations examined WIC in only one state or in a few states; many of the prenatal findings were restricted to those mothers receiving Medicaid (which may represent the most disadvantaged families in WIC); and most evaluations took place over a decade ago, with a different "food package" and different size, composition, and characteristics of the WIC population—all of which could have limited the generalizability of earlier research.

SUMMARY

The evidence of effectiveness of any single macro- or micronutrient supplement for preventing fetal growth restriction, preterm birth, birth defects, and maternal complications (preeclampsia, anemia, and infections) is far from conclusive, with the possible exceptions of periconceptional folic acid supplementation for prevention of neural tube defects and iron and folate supplementation for prevention of maternal anemia. Fish oil for prevention of recurrent preterm birth, balanced protein-energy supplementation for prevention of fetal growth restriction, and calcium supplementation for prevention of preeclampsia in high-risk women also appear promising. No other maternal supplementation interventions have been shown to be effective. However, it is premature to conclude that maternal nutritional interventions do not work, as most trials have focused on supplementing one single nutrient during pregnancy; few studies have examined the impact of multinutrient supplementation that starts before pregnancy. Most evaluations of WIC programs have found evidence of effectiveness for preventing LBW; however, their effectiveness may be overstated due to problems of selection bias, simultaneity bias, and lack of generalizability.



CHAPTER 4

RETHINKING NUTRITION AND INFANT MORTALITY: THE CONTEXT OF RELATIONALITY OVER THE LIFE COURSE

Chapter 1 examined the relationship between nutrition and infant mortality. We found evidence linking poor maternal nutrition to several leading causes of infant mortality (although causality cannot be conclusively established). We also discussed how poor maternal nutrition may contribute to poor child health and development, as well as developmental programming of adult diseases.

Chapter 2 examined the nutritional status of pregnant women in the United States. We found that most pregnant women in the U.S. started off pregnancy overweight or underweight and had inappropriate weight gain during pregnancy. Approximately one of every three low-income women is anemic in the third trimester of pregnancy. Most pregnant women consume a diet high in refined carbohydrates and trans fats; in general, white women consume a higher nutrient-dense diet, and black women consume more calories but fewer nutrients after energy adjustment. We identified multiple micronutrient deficiencies in the dietary intake of pregnant women. Fasting, pica, and fast-food consumption are common, particularly among pregnant African American women.

If poor maternal nutrition is directly or indirectly related to several leading causes of infant mortality, and if the nutritional status of many pregnant women in the U.S. is poor in many respects, it follows that improving maternal nutritional status should be a key strategy for reducing infant mortality in the United States. Chapter 3 reviewed evidence of the effectiveness of nutritional supplementation programs for preventing several leading causes of infant mortality. The evidence of effectiveness of any single macro- or micronutrient supplement for preventing fetal growth restriction, preterm birth, birth defects, and maternal complications (preeclampsia, anemia, and infections) is far from conclusive, with the possible exceptions of periconceptional folic acid supplementation for prevention of neural tube defects and iron and folate supplementation for prevention of maternal anemia. Fish oil for prevention of recurrent preterm birth, balanced protein-energy supplementation for prevention of fetal growth restriction, and calcium supplementation for prevention of preeclampsia in high-risk women also appear promising. No other maternal supplementation interventions have been shown to be effective. Most evaluations of WIC programs have found evidence of effectiveness for preventing LBW; however, their effectiveness

may be overstated due to problems of selection bias, simultaneity bias, and lack of generalizability.

Thus, our review suggests that the evidence of the effectiveness of nutritional interventions during pregnancy is mixed at best. A few macro- or micronutrient supplements have been shown to be effective or promising, but for the most part, maternal supplementation with any single macro- or micronutrient during pregnancy does not work. The evidence of the effectiveness of WIC is far from conclusive, given serious concerns about the validity of evidence of its effectiveness. So why haven't these maternal nutritional intervention programs been proven to be effective, and what can be done to improve their effectiveness? This chapter begins to explore these questions from a different perspective.

We re-examine the problems of infant mortality and maternal nutrition within a context of relationality. We contend that both are, in essence, problems of broken relationships at many levels. These broken relationships create lifelong conditions of high stress and low support, which in turn pattern physiological, psychological, and behavioral responses that put the mother at nutritional risk during pregnancy and her baby at risk for fetal and infant death. African American families are disproportionately affected by these broken relationships, which contribute to the disparities in maternal nutrition and infant mortality. From this perspective, it is perhaps not surprising that most nutritional supplementation interventions have not been proven effective for preventing infant mortality. Similar to the findings of the *Courage to Love: Infant Mortality Commission*, we argue that efforts to improve maternal nutrition must also focus on the repair and support of interpersonal relationships at all levels, and they must do so not only during pregnancy, but across the life course of the woman and her family. That is, in this chapter, we reframe the problem of poor maternal nutrition and imagine possibilities for new actions to improve maternal nutrition and prevent infant mortality in the context of relationality over the life course.

I. INFANT MORTALITY IN THE CONTEXT OF RELATIONALITY

Much like Byllye Avery's assertion that black infants are dying because their mothers are dying in dead relationships (1), we argue that the problem of infant mortality in the U.S. is in essence a problem of broken relationships at many levels—at home, in our neighborhoods and communities, and in our society at large. It is beyond the scope of this paper to review the many broken relationships in our society that contribute to infant mortality; we refer interested readers to Ronald David's framing paper for the *Courage to Love: Infant Mortality Commission*, entitled *The Case*



for *Relationality: The Historical Framework of Policy and Practice on Infant Mortality*, for an in-depth re-examination of infant mortality with an “intentional focus on relationality” (1). In this section, we give a few examples of the associations between broken relationships and infant mortality and discuss possible mechanisms mediating these associations.

A. MARITAL RELATIONSHIP AND INFANT MORTALITY

Infants born to single mothers are at greater risk for infant death than those born to married mothers (2). In 2002, infants of married mothers had a mortality rate of 5.4 per 1,000 live births. The mortality rate for infants of unmarried mothers was 9.9, more than 83 percent higher than the rate for infants of married mothers (2). Across all racial-ethnic groups, marital status offers protection against infant mortality, as shown in **Figure 4.1**. There are important racial-ethnic differences in the proportion of infants born to unmarried mothers (**Figure 4.2**). In 2002, approximately one of every three infants in the U.S. was born to an unmarried mother. However, nearly 70 percent of all black infants, nearly 60 percent of American Indian infants, and 44 percent of Hispanic infants were born to unmarried mothers, compared to 23 percent of white infants and 15 percent of Asian and Pacific Islander infants. Black mothers were three times as likely as white mothers and more than four times as likely as Asian and Pacific Islander mothers to be unmarried (2).

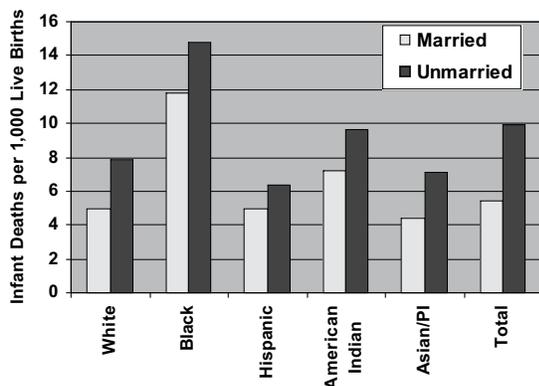
The reasons for the higher rates of infant mortality among infants of unmarried mothers are not known, but have commonly been attributed to these mothers’ relative lack of social support and resources (3, 4). Marital status appears to buffer the impact of stress on birth outcomes by increasing social support. Additionally, a

broken relationship with the baby’s father can be an important source of stress for the mother, and stress, as discussed later in this chapter, is an important risk factor for preterm birth and LBW. Marital status, however, does not always guarantee support and resources. As Bennett (5) observes, the failure of marriage to improve outcomes for teenagers could be an indication that early marriage is less helpful for a pregnant teen than her own family’s support. And not all relationships outside of marriage are broken. In the *Fragile Families and Child Wellbeing* study, four out of five unmarried black couples were romantically involved at the time their children were born (6). Eighty-one percent of the black mothers in the study indicated that the father provided financial help during the pregnancy, and three-fourths reported that the father came to visit her and the infant in the hospital. Nearly all unmarried fathers interviewed reported that they wanted to stay involved in raising their children in the coming years (6). However, these unmarried families are “fragile” because of the multiple risk factors associated with non-marital childbearing and are at risk for disengagement. At one year after a non-marital birth, only a small proportion (six percent) of these relationships resulted in marriage, and one-third of the couples had separated (7). The fragility and instability of these relationships can also be an important source of stress. Several studies have found that infants born to unmarried mothers in co-habiting relationships are at lower risk for infant death than those born to single mothers, but are still at higher risk than those born to married mothers (3, 8-9).

B. SOCIAL CAPITAL AND INFANT MORTALITY

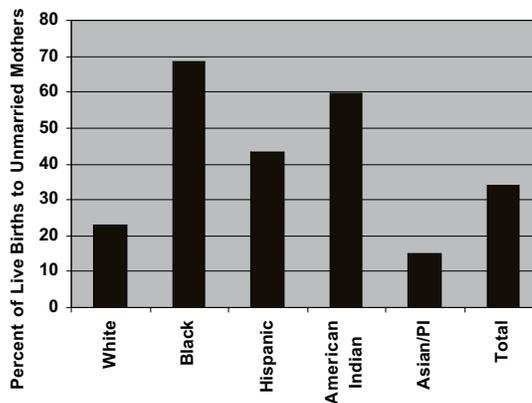
Social capital describes the connectedness among members of a neighborhood or community. It was first proposed by Putnam (10) to refer to those features of social organization, such as networks, norms, and social trust, that facilitate coordination and

FIGURE 4.1: MARITAL STATUS AND INFANT MORTALITY, BY RACE-ETHNICITY, 2002



Source: T. J. Mathews, F. Menacker, and M. F. MacDorman, Centers for Disease Control and Prevention, National Center for Health Statistics, “Infant mortality statistics from the 2002 period: linked birth/infant death data set,” *Natl Vital Stat Rep.* 53, no. 10 (Nov. 24, 2004): 1-29.

FIGURE 4.2: PERCENT OF LIVE BIRTHS TO UNMARRIED MOTHERS, BY RACE-ETHNICITY, 2002



Source: T. J. Mathews, F. Menacker, and M. F. MacDorman, Centers for Disease Control and Prevention, National Center for Health Statistics, “Infant mortality statistics from the 2002 period: linked birth/infant death data set,” *Natl Vital Stat Rep.* 53, no. 10 (Nov. 24, 2004): 1-29.



cooperation for mutual benefit (11). Social capital is characterized by 1) the existence of community networks, 2) civic engagement, 3) local identity and a sense of solidarity and equity with other community members, and 4) trust and reciprocal help and support. The notion of social capital has been applied in various disciplines, from criminology to political science to public health. For example, Sampson et al. found lower levels of social cohesion in neighborhoods with higher homicide rates. Looking at state-level data, Kawachi et al. (11) found that higher levels of civic participation in voluntary organizations were associated with lower age-adjusted all-cause mortality. Additionally, they also found that lower levels of social trust were associated with higher rates of most causes of deaths, including coronary heart disease, malignant neoplasms, cerebrovascular diseases, and unintentional injury. In regression models, variations in the levels of social trust explained 58 percent of the variance in total mortality across states, including infant mortality. Recently, Buka et al. (12) published a multilevel study of neighborhood social capital and birth outcomes. Using a measure conceptually related to social capital, they found a positive association between “neighborhood support” and infant birthweight for white mothers but not black mothers in Chicago neighborhoods.

The inverse relationship between social capital and mortality, including infant mortality, remains largely unexplained. Why would neighborhoods and communities with high levels of social cohesion, civic participation, and social trust have lower infant mortality rates? One possible explanation is that social capital does at the neighborhood level what social support does at the interpersonal level—social capital buffers the impact of stress on birth outcomes. Lack of child care or transportation to prenatal care appointments can be stressful; the ability to count on one’s neighbors for child care or a ride in a pinch buffers the impact of that stress. Food insecurity or housing instability can be stressful; the ability to turn to local food banks, shelters, churches, synagogues, and other community-based or faith-based organizations for food and shelter buffers the impact of that stress. Changing several buses to get to a prenatal care appointment when one is seven months pregnant can be stressful; encountering strangers who offer their seats when one gets on the bus buffers the impact of that stress. A neighborhood or community with low social capital is therefore one with many broken relationships—between neighbors and among strangers—and these broken relationships create a chronic condition of high stress and low support, which can adversely affect the health of the population in general, and infant survival in particular.

C. RACISM AND INFANT MORTALITY

Racism is perhaps most emblematic of the many broken relationships in our society. A small but growing number of studies suggest an association between racial discrimination and low birthweight. Collins et al. (13) found in a small, hospital-based, case-control study that African American women who were exposed to racial discrimination during pregnancy in five domains—“at school,” “getting medical care,” “getting service at a restaurant or store,” “getting housing,” and “at work”—were more than three times as likely to give birth to a VLBW infant, compared to those who did not. Several subsequent studies have confirmed the association between racial discrimination and LBW (14-16).

The few studies on racism and birth outcomes have focused primarily on what Camara Jones (17) refers to as personally mediated racism. This is what most people think of when they hear the word “racism”—it can manifest as lack of respect, suspicion, devaluation, scapegoating, and dehumanization of others based on their race. There is another level of racism that may be even more pervasive and pernicious. Institutionalized racism (17) is defined as differential access to the goods, services, and opportunities of society by race. It can manifest as differential access to quality education, sound housing, gainful employment, appropriate medical facilities, and a clean environment. Residential segregation is a good example of institutionalized racism; Laveist (18) and Polednak (19) found greater black-white disparities in infant mortality in cities that are more racially segregated.

In sum, infants born to mothers who are unmarried, reside in a neighborhood with low social capital, or experience racial discrimination during pregnancy are at greater risk of infant death. The next section examines how these broken relationships at home, in the community, and in our society at large contribute to infant mortality.

D. STRESS AND INFANT MORTALITY

Our main thesis is that broken relationships at many levels create conditions of high stress and low support, which in turn pattern physiological, psychological, and behavioral responses that put the mother at nutritional risk during pregnancy and her baby at risk for fetal and infant death. Here we briefly review the mechanisms by which stress can cause preterm birth, which is a leading cause of infant mortality and the leading cause of racial-ethnic disparities in preterm birth.



Stress is increasingly becoming recognized as an important risk factor for preterm birth (20-23). Stress may be simply defined as any challenge that threatens or is perceived to threaten homeostasis (i.e., the stability of the internal milieu of the organism) (24-25). Stress can be physiological (e.g., infectious, nutritional) or psychological; in this chapter, we focus on maternal psychological stress. Recent research has identified at least four major pathways to spontaneous preterm birth: 1) premature activation of the maternal or fetal hypothalamic-pituitary-adrenal (HPA) axis, 2) decidual-chorioamniotic or systemic inflammation, 3) problems with placental blood flow, and 4) uterine over-distension (49). Stress can activate one or more of these pathways leading to preterm birth (26).

Both term and preterm labor are under endocrine control (27). Placental corticotropin-releasing hormone (CRH) plays a central role in stimulating the onset of labor (27). In term labor, fetal maturation results in greater output of cortisol and dehydroepiandrosterone sulfate (DHEA-S) from the fetal hypothalamic-pituitary-adrenal (HPA) axis, which activates placental production of CRH. Placental CRH, in turn, further stimulates the fetal HPA axis in a positive feedback loop, thereby amplifying the process of labor (Figure 4.3a). That is, term labor begins with placental CRH activation as a result of fetal maturation; the signal for the onset of labor comes from the fetal HPA axis (27).

In preterm labor, it may be the maternal HPA axis, and not the fetal HPA axis, that is prematurely driving placental CRH production (Figure 4.3b) (28, 29). Stress activates maternal HPA axis (“fight or flight” response), resulting in greater output of cortisol, norepinephrine, and other adrenalins, which in turn activate placental production of CRH. Placental CRH then drives the fetal HPA axis in a positive feedback loop, thereby initiating

the process of labor (28, 29). *In vitro* studies of human placental cells have shown that CRH is released from cultured human placental cells in a dose-response manner in response to all the major biological effectors of stress, including cortisol, catecholamines, oxytocin, angiotensin II, and interleukin-1 (30). *In vivo* studies have also found significant correlations between maternal psychosocial stress and maternal plasma levels of CRH, ACTH, and cortisol (31-35). Several studies have related early maternal plasma CRH (which is mostly placental in origin) to the timing of birth (31-34). Hobel and colleagues (34) conducted serial assessments of CRH over the course of gestation and found that, compared with women delivering at term, women delivering preterm had significantly elevated CRH levels as well as a significantly accelerated rate of CRH increase over the course of their gestation. In addition, they found that maternal psychosocial stress levels at mid-gestation significantly predicted the magnitude of increase in maternal CRH levels between mid- and later-gestation. Thus, preterm labor begins with premature placental CRH activation; the signal for the onset of labor can come from the maternal HPA axis in response to stress.

Stress can also alter neuroendocrine modulation of immune functions, leading to increased susceptibility to intra-amniotic infection or inflammation (reviewed in ref 36). Infection or inflammation is responsible for the majority of cases of early preterm birth (less than 32 weeks’ gestation). Chronic stress can depress immune functions, mediated largely through increased glucocorticoids. Recent research has demonstrated an association between chronic stress and greater risk for bacterial vaginosis during pregnancy (37, 38). Moreover, chronic stress can also cause chronic inflammation and inflammatory dysregulation (through mechanisms described later in the chapter), which leads to greater susceptibility to preterm labor caused by inflammation.

FIGURE 4.3A: ENDOCRINE MECHANISMS OF LABOR

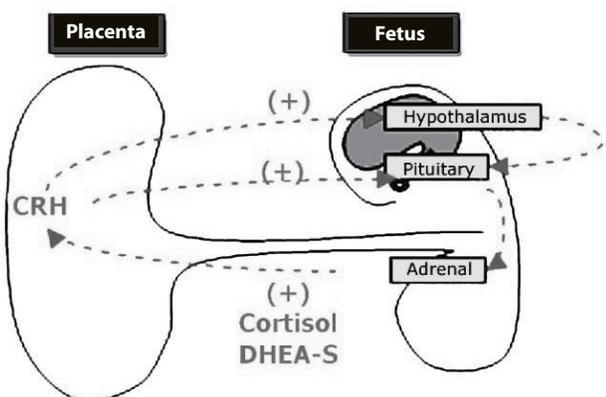
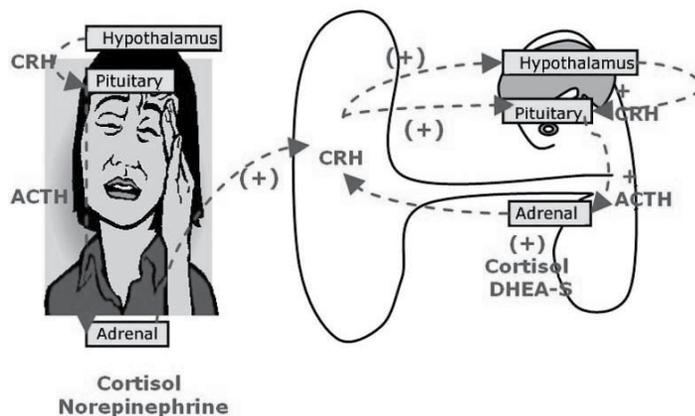


FIGURE 4.3B: MATERNAL STRESS CAN PREMATURELY ACTIVATE ENDOCRINE MECHANISMS OF LABOR





Stress can also alter placental blood flow. Several studies have reported a significant association between high levels of maternal anxiety and blood flow in the placenta (as demonstrated by Doppler studies), although few studies have linked stress vascular response to birth outcomes (reviewed in ref 36). McCubbin and colleagues (39) reported a significant association between vascular reactivity to a standardized psychological challenge and infant birthweight and length of gestation. After adjusting for maternal age, race, baseline blood pressures, the trimester of stress testing, and expired carbon monoxide, women with larger diastolic blood pressure responses during stress had infants with lower birthweights and decreased gestational age. Thus, there is some preliminary evidence linking maternal stress to increased diastolic blood pressure and decreased placental blood flow, but further studies are needed to clarify the relationship between maternal stress, placental blood flow, and preterm birth.

In sum, stress is an important risk factor for preterm birth. Maternal stress can prematurely activate placental CRH production, thereby precipitating the biological cascade leading to preterm birth. Stress can also alter neuroendocrine modulation of the immune system, leading to greater susceptibility for intra-amniotic or systemic infection or inflammation. Stress may compromise placental blood flow, leading to preterm birth and fetal growth restriction, although further studies are needed to clarify these pathways.

E. A LIFE-COURSE PERSPECTIVE ON THE RELATIONAL CONTEXT OF INFANT MORTALITY

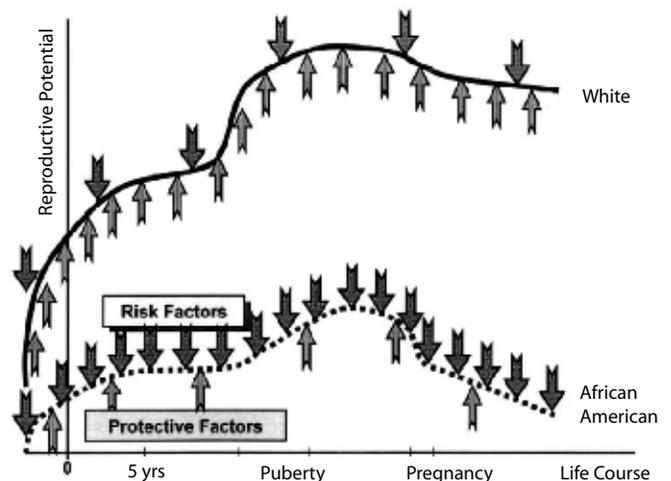
An emerging paradigm in maternal and child health is the life-course perspective. Increasingly, birth outcomes are viewed as the end product of not only the nine months of pregnancy, but the entire life course of the woman (as well as her family and her partner). Recently Lu and Halfon (40) applied this life-course perspective to re-examine racial-ethnic disparities in birth outcomes. Disparities, they argued, are the consequences of differential exposures to risk factors (e.g., chronic stress) and protective factors (e.g., social support) not only during pregnancy, but across the entire life span (Figure 4.4). In this section, we re-examine the relational context of infant mortality from a life-course perspective.

We posit that infant mortality is, in essence, a problem of broken relationships and that these broken relationships create chronic conditions of high stress and low support, which disproportionately affect African American women. We are not simply referring to broken relationships during pregnancy; we are referring

to broken relationships over the entire life course. In a relational context, each one of the downward arrows in Figure 4.4 represents a broken relationship; the more broken relationships there are (particularly during critical periods of development), the worse the birth outcome. In contrast, each upward arrow represents a positive relationship. We believe this is where resilience comes from—the cumulative lift of these positive relationships. The more upward arrows, the stronger the resilience. That is, much of what we call resilience (e.g., self-esteem or self-efficacy, human capital, social support, and spirituality) is determined by the strength of not only current relationships, but also that of past relationships. It is a belief that we can count on our family, friends, neighbors, and God (or a higher Being or Beings) to come through for us in good times and in bad times, a belief that is shaped by our past experiences.

A pregnant woman’s current relationships may be shaped by her past relationships, including that with her father (as well as that between her father and mother) as she was growing up. Anecdotally, it has been observed that some teenage girls “look for love in all the wrong places” because they did not have the stable, secured presence of a loving father or father figure. Father absence may, in part, account for higher rates of teen pregnancies and STDs in some black and Hispanic communities. Similarly, fathers can be a vital source of support for the mother and resilience for the child. Studies have shown that fathers’ involvement in their children’s lives is much influenced by their own fathers’ involvement in their lives growing up. Men whose fathers were involved in raising them have been found to be more involved with their own children, to take more responsibility for them, to show more warmth,

FIGURE 4.4: A LIFE-COURSE PERSPECTIVE ON BLACK-WHITE DISPARITIES IN BIRTH OUTCOMES (Upward arrows indicate protective factors while downward arrows indicate risk factors)





and to more closely monitor their behaviors and activities (41, 42). Conversely, men who did not have a positive fathering model are less involved with their children. One study found that fathers who had experienced a stressful rearing environment spent less time living with their first-born child (43). In a qualitative study, young black fathers cited problems with their self-concept and self-esteem as a barrier to adequate father involvement, generally stemming from their own experiences of abusive and neglectful parenting and the reinforcement of the notion that they are “no good” (44). Thus, past father-daughter or father-son relationships may shape current marital/non-marital relationships, as well as parent-child relationships. Broken relationships in one generation may contribute to broken relationships in the next generation.

Several researchers have also examined the impact on birth outcomes of experiences of racial discrimination during pregnancy and across the life course. In a prospective cohort study (16), participants completed a discrimination questionnaire that asked them whether they had “ever experienced discrimination, been prevented from doing something or been hassled or made to feel inferior... because of their race or color” in any of seven domains: “at school, getting a job, at work, getting housing, getting medical care, on the street or in a public setting, and from the police or in the courts.” Among black women, 50 percent of those with preterm deliveries and 61 percent of those with LBW infants reported having experienced racial discrimination in at least three situations. The unadjusted odds ratio for preterm delivery among black versus white women was 2.54, but this value decreased to 1.88 after adjustment for experiences of lifetime racial discrimination, suggesting that racial discrimination may play a significant role in racial-ethnic disparities in preterm birth. This study asked about lifetime exposure, rather than pregnancy exposure, to racial discrimination. Another study that asked about lifetime exposure similarly found that the adjusted odds ratio of VLBW infants for maternal lifetime exposure to interpersonal racism in three or more domains equaled 2.6 (95%CI: 1.2, 5.3) (14).

Earlier in this chapter, the mechanisms by which stress can cause preterm birth were discussed. We now propose that vulnerability to preterm birth can be traced not only to exposure to stress during pregnancy, but more importantly to stress response that has been patterned by lifelong conditions of high stress and low support. Early life experiences, including *in utero* exposure to maternal stress, can program one’s stress response (early programming model). Chronic and repeated stress can also pattern one’s stress response (cumulative pathways model). Each model is briefly described to demonstrate how broken relationships over the life course can program one’s stress response during pregnancy.

1. Early Programming Model

The early programming model suggests that exposures and experiences during particular sensitive developmental periods in early life may encode the functions of organs or systems that become manifest in health and disease later in life. As discussed in Chapter 1, a growing body of research has documented the influences of prenatal factors, including nutrition, on lifelong chances of developing coronary heart disease, diabetes mellitus, and hypertension (45). Others have similarly demonstrated the long reach of childhood risk exposure on the development of adult chronic diseases (46). Systematic differences in experiences and exposures, from conception onward, may thus become embedded in developmental biology and manifested later in life as socioeconomic gradient and racial-ethnic disparities in health.

It has been shown in both animal and human studies that perinatal stress is associated with high HPA reactivity that persists well into adulthood (47-49). This, in turn, may be related to feedback resistance as a result of decreased expression of glucocorticoid receptors in the developing brain (48). In humans, this programming may continue during infancy and early childhood (51, 52). Exposure to stress hormones during sensitive periods of immune maturation in early life may also alter immune function, leading to increased susceptibility to infectious or inflammatory diseases later on in life (53). Hypothetically, maternal stress during pregnancy could prime the neuroendocrine and immune systems of the developing fetus with stress hormones, leading to higher stress reactivity and immune-inflammatory dysregulation that could increase a female offspring’s vulnerability for preterm labor and LBW later in life. Thus, vulnerability to preterm birth and LBW may be traced not only to stress exposure during pregnancy, but to stress reactivity and immune-inflammatory dysregulation programmed during critical periods of HPA and immune development in early life and possibly even *in utero*.

2. Cumulative Pathways Model

Alternatively, the cumulative pathways model highlights how chronic stress and strain create wear and tear on the body’s adaptive systems—what Bruce McEwen refers to as allostatic load. This wear and tear can add up over time to affect health and function (54). Several studies have related health disparities to cumulative differential exposures to damaging physical and social environments at different life stages (55-58). As previously discussed, individuals who are exposed to chronic and repeated stress have been found to have higher HPA reactivity, possibly due to the loss of feedback inhibition via down-regulation of glucocorticoid receptors in the central nervous system (59, 60). Studies have



also demonstrated a loss of counter-regulation of the immune-inflammatory system by the neuroendocrine system in animals and humans who are subjected to repeated or chronic stress, partly as a result of the down-regulation of glucocorticoid receptors in the immune cells (61). Thus, women who are exposed to repeated or chronic stress are at increased risk for preterm delivery caused by infections not only because of their relative immune suppression, but also because of the inflammatory dysregulation that results in persistent, chronic inflammation that cannot be easily turned off.

Evidence supporting the cumulative pathways mechanism comes from research on the weathering hypothesis. Using linked birth-death certificates, Geronimus (62) found increased risk for LBW and VLBW births with increasing age among African American women, particularly among disadvantaged African American women, but not among white women. She also noted a more rapid decline in the health status of African American women with increasing age, compared to that of white women. Geronimus attributed this accelerated decline in health status with increasing age to the chronic stress and strain that African American women, particularly disadvantaged African American women, have to weather on a daily basis. She hypothesized that the effects of social inequality on the health of populations compound with age, leading to growing gaps in health status through young and middle adulthood that can affect fetal health (62).

In sum, the life-course perspective views birth outcomes as the end product of not only the nine months of pregnancy, but the entire life course of the woman (as well as her family and partner). Vulnerability to preterm birth, therefore, can be traced not only to stress exposure during pregnancy, but more importantly to stress response (neuroendocrine, immune-inflammatory, vascular) that has been programmed over the life course. Both early life (including *in utero*) experiences and chronic exposure to stress can result in dysregulation of stress response, leading to greater susceptibility to preterm birth.

F. RETHINKING INFANT MORTALITY IN THE CONTEXT OF RELATIONALITY OVER THE LIFE COURSE

In this section we have argued that infant mortality is, in essence, a problem of broken relationships over the life course. These broken relationships, occurring at multiple levels, create lifelong conditions of high stress and low support, which pattern one's physiological (neuroendocrine, immune-inflammatory, vascular), psychological, and behavioral responses. These responses, in turn, put a baby at greater risk for fetal and infant death. Byllye Avery's assertion that black infants are dying because their mothers are dying in dead relationships rings true. Infant mortality is not a

problem that can be fixed with a silver bullet, such as antibiotics or nutritional supplements. Going back to the recommendation of the Courage to Love: Infant Mortality Commission, "Efforts to reduce maternal and infant mortality and morbidity must focus on the repair and support of interpersonal relationships at all levels." Moreover, these efforts must do so not only during pregnancy, but over the life course of the woman and her family. Later in the chapter we discuss how to rebuild these relationships in our society. First, we turn our attention to re-examining the problem of poor maternal nutrition in the context of relationality over the life course.

II. MATERNAL NUTRITION IN THE CONTEXT OF RELATIONALITY

In our society, maternal nutrition is commonly seen as a matter of individual choice. If the mother's nutrition is poor, then she is making some bad choices. Much of current public health interventions to improve maternal nutrition have focused on health education to help mothers make better choices about nutrition. Alternatively, some have argued that poor nutrition results from lack of access to healthy foods. Provision of food supplements to mothers (e.g., WIC) is intended to increase access to healthy foods.

While we acknowledge that maternal nutrition is a matter of both individual choice and access, in this section we reframe the issue of maternal nutrition in the context of relationality. We argue that poor maternal nutrition results from broken relationships over the life course. These broken relationships are manifested in the lack of support for breastfeeding, the decline in family meals concomitant to the rise of fast food, the marketing of junk foods to children, and the prevalence of food insecurity in a land of plenty. We also discuss how chronic stress makes us reach out for "comfort foods." Poor maternal nutrition does not start with pregnancy; it usually follows a lifelong pattern of poor nutrition from infancy onward and, as we argue, is driven largely by broken relationships at many levels.

A. BREASTFEEDING

Breastfeeding has been shown to confer health, nutritional, immunologic, developmental, psychological, social, economic, and environmental benefits to infants, mothers, families, and society (63). The American Academy of Pediatrics recommends exclusive breastfeeding for approximately six months and continued breastfeeding for at least twelve months (63). Healthy People 2010 goals are to increase the proportion of women who breastfeed to 75 percent in the early postpartum period, 50 percent at six months, and 25 percent at one year (64).



U.S. national statistics continue to show significant gaps in meeting these goals (65). In 2001, only 70 percent of women breastfed their infants in the early postpartum period, and only 36 percent and 18 percent continued to do so at six and twelve months, respectively. The initiation and duration of breastfeeding are significantly lower among certain groups of women. For example, only 54 percent of black women breastfed their infants in the early postpartum period, and only 23 percent and 10 percent continued to breastfeed at six and twelve months, respectively. These disparities are of special concern because black infants could be expected to benefit most from breastfeeding, given their increased risk for infancy and childhood morbidities.

A comprehensive review of all the reasons for the low rates of breastfeeding among U.S. women, particularly among African American women, is beyond the scope of this paper. We refer interested readers to an excellent review by Dr. Barbara Philipp and Sheina Jean-Marie, commissioned by the Courage to Love: Infant Mortality Commission. Entitled *African American Women and Breastfeeding*, the report provides an in-depth examination of various obstacles to breastfeeding (65). Here we simply reframe the problem as one of broken relationships.

Breastfeeding is one of the first acts of relationship building between the mother and her infant. Thus, we see failure to breastfeed as a problem not only of broken bond between mother and child, but also of broken relationships between the mother and her family, friends, and healthcare providers (interpersonal level); the mother and her workplace and birthing hospital (community level); and the mother and our social institutions, including the government and media (institutional and societal level). Failure to breastfeed is the failure of the partner and the healthcare providers to support and encourage breastfeeding. Partner support and provider encouragement are two of the strongest determinants of a woman's decision whether or not to breastfeed. Using data from a national survey, Lu et al. (66) found that women who were encouraged by their "doctors/nurses" to breastfeed were more than four times (RR = 4.39; 95%CI: 2.96, 6.49) as likely to initiate breastfeeding as women who did not receive encouragement. In populations traditionally less likely to breastfeed, provider encouragement significantly increased breastfeeding initiation—by more than threefold among low-income, young, and less-educated women; by nearly fivefold among black women; and by nearly eleven-fold among single mothers. However, more than one in four women in the survey reported that their doctors/nurses never once encouraged them to breastfeed (66).

Failure to breastfeed is the failure of the workplace to create a supportive environment for breastfeeding and the failure of birthing hospitals to adopt baby-friendly policies. Worldwide, approxi-

mately 19,250 sites have achieved baby-friendly status, including 6,312 baby-friendly hospitals in China and 1,147 in Nigeria. In some countries, such as Sweden and Oman, every hospital is baby-friendly. As of 2005, only 52 sites (out of 4,000 possible sites) are located in the United States (65). Failure to breastfeed is also the failure of our government and the media to promote breastfeeding. One of the biggest drop-offs in breastfeeding rates occurs when the mother returns to work. A national parental leave policy (Family and Medical Leave Act) that guarantees a short (6-8 weeks), unpaid leave to less than half of working mothers employed in the private sector (FMLA makes multiple exemptions for small businesses, part-time workers, etc.) does little to support our national goals for breastfeeding duration, especially in light of the little support most women receive for breastfeeding at their workplace. The media have the ability to influence perceptions and beliefs and, as Dr. Philipp observes, contribute to the misperception that formula feeding is the norm and breastfeeding is not. In addition, breastfeeding is often misrepresented in the media as dangerous, difficult, and painful (65).

In caring relationships, the partner, family, friends, healthcare providers, workplace, birthing hospitals, government, and media all take an active role in supporting breastfeeding. In a careless society, that does not happen. Failure to breastfeed is the first manifestation of the lifelong influences of broken relationships on nutrition over the child's life course.

B. FAMILY MEALS AND FAST FOOD

The family meal provides an important opportunity for enhancing family connectedness and role-modeling of healthful eating habits (67). Research has shown that the development of family unity through family meals is important during adolescence (68, 69) and may provide the structure and sense of unity and connectedness that young children need to feel safe and secure (70-72). Moreover, family meal frequency is positively associated with nutritional intake among children (73) and adolescents (74, 75) and inversely associated with eating disturbances or unhealthy eating patterns, including skipping breakfast (75). A cross-sectional survey of a national convenience sample of children ages nine to fourteen found that eating family dinner was associated with healthful dietary intake patterns, including more fruits and vegetables, less fried food and soda, less saturated and trans fat, lower glycemic load, and more fiber and micronutrients from food. Adolescents who reported more frequent family meals, a high priority for family meals, a positive atmosphere at family meals, and a more structured family meal environment were less likely to engage in disordered eating even after controlling for more global family factors such as connectedness (76).



A general decline in the frequency of family meals has been observed over the past few decades; children's reports of consuming a home dinner decreased from 89.2 percent in 1973 to 75.9 percent in 1994, while eating a dinner prepared outside the home increased from 5.4 percent to 19.0 percent ($P < .01$) (77). Using data from a nationally representative sample of 16,810 individuals ages 12-29 in the 1977-1978 Nationwide Food Consumption Survey and the 1989-1991 and 1994-1996 Continuing Surveys of Food Intake by Individuals, Nielsen et al. (78) reported that the percentage of energy obtained from meals consumed at home had decreased from 74 percent in 1977-78 to 59 percent in 1994-95. The decline in family meals has been concomitant with the rise in fast-food consumption; Nielsen et al. reported that the percentage of energy obtained from restaurant and fast-food meals increased from 6 percent in 1977-78 to 21 percent in 1994-95. One study (79) of nearly 5,000 middle and high school students from Minneapolis/St. Paul public schools found that African American children consumed, on average, the fewest number of family meals per week (4.1), while Hispanic children reported 4.5 family meals per week. Native American and non-Hispanic white children did not do much better, reporting 4.2 family meals per week, while Asian and Pacific Islander students consumed the highest number (5.3).

The reasons for the decline in family meals are unclear. Opportunities for family meals have been negatively affected by changes in our society, including changes in family structure and living arrangements, participation of women in the workforce (74), increased availability of convenience foods (80), and increased eating outside of the home (77). We see the decline in family meals as yet another manifestation of broken relationships at many levels in our society. This is most apparent in many single-parent families headed by working mothers, who must juggle work demands and household responsibilities—who has time to prepare and eat a family meal regularly? Two-parent families with both parents working do not have it much easier—the decline in family meals reflects a growing disconnect between over-worked parents and over-scheduled children. And while the decline in family meals is primarily a manifestation of broken relationships at home, it suggests increasing disconnect with friends, neighbors, and communities as well. After all, if you are too busy to sit down and eat a family meal with your children (and too tired to prepare one), how are you going to find time to get together with your friends, volunteer for your church, participate in the PTA, or join in a cause? Thus, the decline in family meals and the concomitant rise in fast-food consumption symbolize a growing disconnect at many levels in our society, and it is this disconnect that patterns nutritional behaviors of children growing up, which become difficult to change when female children become mothers themselves.

C. MARKETING OF JUNK FOODS TO CHILDREN

Another disturbing trend is the marketing of junk foods to children. An Institute of Medicine (IOM) report, "Food Marketing to Children and Youth: Threat or Opportunity?" (81), estimated that more than \$10 billion per year is spent for all types of food and beverage marketing to children and youth in America. Moreover, the preponderance of the products introduced and marketed to children and youth have been high in total calories, sugars, salt, and fat, and low in nutrients. Marketing approaches have become multifaceted and sophisticated, moving far beyond television advertising. A 2003 report by the nonprofit Center for Science in the Public Interest (CSPI) identified a plethora of ways that companies target kids in their homes, in their schools, on the Web, and wherever else kids go (82). Examples highlighted in the report include:

- McDonald's Barbie has the doll dressed up as a McDonald's clerk, feeding French fries, burgers, and Sprite to kid-sister Kelly in a restaurant playset. Other junk-food ads are disguised as toys, like Play Doh's Lunchables kit, where kids are encouraged to assemble Play Doh versions of Oscar Mayer's notoriously fatty and salty lunch box items.
- The Oreo Adventure game on Kraft Foods' Nabiscoworld.com website is one of many corporate "advergaming." In this video game, children's "health" is reset to "100 percent" when kids acquire golden cookie jars on a journey to a Temple of the Golden Oreo. The Oreo Matchin' Middles shape-matching game, produced with Fisher Price, turns playtime into a chance for companies to cultivate brand loyalty and sell junk food.
- Pepsi's website profile of New York Yankees baseball star Jason Giambi, which prominently displays the quote, "I usually have several Pepsis each day—it really lifts me up," is one of many examples of a junk-food marketer linking consumption of its product with fitness.
- Campbell's "Labels for Education" program encourages families to collect labels from Campbell products that schools can redeem for equipment. It is hardly model philanthropy, says CSPI, seeing that kids' parents would have to buy some \$2,500 worth of soup for the school to qualify for a \$59 stapler.



- Krispy Kreme “Good Grades” program offers elementary school kids one doughnut for each “A” on their report cards. CSPI points out that some states wisely prohibit or discourage using food as a reward for good behavior or academic performance.

More troubling is the trend toward increasing marketing of junk foods to children in their schools. Sales of candies, carbonated soft drinks, salty snacks, and other junk foods are promoted at schools as a source of revenue for underfunded school activities. Approximately \$750 million is spent annually selling snacks and processed foods in schools (82).

As the IOM report points out, an important issue in discussions about the marketing of junk foods to children relates to the stages of discernment (81). Before a certain age, children lack the defenses, or skills, to discriminate commercial from noncommercial content, or to attribute persuasive intent to advertising. Children generally develop these skills at about age eight, but children as old as eleven may not activate their defenses unless explicitly cued to do so. The IOM report found strong evidence that television advertising of foods and beverages has a direct influence on what children choose to eat, particularly for children ages two to eleven. In the 1970s and 1980s, the Federal Trade Commission considered restrictions on junk-food advertising aimed at kids, but those efforts were blocked by food, toy, broadcasting, and advertising industries. Many other countries have done a better job of protecting their children from the marketing of junk foods (83). Sweden, Norway, Austria, and Luxembourg have all banned television advertising to children. School-based marketing has been banned in Belgium, France, Luxembourg, Portugal, and Vietnam. In Ireland, where television commercials for candy and fast foods are banned, wrappers must carry warnings that fast food should be eaten in moderation and that sugary foods cause tooth decay.

We see failure to protect children from the marketing of junk foods as yet another manifestation of broken relationships at many levels. It is the failure of the government to regulate marketing of junk foods during children’s television programming and promote more healthful diets through various approaches (e.g., regulation of and incentives to food and beverage companies, provision of healthier foods in school lunch programs). It is the failure of food and beverage companies and full-serve and fast-food restaurants to use their creativity, resources, and full range of marketing practices to promote and support more healthful diets for children and youth. It is the failure of the food, beverage, restaurant, and marketing industries, along with government, scientific, public health, and consumer groups, to establish and enforce

the highest standards for the marketing of foods, beverages, and meals, and the failure of the media and entertainment industry to promote healthful foods and beverages to children and youth. It is the failure of state and local educational authorities to promote healthful diets for children and youth in all aspects of the school environment (e.g., commercial sponsorships, meals and snacks, curriculum), instead of promoting the sale of junk foods in exchange for revenues to sponsor school activities that they underfunded. Most importantly, parents and families remain the central influence on children’s attitudes and behaviors concerning nutrition. Failure to protect children from the marketing of junk foods is the parents’ failure to withstand the pestering of children (driven by advertising and marketing) for junk foods and to promote and provide healthy nutrition instead. Our concern is that if children are not protected from these influential marketing practices, they are more likely to develop unhealthful habits that are carried into adulthood, which become manifest in poor maternal nutrition during pregnancy.

D. FOOD INSECURITY IN A LAND OF PLENTY

The United States is the largest producer of agricultural products in the world. We are also now the world’s largest agricultural exporter (84). Thus, the prevalence of food insecurity in a land where foods are supposedly plentiful and cheaply produced is quite troubling. In 2003, 11.2 percent of all U.S. households, representing greater than 36 million people, experienced food insecurity (85), defined as “whenever the availability of nutritionally adequate and safe food, or the ability to acquire acceptable foods in socially acceptable ways, is limited or uncertain” (86).

Food insecurity has many health consequences for women in low-income households. For example, food insecurity leads to reduced micronutrient intake among women of child-bearing age (87), overweight (88, 89), and an inability during peak weight-gaining years to return to prepregnancy weight status (90). Laraia et al. (91) reported from the Pregnancy, Infection, and Nutrition (PIN) study the prevalence and psychosocial correlates of food insecurity in a cohort of 606 pregnant women with incomes of less than or equal to 400 percent of the federal poverty level. Among these women, 15 percent were from marginally food-secure households and 10 percent were from food-insecure households. Consistent with findings for the general population (92-93), pregnant women who experienced food insecurity had significantly lower income levels, less education, and were more likely to be single and black than women from fully food-secure households. More than one in three (37 percent) black women was food insecure or marginally food secure, a prevalence twice that among white women (19 percent).



Importantly, Laraia et al. (91) found that psychosocial indicators are consistently associated with food security status, with evidence of a dose-response relation. Women who perceived more stress in their lives, or who had poorer scores on psychological state evaluations (trait anxiety, depression symptoms, chance locus of control, powerful others locus of control), were more likely to experience food insecurity, whereas women who had higher scores on personal disposition indices, such as self-esteem and mastery, were less likely to experience food insecurity during pregnancy, adjusting for income, race, education, marital status, age, and number of children. While the direction of causation between psychosocial indicators and food insecurity cannot be determined using these data, given the relative permanence of several of these psychological measures, Laraia et al. (91) suggest that the “full context of life stress and coping behaviors may be as important as income in determining an individual’s risk for food insecurity.” That is, perceived stress, depressive symptoms, and other negative affective states may be important barriers to good maternal nutrition. These women may be in need of psychological counseling in addition to increased access to optimal nutrition. Several studies have found that a high proportion of pregnant and postpartum women who are eligible for WIC but are not receiving benefits are food insecure (94). For some women, improving their nutritional status during pregnancy will take more than improving choices of and access to healthy nutrition; it will take outreach and referrals to social support and psychological counseling.

We see food insecurity in a land of plenty as yet another manifestation of broken relationships in our society. People who are socially isolated are more likely to be food insecure. Food insecurity reflects the failure of healthcare providers to identify, and of public health programs to reach out to, those individuals and families at risk and refer them for food assistance. It reflects the failure of community-based and faith-based organizations to come to the aid of individuals and families in need in their communities. It reflects the failure of state and local governments to fully participate in federal food assistance programs (e.g., the Food Stamps Program), and of the federal government to adequately fund such programs. It reflects the failure of our society to provide living wages and affordable housing to help reduce the competition between household economic pressures—such as child care and health care—and adequate nutritious food (94). In a caring society, no households in America (the largest agricultural producer and exporter in the world) should be food insecure, especially not pregnant women and young children; in a careless society, food insecurity becomes a fact of life for millions of American families.

E. STRESS AND “COMFORT FOODS”

Earlier in the chapter, we reviewed how stress can alter neuroendocrine, immune-inflammatory, and placental vascular responses. Stress can affect health through one more pathway—by influencing behaviors as a means of coping with stress. With respect to nutrition, chronic stress induces “comfort food”-seeking behaviors (95). Students under stress report shifting ingestion from normal (fruits, vegetables, fish, and meat) to sweet and savory foods (96). Moreover, stress precipitates binge eating (97, 98), and women under acute lab stress increase “comfort food” intake (99).

Chronic stress results in hyperactivity of the HPA axis; “comfort foods” appear to blunt the HPA response (reviewed in ref 95). In animal studies, provision of “comfort foods” as high fat, high sugar concentrations, or a combination reduces both autonomic and HPA responses to repeated stressors in rats. Moreover, there is some evidence that “comfort foods” given to chronically stressed rats may negate chronic stress-induced inhibition of dopamine release that occurs in the nucleus Accumbens. “Comfort foods” reduce the negative effects of chronic stressors in the nucleus Accumbens by stimulating the anterior, more pleasure-associated part and inhibiting the stress-stimulated posterior, more defensive part of this cell group. However, the shift in caloric intake from standard chow to preference for “comfort foods,” together with elevated glucocorticoids and insulin, reorganize energy stores from a peripheral to a central distribution (primarily as abdominal fat), thereby predisposing central obesity. An unspecified signal from the abdominal fat stores also acts at the level of the brain to reduce the adverse effects of a recruited chronic stress response network and probably makes animals (and people) feel better under conditions of chronic stress (95). Thus, chronic, uncontrollable stress in our lives, particularly among poor women of color, may be partly responsible for poor maternal nutrition as well as the growing epidemic of obesity in our society.

In sum, poor maternal nutrition follows a lifelong pattern of poor nutrition, driven largely by broken relationships at many levels. These broken relationships are manifested in the lack of support for breastfeeding, the decline in family meals concomitant to the rise of fast food, the marketing of junk foods to children, and the prevalence of food insecurity in a land of plenty. These broken relationships create lifelong conditions of high stress and low support, which put the mother at risk for poor nutrition during pregnancy and her baby at risk for fetal and infant death. Improving maternal nutrition during pregnancy, therefore, will take more than improving food choices and access; it will take repairing broken relationships at many levels and over the life course. In the next section, we begin to explore how to repair the broken relationships in our society.



III. REPAIRING BROKEN RELATIONSHIPS

If “black infants are dying because their mothers are dying in dead relationships,” then dead relationships must be revived to keep babies from dying. Earlier we cited marital relationships, social capital, and racism as manifestations of the many broken relationships in our society. We now turn to these as examples of how to repair broken relationships.

A. RESTORING FATHERS TO FAMILIES

Men can be a vital source of support to their partners and resiliency to their children. Yet today, many men do not stay involved in the pregnancies they have caused or in raising the children from these pregnancies. Father absence affects families of all racial-ethnic and socioeconomic backgrounds, but it disproportionately affects African American families. In 2002, 68 percent of African American infants were born to unmarried mothers, up from 22 percent in 1960 (2). Among poor black infants, only three percent were born into married families (100). Approximately one-third of poor black infants were born into single-mother families with little or no father involvement. More than half (53 percent) were born into so-called “fragile families,” in which the child’s biological parents were unmarried but cohabiting or the father visited at least once a week. While many of these unmarried fathers may have been actively involved at birth, over time their involvement declined. As a result, nearly half (49 percent) of all poor black children lived in single-mother families with little or no father involvement (100).

The cause of father absence in the African American community is not clearly understood (101). Some see father absence as historically rooted in the legacy of slavery, whereby the male slave could fulfill none of the duties of husband and father (102). Others point to changing cultural norms and attitudes toward sex and marriage leading to high rates of out-of-wedlock births, low rates of marriage, and conflictual gender relations (103). Still others blame economic dislocation and marginality, which make it nearly impossible for a great number of black men to be adequate providers for their families (104). And while father absence is not unique to the black community, its toll on black women and children is especially high. Studies have shown that, controlling for parental education, income, and other confounding factors, children growing up in father-absent families are at greater risk for various educational or behavioral problems and poorer developmental outcomes (105).

How can we repair these broken relationships at home? How do we restore black fathers to black families? Prevailing “male

involvement” programs that focus primarily on Sexually Transmitted Disease screening, or “father involvement” programs that focus solely on collection of child support, will not work. What is needed is a multilevel, comprehensive approach, guided by both an ecological model (106) and the life-course perspective (40). An ecological model addresses barriers to father involvement at multiple levels. At the individual level, fathers need educational programs, employment-related services, and legal and social services (107). At the interpersonal level, efforts should focus on improving the relationships between black men and women, including marriage counseling or skills training in communication and conflict resolution. At the community and institutional level, some black churches, universities, and media have taken on leadership of the fatherhood movement, but more need to do so (102). These institutions can help promote changes in norms, values, and expectations that support marriage and strengthen the father-child bond. At the policy level, public policy needs to support fragile families so that they can stay together. Current policies often do the opposite. Policy reforms that remove disincentives for father involvement in Temporary Assistance for Needy Families (e.g., eliminating the distinction between single- and two-parent families for eligibility determination), the Earned Income Tax Credit (e.g., allowing a second-earner deduction), and child support (e.g., establishing amnesty programs or extending TANF, EITC, and other support services to non-custodial fathers who pay child support) are needed to keep fragile families intact (108, 109). Most importantly, opening up economic opportunities by promoting full and consistent employment, job skills training and retraining, fair trade, and unionization will go a long way toward restoring black fathers to black families (102-104). The life-course perspective recognizes that, much like mothers, fathers have a life history of their own (101). Their involvement in their children’s lives is determined in part by their own life experiences, including their father’s involvement in their lives growing up. Their capacity to support and nurture needs to be cultivated not only after they become a father, but over their entire life course. What is needed are more research studies on how boys learn to become fathers and more programs that reach out to boys and young men and give them the life skills and opportunities to become responsible fathers.

In sum, fathers can be a vital source of support, or a great source of stress, in pregnancy and parenting. A number of historical, economic, and cultural trends have led to father absence in black America. Restoring black fathers to black families will take a multilevel, life-course approach.



B. BUILDING REPRODUCTIVE SOCIAL CAPITAL

Earlier in the chapter, we discussed the relationship between social capital and community health—the more connected you are to your neighbors, the healthier you will be. Here we introduce a related concept: reproductive social capital. Reproductive social capital can be defined as those features of social organization (e.g., networks, norms, and social trust) that facilitate coordination and cooperation to promote reproductive health within a community (110). With respect to pregnancy, it describes the degree of social connectedness of the pregnant woman to her community. It is characterized not only by how many neighbors a pregnant woman can turn to for help and support, but also by how a community treats a pregnant woman. Does the community provide safeguards and resources for pregnant women? How actively engaged is the community in promoting healthy pregnancy? Do members of the community feel a sense of solidarity with the pregnant woman and of responsibility for the well-being of that pregnancy (110)?

Presently, little is known about how to create reproductive social capital in a community. An innovative community-based program in South and Central Los Angeles may be instructive (110). One Hundred Intentional Acts of Kindness toward a Pregnant Woman was conceived by Healthy African American Families (HAAF) as a media campaign to create reproductive social capital for pregnant women. Through focus groups, pregnant women were asked to identify actions that families, friends, and even strangers could do to make their pregnancies better. From families and friends, pregnant women primarily wanted emotional and instrumental support (e.g., help with cooking, housecleaning, child care or transportation, as well as encouragement); from strangers they wanted respect for personal space (e.g., ask permission to touch my belly) and common courtesy (e.g., offer me your seat on the bus; allow me to go in front of you at a grocery store counter or while waiting for the restroom). Based on the responses gathered from focus groups, a list of one hundred “Intentional Acts of Kindness” was created. This list has been printed on fans and distributed in churches, barber shops, nail salons, and other locations, and will be disseminated widely in the community through other media. While the effectiveness of One Hundred Acts is currently being evaluated, the program provides an example of what a community could do to create reproductive social capital and help increase social support for pregnant women in their everyday lives.

One Hundred Acts offers several important lessons for repairing broken relationships and building new ones. First, the kinds of social support that pregnant women wished for from families, friends, or even strangers are not what is typically provided

through public health case management or home visitations. What many pregnant women wanted from families and friends was consistent, daily emotional and instrumental support (e.g., “ask me if there is anything I need,” “don’t argue with me,” “fix me a meal,” and “take a walk with me”). What they wanted mostly from strangers was common courtesy, which included respect for personal space and avoidance of prying (e.g., “don’t talk to me/leave me alone,” and “ask permission to touch my belly”), as well as simple acts of kindness (e.g., “offer me your seat on the bus,” “let me sit near the bathroom at church,” and “allow me to go ahead of you in line”). These simple acts of kindness from families, friends, and even strangers may help buffer against the chronic stress that many pregnant African American women experience daily, and hypothetically may help prevent activation of the body’s physiologic response to stressors that could lead to preterm labor.

Second, One Hundred Acts has the potential to do more than increase social support at the individual level; it could also help create reproductive social capital at the community level. By asking for acts of kindness from families, friends, and even strangers, it strengthens their sense of connectedness to, solidarity with, and responsibility for the pregnant women in their community. It establishes new social norms about how pregnant women are valued, treated, and respected. It engages active participation from community members in promoting healthy pregnancy for all women. While “Acts of Kindness” do not by themselves address structural issues—such as housing, employment, and discrimination—that are the source of chronic stressors for many pregnant African American women, we believe that they can help create a sense of solidarity and collective efficacy in the community. It takes a village to have a healthy baby.

Third, One Hundred Acts demonstrates the power of participatory action research (PAR). PAR shares with “community-based action research,” “community partner participatory research,” “mutual inquiry,” and “empowerment evaluation” a series of core principles and values, among which is a shared commitment to actively engage those who live in the community and whose lives are affected by the issue under study in every phase of the research process (111). The traditional lines between the researcher and the researched are blurred “through processes that accent the wealth of assets that community members bring to the process of knowing and creating knowledge and acting on that knowledge to bring about change.” One Hundred Acts was conceived by such processes. While academic partners contributed theories and resources and provided some technical assistance, One Hundred Acts has been driven from the outset by community partners in its design, implementation, and evaluation. It is in repairing old, broken relationships and building new relationships—between



strangers on a bus, between pregnant women and their communities, and between academic researchers and community partners—that social capital is created.

C. UNDOING RACISM

In addressing racial-ethnic disparities in infant mortality, the “elephant in the room” is racism. We will not eliminate health disparities in the U.S. without confronting racism in our society. A growing body of evidence suggests that racism may be the “cause of the causes” of health disparities in the United States, including disparities in birth outcomes. Earlier in the chapter, we discussed different levels of racism; Camara Jones (17) proposed a theoretical framework for understanding racism on 3 levels: internalized, personally mediated, and institutionalized. Internalized racism is defined as acceptance by members of the stigmatized races of negative messages about their own abilities and intrinsic worth. Personally mediated racism is defined as prejudice or discrimination—i.e., differential assumptions about or actions toward others according to their race. Institutionalized racism is defined as differential access to the goods, services, and opportunities of society by race. It can manifest as differential access to quality education, sound housing, gainful employment, appropriate medical facilities, and a clean environment. Residential segregation exemplifies institutionalized racism; a greater black-white gap in infant mortality has been found in cities that are more segregated (18, 19).

Dr. Jones uses an allegory about a gardener with two flower boxes to illustrate the three levels of racism (17). One of the flower boxes has rich and fertile soil; the other has poor and rocky soil. In the rich soil, the gardener planted seeds for red flowers, and in the poor soil, she planted seeds for pink flowers. As expected, all the red flowers grow full and vigorous and strong, while the pink flowers struggle to survive. In time, the flowers in the two boxes go to seed, dropping their progeny into the same soil in which they were growing. Year after year, the red flowers flourish while the pink flowers languish. To put things right in this garden, according to Dr. Jones, it is not enough to address internalized racism by telling the pink flowers, “Pink is beautiful!” or to address personally mediated racism by conducting educational workshops with the gardener. To grow strong pink flowers, we need to “break down the boxes and mix up the soil, or... leave the two boxes separate but fertilize the poor soil until it is as rich as the fertile soil.” She concluded that institutionalized racism is the most fundamental of the three levels and must be addressed for important change to occur.

Thus, undoing racism will take more than changing knowledge, attitudes, and behaviors at the individual level; it will take

addressing institutionalized racism in health care, education, criminal justice, economic development, and many other domains of our lives (112). With respect to infant mortality, researchers need to do more to better understand the impact of racism on birth outcomes, including developing better measures of racism, mapping out causal pathways linking racism to outcomes, and studying not only current or recent racism, but racism over the life course and across generations. Researchers also need to develop intervention studies to address the impact of institutionalized racism on birth outcomes. Healthcare providers and other service providers are not exempt from addressing racism just because it is perceived to be outside of the clinical domain. For a start, they need to critically examine their personal attitudes and behaviors, as well as institutional practices and policies, to ensure that all of their patients or clients, regardless of race and ethnicity, receive equal treatment (113). Public health professionals need to make racism a leading public health issue, much as the Boston Public Health Commission and CityMatCH have begun to do. They can address racism through the core functions of public health (assessment, assurance, and policy development), including collecting data on racism as part of population surveillance and community health assessment, ensuring equal access to quality health care and monitoring for discriminatory practices, and making policies (or collaborating with other public agencies such as housing, labor, and welfare to make policies) that address differential access to goods, services, and opportunities vital to maternal and child health. In research, practice, and policy development to address racism, community voices must be heard. They have been telling us for a long time that racism is the “elephant in the room,” the “cause of the causes” of disparities in birth outcomes; now the community must be included as partners in our collective effort to undo racism.

SUMMARY

In this chapter, we examined the problems of infant mortality and poor maternal nutrition in a context of relationality. We contend that both are, in essence, problems of broken relationships at many levels, which are manifested in the lack of support for breastfeeding, the decline in family meals concomitant to the rise of fast food, the marketing of junk foods to children, and the prevalence of food insecurity in a land of plenty. These broken relationships create lifelong conditions of high stress and low support, which in turn pattern physiological, psychological, and behavioral responses that put the mother at risk for poor nutrition during pregnancy and her baby at risk for fetal and infant death. African American families are disproportionately affected by these broken relationships, which contribute to disparities in maternal nutrition and infant mortality.



CHAPTER 5 RECOMMENDATIONS

There is no quick fix or silver bullet for our nation's infant mortality problem. Nutrition can play a key role in preventing several leading causes of infant mortality, but only as part of a longitudinally and contextually integrated strategy for improving maternal and family health. In this report, we examined the problems of infant mortality and poor maternal nutrition in a context of relationality. We contend that both are, in essence, problems of broken relationships at many levels. These broken relationships create lifelong conditions of high stress and low support, which in turn pattern physiological, psychological, and behavioral responses that put the mother at risk for poor nutrition during pregnancy and her baby at risk for fetal and infant death. African American families are disproportionately affected by these broken relationships, which contribute to disparities in maternal nutrition and infant mortality. As noted earlier, we concur with the recommendation of the Courage to Love: Infant Mortality Commission that efforts to reduce maternal and infant mortality and morbidity must focus on the repair and support of relationships at all levels and across the life course.

We conclude this report with recommendations for priority areas in research, practice, and policy related to maternal nutrition and infant mortality.

I. RESEARCH

PRIORITY AREA ONE: PRECONCEPTIONAL AND INTERCONCEPTIONAL NUTRITION

Several leading causes of infant mortality, including preterm birth, fetal growth restriction, birth defects, and preeclampsia, may have their onset early in pregnancy, around the time of implantation and placentation. Moreover, maternal nutritional status before pregnancy may influence a mother's susceptibility to these pregnancy complications. Maternal nutritional supplementation programs that begin during pregnancy, often with initiation of prenatal care, probably do too little too late to avert these complications. The effectiveness of maternal nutritional interventions can be improved if they are begun before pregnancy.

- **Recommendation:** Researchers should conduct more intervention studies of maternal nutritional supplementation that begin before (preconceptional) and between (interconceptional) pregnancies. The National Institutes of Health and other funding agencies should support these studies.

PRIORITY AREA TWO: CONTENT OF NUTRITIONAL SUPPLEMENTATION

Most women with nutritional deficits are deficient in not only one nutrient, but multiple macro- and micronutrients. Yet most randomized controlled trials to date have focused on studying the impact of single-nutrient supplementation. It is premature to conclude from these single-nutrient supplementation trials that nutritional interventions do not work. Future studies need to examine the impact of a combination, bundle, or package of nutrients on both short-term and long-term maternal and child health outcomes.

- **Recommendation:** Researchers should conduct more intervention studies of multinutrient maternal nutritional supplementation. The National Institutes of Health and other funding agencies should support these studies.

PRIORITY AREA THREE: RELATIONAL CONTEXT OF MATERNAL NUTRITION

A central theme of this report is that poor maternal nutrition follows a lifelong pattern of poor nutrition, driven largely by broken relationships at many levels in our society. As discussed in Chapter 4, these broken relationships are manifested in the lack of support for breastfeeding, the decline in family meals concomitant to the rise of fast food, the marketing of junk foods to children, and the prevalence of food insecurity in a land of plenty. Thus, improving maternal nutrition will take more than nutritional supplementation during pregnancy; it will take repairing and supporting relationships at all levels and across the life course.

- **Recommendation:** Researchers should conduct more research studies of the influences of partner support, provider encouragement, social network, social capital, and other relational contexts on maternal nutritional status and behaviors. The National Institutes of Health and other funding agencies should support such research.
- **Recommendation:** Researchers should conduct more intervention studies that build upon these relational contexts for improving maternal nutritional status and behaviors before and during pregnancy. The National Institutes of Health and other funding agencies should support these studies.



PRIORITY AREA FOUR: PSYCHOSOCIAL FACTORS AND MATERNAL NUTRITION

Psychosocial factors such as stress and social support can strongly influence maternal nutritional status and behaviors before and during pregnancy. As discussed in Chapter 4, broken relationships over the life course create lifelong conditions of high stress and low support, which put the mother at risk for poor nutrition during pregnancy and her baby at risk for fetal and infant death. For example, social isolation has been linked to food insecurity. Further research is needed to examine the influences of psychosocial factors on maternal nutritional status and behaviors.

- **Recommendation:** Researchers should conduct more research studies of the influences of psychosocial factors on maternal nutritional status and behaviors before and during pregnancy. Such research efforts should be guided by an integrative framework that takes into account the multilevel influences of stress and nutrition. The National Institutes of Health and other funding agencies should support this research.
- **Recommendation:** Researchers should conduct more intervention studies that test different social support strategies for improving maternal nutritional status and behaviors before and during pregnancy. Development of such strategies should be guided by the principles and methods of community participatory action research. The National Institutes of Health and other funding agencies should support these studies.

PRIORITY AREA FIVE: LIFE-COURSE INFLUENCES ON MATERNAL NUTRITION

As noted above, poor nutrition usually does not begin during pregnancy; it follows a lifelong pattern of poor nutrition. Further research is needed to examine life-course influences on maternal nutritional status and behaviors before and during pregnancy.

- **Recommendation:** Researchers should conduct more studies that identify critical influences over the life course on maternal nutritional status and behaviors. The National Institutes of Health and other funding agencies should support this research.
- **Recommendation:** Researchers should conduct more intervention studies that address these critical life-course influences for improving maternal nutritional status and behaviors before and during pregnancy and

over the life course. The National Institutes of Health and other funding agencies should support such studies.

II. PRACTICE

PRIORITY AREA SIX: INCREASE SURVEILLANCE OF FOOD INSECURITY

Food insecurity is common among pregnant women in the U.S. For example, in a study of 606 pregnant women by Laraia et al., one of every ten was food insecure and one in six was marginally food secure; one of every three African American women in the study was food insecure or marginally food secure. Presently, food insecurity is not routinely screened in prenatal care, nor is it routinely monitored in maternal and child health (MCH) population surveillance. Increasing clinical assessment and population surveillance of food insecurity must be made a practice priority.

- **Recommendation:** The American College of Obstetricians and Gynecologists (ACOG), the American Academy of Pediatrics, and other professional organizations should recommend routine assessment of food insecurity during perinatal care as practice guidelines, and the Centers for Disease Control and Prevention, the Maternal and Child Health Bureau, and state and local Title V agencies should routinely monitor food insecurity in MCH populations.
- **Recommendation:** Prenatal care providers must ensure that women and families who screened positive for food insecurity receive appropriate nutritional services, and state and local Title V agencies must ensure that such services are available and accessible to women and families who need them. All eligible women and families who are at nutritional risk must be referred to WIC.

PRIORITY AREA SEVEN: ADDRESS PSYCHOSOCIAL FACTORS IN PREGNANCY

Psychosocial factors such as stress and support can have a significant impact on maternal nutrition and pregnancy outcomes. For example, women who perceive more stress in their lives or who have poorer scores on psychological state evaluations are more likely to experience food insecurity, whereas women who have higher scores on personal disposition indices such as self-esteem and mastery are less likely to experience food insecurity during pregnancy. Presently, these psychosocial factors are not adequately addressed in perinatal care.



- **Recommendation:** Perinatal care providers, including WIC programs, should routinely assess psychosocial factors, including stress and social support, among pregnant women and their families. Those who screen positive should be referred to appropriate support services (e.g., psychological counseling for women with depressive or other affective disorders, support services for women and children who are victims of intimate partner violence).

PRIORITY AREA EIGHT: SUPPORT HEALTHY NUTRITION IN THE RELATIONAL CONTEXTS

Maternal nutritional behaviors are strongly influenced by a woman's partner, family, peers, healthcare providers, and significant others. These existing relationships can be used to support and encourage healthy nutrition before and during pregnancy. For example, a pregnant woman's mother, sister, or friend from church can be recruited and trained to serve as a "maternity buddy" to support and encourage healthy behaviors during pregnancy, including nutritional behaviors. Other sources of support and encouragement can be found through *doulas* or other pregnant women.

- **Recommendation:** Perinatal care providers, including WIC programs, should experiment with the use of a pregnant woman's own personal relationships to support her nutritional behaviors. Such experiments should be supported and encouraged by health plans, Title V programs, and other public agencies or private foundations. The use of *doulas*, group prenatal care (e.g., Centering Pregnancy), and other forms of social support for improving maternal nutrition should also be explored.

III. POLICY

PRIORITY AREA NINE: PROMOTE BABY-FRIENDLY HOSPITALS AND WORKPLACE

We can get babies off to a good nutritional start by promoting baby-friendly policies in birthing hospitals and in the workplace. A ten-step baby-friendly program has already been adopted by 19,250 sites worldwide; only 52 sites are located in the U.S.

- **Recommendation:** The Department of Health and Human Services, the Centers for Disease Control and Prevention, the Maternal and Child Health Bureau, state and local Title V agencies, and other public and private organizations should promote the adoption of baby-friendly policies in birthing hospitals in the U.S. All

public agencies should adopt baby-friendly policies in the workplace, including provision of a breastfeeding room for nursing mothers.

PRIORITY AREA TEN: REGULATE MARKETING OF JUNK FOODS TO CHILDREN

Chapter 4 discussed the marketing of junk foods to children in schools and through various media, including television. Such marketing practices can have strong influences on a child's nutritional behaviors that may persist into adulthood (and pregnancy). As demonstrated in other countries, much more can be done through public policy to protect our children from the marketing of junk foods.

- **Recommendation:** The Federal Trade Commission should regulate the marketing of junk foods during children's television programming. State and local educational authorities should promote healthful diets for children and youth in all aspects of the school environment (e.g., restrictions on commercial sponsorships of school activities, banning of soda and other junk food vending machines from schools, provision of healthy nutrition through school lunch programs, and development of educational curricula to promote healthy nutrition).

PRIORITY AREA ELEVEN: STRENGTHEN WOMEN, INFANTS, AND CHILDREN (WIC) PROGRAMS

WIC is not a magic bullet to our nation's infant mortality problem. Given what is known about the pathogenic processes underlying some of the leading causes of infant mortality (e.g., preterm birth, fetal growth restriction), WIC alone cannot be expected to prevent these adverse outcomes, nor should its effectiveness be evaluated based on these outcomes. Nonetheless, WIC remains the one program with the greatest promise for improving maternal and family nutrition in the U.S. and, given the growing body of evidence of developmental programming, the greatest promise for optimizing child health and development.

- **Recommendation:** The Department of Agriculture should continue to explore ways to strengthen WIC. The commissioned Institute of Medicine report on repackaging WIC food packages is a good start, but more can be done to enhance the content of WIC food packages to improve maternal health and optimize developmental programming. Additionally, more efforts and resources are needed to improve outreach, health education, service



coordination and systems integration, and community building to increase food access and quality, particularly in disadvantaged communities. More attention must be paid to addressing psychosocial barriers to WIC participation. WIC alone is not the answer to infant mortality, however. The Department of Agriculture needs to join forces with the Centers for Disease Control and Prevention, the Maternal and Child Health Bureau, the National Institute of Child Health and Development, and other leaders in MCH to develop a roadmap for addressing this nation's infant mortality problem, using an integrative approach and making improvement of maternal and infant nutrition a priority.

PRIORITY AREA TWELVE: SUPPORT FRAGILE FAMILIES AND STRENGTHEN PARTNER/FATHER INVOLVEMENT

Men can be a vital source of support for pregnant women and a source of resiliency for their children. However, our tax (Earned Income Tax Credit), welfare (Temporary Assistance for Needy Families), and child support policies are tearing up families and creating an underground cadre of fathers who want to be involved in their children's lives but cannot. Supporting fragile families and strengthening partner/father involvement must be a policy priority in our national efforts to improve maternal nutrition and reduce infant mortality.

- **Recommendation:** Federal and state governments should pass legislation to encourage family formation and remove disincentives for partner/father involvement in TANF (e.g., eliminating the distinction between single- and two-parent families for eligibility determination), EITC (e.g., allowing a second-earner deduction), and child support programs (e.g., establishing amnesty programs; allowing greater "pass-through" of child-support payments to children; and extending TANF, EITC, and other support services to non-custodial fathers who pay child support). They should also increase support for educational programs, employment-related services, legal and social services, and marriage counseling for low-income fathers to strengthen their capacity to fulfill the roles and responsibilities of fatherhood.

PRIORITY AREA THIRTEEN: ADDRESS INSTITUTIONALIZED RACISM IN MATERNAL NUTRITION

Institutionalized racism is defined as differential access to the goods, services, and opportunities of society by race. With respect to nutrition, institutionalized racism is manifested in differential access to healthy foods. In many poor communities and communities of color, there are more liquor stores than grocery stores and more fast-food restaurants than healthy restaurants. The price of food is higher, and the quality is lower, in poor than in non-poor communities. Differential access to healthy nutrition over the life course becomes manifest in differential maternal nutritional status and behaviors during pregnancy.

- **Recommendation:** State and local governments, civic and business leaders, community- and faith-based organizations, and other community leaders can all play an active role in improving maternal and family access to healthy nutrition. Tax incentives, subsidies, and other incentives can be provided to encourage grocers, restaurants, and farmers' markets to do business in the community, and to induce fast-food restaurants and sundry stores (even liquor stores) to sell healthy produce. Multilevel, multichannel health messaging should be undertaken to stimulate demand for healthy nutrition. Title V and other public health agencies need to routinely monitor differential access to healthy nutrition and study its impact on health disparities.

REFERENCES

CHAPTER 1

MATERNAL NUTRITION AND INFANT MORTALITY

1. Hoyert DL, Mathews TJ, Menacker F, Strobino DM, Guyer B. Annual summary of vital statistics: 2004. *Pediatrics*. 2006;117:168-83.
2. Liu K, Moon M, Sulvetta M, Chawala J. International infant mortality rankings: a look behind the numbers. *Health Care Financ Rev*. 1992;13 :105-118.
3. Sepkowitz S. International rankings of infant mortality and the United States' vital statistics natality data collecting system: failure and success. *Int J Epidemiol*. 1995;24:583-8.
4. Mathews TJ, Menacker F, MacDorman MF; Centers for Disease Control and Prevention, National Center for Health Statistics. Infant mortality statistics from the 2002 period: linked birth/infant death data set. *Natl Vital Stat Rep*. 2004 Nov 24;53(10):1-29.
5. Geronimus AT. Black/white differences in the relationship of maternal age to birthweight: a population-based test of the weathering hypothesis. *Soc Sci Med*. 1996;42:589-97.
6. Raatikainen K, Heiskanen N, Heinonen S. Marriage still protects pregnancy. *BJOG* 2005;112:1411-6.
7. Waldron I, Hughes ME, Brooks TL. Marriage protection and marriage selection – prospective evidence for reciprocal effects of marital status and health. *Soc Sci Med*. 1996;43:113-23.
8. Lu MC, Halfon N. Racial and ethnic disparities in birth outcomes: a life-course perspective. *Matern Child Health J*. 2003;7:13-30.
9. Guendelman S, English PB. Effect of United States residence on birth outcomes among Mexican immigrants: an exploratory study. *Am J Epidemiol*. 1995;142:S30-8.
10. See http://www.cdc.gov/nchs/data/nvsr/nvsr53/nvsr53_10.pdf.
11. Mitchell LE, Adzick NS, Melchionne J, Pasquariello PS, Sutton LN, Whitehead AS. Spina bifida. *Lancet*. 2004 Nov 20-26;364(9448):1885-95.
12. Mulinare J, Cordero JF, Erickson JD, Berry RJ. Periconceptional use of multivitamins and the occurrence of neural tube defects. *JAMA* 1988;260:3141-5.
13. Bower C, Stanley FJ. Dietary folate as a risk for the neural-tube defects: evidence from a case-control study in Western Australia. *Med J Aust*. 1989;150:613-9.
14. Werler M, Shapiro S, Mitchell AA. Periconceptional folic acid exposure and risk of occurrent neural tube defects. *JAMA* 1993;269:1257-61.
15. Friel JK, Frecker M, Fraser FC. Nutritional patterns of mothers of children with neural tube defects in Newfoundland. *Am J Med Genet*. 1995;55:195-9.
16. Shaw GM, Schaffer D, Velie EM, Morland K, Harris JA. Periconceptional vitamin use, dietary folate, and the occurrence of neural tube defects. *Epidemiology* 1995;6:219-26.
17. Milunsky A, Jick H, Jick SS, Bruell CL, MacLaughlin DS, Rothman KJ, Willett W. Multivitamin/folic acid supplementation in early pregnancy reduces the prevalence of neural tube defects. *JAMA* 1989;262:2847-52.
18. Berry RJ, Li Z, Erickson JD, Li S, Moore CA, Wang H, et al. Prevention of neural-tube defects with folic acid in China. *N Engl J Med*. 1999;341:1485-90.
19. Moore LL, Bradlee ML, Singer MR, Rothman KJ, Milunsky A. Folate intake and the risk of neural tube defects: an estimation of dose-response. *Epidemiology* 2003;14:200-5.
20. Rosenquist TH, Ratashak SA, Selhub J. Homocysteine induces congenital defects of the heart and neural tube: effects of folic acid. *Proc Natl Acad Sci*. 1996;93:15227–32.
21. Ray JG, Blom HJ. Vitamin B12 insufficiency and the risk of fetal neural tube defects. *QJM* 2003;96:289–95.
22. Schorah CJ, Smithells RW. Maternal vitamin nutrition and malformations of the neural tube. *Nutr Res Rev*. 1991;4:33-49.
23. Suarez L, Hendricks K, Felkner M, Gunter E. Maternal serum vitamin B12 levels and risk for neural tube defects in a Texas-Mexico border population. *Epidemiology* 2003;13:81-8.
24. Keen CL, Clegg MS, Hanna LA, Lanoue L, Rogers JM, Daston GP, Oteiza P, Uriu-Adams JY. The plausibility of micronutrient deficiencies being a significant contributing factor to the occurrence of pregnancy complications. *J Nutr*. 2003;133:1597S-1605S.
25. Gabbe SG, Graves CR. Management of diabetes mellitus complicating pregnancy. *Obstet Gynecol*. 2003;102:857-68.
26. Institute of Medicine. *Preterm birth: Causes, consequences, and prevention*. Washington DC: National Academies Press. In press.
27. Iyasu S, Becerra JE, Rowley DL, Hogue, CJ. Impact of very low birth weight on the black-white infant mortality gap. *Am J Prev Med*. 1992;8:271-7.

28. Subcommittee on Nutritional Status and Weight Gain During Pregnancy, Food and Nutrition Board, U.S. Institute of Medicine/ National Academy of Sciences. *Nutrition During Pregnancy*. Washington, DC: National Academy Press, 1990.
29. WHO Collaborative Study. Maternal anthropometry and pregnancy outcomes. *Bulletin of the World Health Organization* 1995;73(Suppl.):1-98.
30. Kramer MS, Seguin L, Lydon J, Goulet L. Socio-economic disparities in pregnancy outcome: why do the poor fare so poorly? *Paediatr Perinat Epidemiol*. 2000 Jul;14(3):194-210.
31. Kramer MS. The epidemiology of adverse pregnancy outcomes: an overview. *J Nutr*. 2003;133:1592S-96S.
32. Lockwood CJ, Kuczynski E. Risk stratification and pathological mechanisms in preterm delivery. *Paediatr Perinat Epidemiol*. 2001;15:78-89.
33. McGregor JA, Allen KG, Harris MA, Reece M, Wheeler M, French JI, Morrison J. The omega-3 story: nutritional prevention of preterm birth and other adverse pregnancy outcomes. *Obstet Gynecol Surv*. 2001 May;56(5 Suppl. 1):S1-13.
34. Olsen SF, Secher NJ, Björnsson S, Weber T, Atke A. The potential benefits of using fish oil in relation to preterm labor: the case for a randomized controlled trial? *Acta Obstetrica et Gynecologica Scandinavica* 2003;82(11):978-82.
35. Olsen SF, Sorensen JD, Secher NJ, Hedegaard M, Henriksen TB, Hansen HS, et al. Randomised controlled trial of effect of fish-oil supplementation on pregnancy duration. *Lancet* 1992;339:1003-7.
36. Olsen SF, Secher NJ, Tabor A, Weber T, Walker JJ, Gluud C. Randomised clinical trials of fish oil supplementation in high risk pregnancies. *Br J Obstet Gynaecol*. 2000;107:382-95.
37. Bloomfield FH, Oliver MH, Hawkins P, Campbell M, Phillips DJ, Gluckman PD, Challis JR, Harding JE. A periconceptional nutritional origin for noninfectious preterm birth. *Science* 2003;300:606.
38. Kumarasamy V, Mitchell MD, Bloomfield FH, Oliver MH, Campbell ME, Challis JR, Harding JE. Effects of periconceptional undernutrition on the initiation of parturition in sheep. *Am J Physiol Regul Integr Comp Physiol*. 2005;288:R67-72.
39. McMillen IC, Schwartz J, Coulter CL, Edwards LJ. Early embryonic environment, the fetal pituitary-adrenal axis and the timing of parturition. *Endocr Res*. 2004;30:845-50.
40. Herrmann TS, Siega-Riz AM, Hobel CJ, Aurora C, Dunkel-Schetter C. Prolonged periods without food intake during pregnancy increase risk for elevated maternal corticotropin-releasing hormone concentrations. *Am J Obstet Gynecol*. 2001;185:403-12.
41. Ronnenberg AG, Wang X, Xing H, Chen C, Chen D, Guang W, Guang A, Wang L, Ryan L, Xu X. Low preconception body mass index is associated with birth outcome in a prospective cohort of Chinese women. *J Nutr*. 2003 Nov;133(11):3449-55.
42. Carmichael SL, Abrams B. A critical review of the relationship between gestational weight gain and preterm delivery. *Obstetrics and Gynecology* 1997;89:865-73.
43. Rush D. Maternal nutrition and perinatal survival. *Journal of Health, Population and Nutrition* 2001;19:S217-64.
44. Sullivan FM, Barlow SM. Review of risk factors for sudden infant death syndrome. *Paediatr Perinat Epidemiol*. 2001 Apr;15(2):144-200.
45. Durlach J. New data on the importance of gestational Mg deficiency. *J Am Coll Nutr*. 2004 Dec;23(6):694S-700S.
46. Chang J, Elam-Evans LD, Berg CJ, Herndon J, Flowers L, Seed KA, Syverson CJ. Pregnancy-related mortality surveillance—United States, 1991–1999. *MMWR Surveill Summ*. 2003 Feb 21;52(2):1-8.
47. Roberts JM, Balk JL, Bodnar LM, Belizan JM, Bergel E, Martinez A. Nutrient involvement in preeclampsia. *J Nutr*. 2003;133:1684S-92S.
48. Morris CD, Jacobson SL, Anand R, Ewell MG, Hauth JC, Curet LB, Catalano PM, Sibai BM, Levine RJ. Nutrient intake and hypertensive disorders of pregnancy: Evidence from a large prospective cohort. *Am J Obstet Gynecol*. 2001;184:643-51.
49. Burton GJ, Jauniaux E. Placental oxidative stress: from miscarriage to preeclampsia. *J Soc Gynecol Investig*. 2004;11:342-52.
50. Merviel P, Carbillon L, Challier JC, Rabreau M, Beaufile M, Uzan S. Pathophysiology of preeclampsia: links with implantation disorders. *Eur J Obstet Gynecol Reprod Biol*. 2004;115:134-47.
51. Dey SK, Lim H, Das SK, Reese J, Paria BC, Daikoku T, et al. Molecular cues to implantation. *Endocrine Rev*. 2004;25:345-73.
52. Fazleabas AT, Kim JJ, Strakova Z. Implantation: Embryonic signals and the modulation of the uterine environment—A review. *Placenta* 2004;18:S26-31.
53. Norwitz ER, Schust D, Fisher SJ. Implantation and the survival of early pregnancy. *N Engl J Med*. 2001;345:1400-8.
54. Prema K, Ramalagshmi BA, Neelakumari S, et al. Serum copper and zinc in pregnancy. *Ind Med Res*. 1980;71:547-53.

55. Cherry FF, Bennett EA, Bazzono GS, et al. Plasma zinc in hypertension/toxaemia and other reproductive variables in adolescent pregnancy. *Am J Clin Nutr.* 1981;34:194-201.
56. Chesters JK. Metabolism and Biochemistry of zinc. In *Clinical biochemistry and nutritional aspects of trace elements*, ed. Prasad AS. New York: Alan R. Liss, 1982.
57. Van den Broek N. Anaemia and micronutrient deficiencies. *Br Med Bull.* 2003;67:149-60.
58. Mahomed K. Iron supplementation in pregnancy. *Cochrane Database Syst Rev.* 2000;(2):CD000117.
59. Bothwell TH. Iron requirements in pregnancy and strategies to meet them. *Am J Clin Nutr.* 2000;72(Suppl.):257S-64S.
60. Casanueva E, Pfeffer F, Drijanski A, et al. Iron and folate status before pregnancy and anemia during pregnancy. *Ann Nutr Metab.* 2003;47:60-3.
61. Allen LH. Multiple micronutrients in pregnancy and lactation: An overview. *Am J Clin Nutr.* 2005;81:1206S-12S.
62. Goldenberg RL, Hauth JC, Andrews WW. Intrauterine infection and preterm delivery. *N Engl J Med.* 2000 May 18;342(20):1500-7.
63. Tomkins A. Nutrition and maternal morbidity and mortality. *Br J Nutr.* 2001 May;85(Suppl. 2):S93-9.
64. Scholl TO. High third-trimester ferritin concentration: associations with very preterm delivery, infection, and maternal nutritional status. *Obstet Gynecol.* 1998 Aug;92(2):161-6.
65. Cox SE, Staalsoe T, Arthur P, Bulmer JN, Tagbor H, Hviid L, Frost C, Riley EM, Kirkwood BR. Maternal vitamin A supplementation and immunity to malaria in pregnancy in Ghanaian primigravids. *Trop Med Int Health* 2005 Dec;10(12):1286-97.
66. Saugstad OD. Hypoxanthine as an indicator of hypoxia: its role in health and disease through free radical production. *Pediatric Research* 1988;23:143-50.
67. Saugstad OD. Update on oxygen radical disease in neonatology. *Current Opinion in Obstetrics and Gynecology* 2001;13(2):147-53.
68. Myatt L, Cui X. Oxidative stress in the placenta. *Histochemistry and Cell Biology* 2004;122(4):369-82.
69. Roberts JM, Hubel CA. Is oxidative stress the link in the two-stage model of pre-eclampsia? *Lancet* 1999;353:788-9.
70. Woods JR Jr, Plessinger MA, Miller RK. Vitamins C and E: missing links in preventing preterm premature rupture of membranes? *American Journal of Obstetrics and Gynecology* 2001;185(1):5-10.
71. Diplock AT, Charleux JL, Crozier-Willi G, Kok FJ, Rice-Evans C, Roberfroid M, et al. Functional food science and defence against reactive oxidative species. *British Journal of Nutrition* 1998;80(Suppl. 1):S77-112.
72. Rumbold A, Duley L, Crowther C, Haslam R. Antioxidants for preventing preeclampsia. *Cochrane Database Syst Rev.* 2005 Oct 19;(4):CD004227.
73. Rush D. Nutrition and maternal mortality in the developing world. *Am J Clin Nutr.* 2000;72:212S-40S.
74. West KP Jr, Katz J, Khatry SK, LeClerq SC, Pradhan EK, Shrestha SR, Connor PB, Dali SM, Christian P, Pokhrel RP, Sommer A. Double blind, cluster randomised trial of low dose supplementation with vitamin A or beta carotene on mortality related to pregnancy in Nepal. The NNIPS-2 Study Group. *BMJ* 1999;318:570-5.
75. de Pee S, Bloem MW, Sari M, Kiess L, Yip R, Kosen S. The high prevalence of low hemoglobin concentration among Indonesian infants aged 3-5 months is related to maternal anemia. *J Nutr.* 2002;132:2215-21.
76. Kaneshige E. Serum ferritin as an assessment of iron stores and other hematologic parameters during pregnancy. *Obstet Gynecol.* 1981;57:238-42.
77. Colomer J, Colomer C, Gutierrez D, et al. Anaemia during pregnancy as a risk factor for infant iron deficiency: report from the Valencia Infant Anaemia Cohort (VIAC) study. *Paediatric and Perinatal Epidemiology* 1990;4:196-204.
78. de Benaze C, Galan P, Wainer R, Hercberg S. Prevention of iron deficiency during gestation by early supplementation: a controlled study. *Rev Epidemiol Sante Publ.* 1989;27:109-19.
79. Preziosi P, Prual A, Galan P, Daouda H, Boureima H, Hercberg S. Effect of iron supplementation on the iron status of pregnant women: consequences for newborns. *Am J Clin Nutr* 1997;66:1178-82.
80. Valero De Bernabe J, Soriano T, Albaladejo R, Juarranz M, Calle ME, Martinez D, Dominguez-Rojas V. Risk factors for low birth weight: a review. *Eur J Obstet Gynecol Reprod Biol.* 2004;116:3-15.
81. Starfield B, Shapiro S, McCormick M, Bross D. Mortality and morbidity in infants with intrauterine growth retardation. *J Pediatr.* 1982;101:978-83.
82. Ashworth, A. Effects of intrauterine growth retardation on mortality and morbidity in infants and young children. *Eur J Clin Nutr.* 1998;52:S34-41.
83. Huttly SR, Morris SS, Pisani V. Prevention of diarrhoea in young children in developing countries. *Bull World Health Organ.* 1997;75:163-74.

84. Delgado HL, et al., Relationship of maternal and infant nutrition to infant growth. *Early Hum Dev.* 1982;6:273-86.
85. Ahn CH, MacLean WC Jr. Growth of the exclusively breast-fed infant. *Am J Clin Nutr.* 1980;33:183-92.
86. Neumann CG, Harrison GG. Onset and evolution of stunting in infants and children. Examples from the Human Nutrition Collaborative Research Support Program. Kenya and Egypt studies. *Eur J Clin Nutr.* 1994;48:S90-102.
87. Henriksen T. Foetal nutrition, foetal growth restriction and health later in life. *Acta Paediatr Suppl.* 1999;88:4-8.
88. Woods DL, Malan AF, Heese HV. Patterns of retarded fetal growth. *Early Hum Dev.* 1979;3:257-62.
89. Bhatia BD, Agarwal KN, Jain NP. Developmental assessment of intrauterine growth retarded babies of varying maternal etiology. *Indian J Pediatr.* 1990;57:99-104.
90. Hack M. Effects of intrauterine growth retardation on mental performance and behavior, outcomes during adolescence and adulthood. *Eur J Clin Nutr.* 1998;52:S65-70.
91. Choudhury N, Gorman KS. Subclinical prenatal iodine deficiency negatively affects infant development in Northern China. *J Nutr.* 2003;133:3162-5.
92. Qian M, Wang D, Watkins WE, Gebiski V, Yan YQ, Li M, Chen ZP. The effects of iodine on intelligence in children: a meta-analysis of studies conducted in China. *Asia Pac J Clin Nutr.* 2005;4:32-42.
93. Armitage JA, Khan IY, Taylor PD, Nathanielsz PW, Poston L. Developmental programming of the metabolic syndrome by maternal nutritional imbalance: how strong is the evidence from experimental models in mammals? *J Physiol.* 2004;561:355-77.
94. Barker DJ, Bull AR, Osmond C, Simmonds SJ. Fetal and placental size and risk of hypertension in adult life. *BMJ* 1990;301:259-62.
95. Phillips DI, Barker DJ, Hales CN, Hirst S, Osmond C. Thinness at birth and insulin resistance in adult life. *Diabetologia* 1994;37:150-154.
96. Martyn CN, Barker DJ, Jespersen S, Greenwald S, Osmond C, Berry C. Growth in utero, adult blood pressure, and arterial compliance. *Br Heart J.* 1995;73, 116-121.
97. Yajnik C. Interactions of perturbations in intrauterine growth and growth during childhood on the risk of adult-onset disease. *Proc Nutr Soc.* 2000;59:257-65.
98. Barker DJ, Martyn CN, Osmond C, Hales CN, Fall CH. Growth in utero and serum cholesterol concentrations in adult life. *BMJ* 1993;307:1524-7.
99. Barker DJP. *Mothers, babies and health in later life.* Edinburgh: Churchill Livingstone, 1998.
100. Ravelli AC, van Der Meulen JH, Osmond C, Barker DJ, Bleker OP. Obesity at the age of 50 y in men and women exposed to famine prenatally. *Am J Clin Nutr.* 1999;70:811-6.
101. Roseboom TJ, van der Meulen JH, Ravelli AC, Osmond C, Barker DJ, Bleker OP. Effects of prenatal exposure to the Dutch famine on adult disease in later life: an overview. *Mol Cell Endocrinol.* 2001;185:93-8.
102. Roseboom TJ, van derMeulen JH, Ravelli AC, van Montfrans GA, Osmond C, Barker DJ, et al. Blood pressure in adults after prenatal exposure to famine. *J Hypertens.* 1999;17:325-30
103. Rasmussen KM. The "fetal origins" hypothesis: challenges and opportunities for maternal and child nutrition. *Annu Rev Nutr.* 2001;21:73-95.
104. Lewis RM, Forhead AJ, Petry CJ, Ozanne SE, Hales CN. Long-term programming of blood pressure by maternal dietary iron restriction in the rat. *Br J Nutr.* 2002;88:283-90.
105. Langley SC, Jackson AA. Increased systolic blood pressure in adult rats induced by fetal exposure to maternal low protein diets. *Clin Sci.* ;86:217-22.
106. Kind KL, Simonetta G, Clifton PM, Robinson JS, Owens JA. Effect of maternal feed restriction on blood pressure in the adult guinea pig. *Exp Physiol.* 2002;87:469-77.
107. Gopalakrishnan GS, Gardner DS, Rhind SM, Rae MT, Kyle CE, Brooks AN, et al. Programming of adult cardiovascular function after early maternal undernutrition in sheep. *Am J Physiol Regul Integr Comp Physiol.* 2004;287:R12-R20.
108. Philips DI. Fetal programming of the neuroendocrine response to stress: links between low birth weight and the metabolic syndrome. *Endocr Res.* 2004;30:819-26.
109. Langley-Evans SC, Welham SJ, Jackson AA. Fetal exposure to a maternal low protein diet impairs nephrogenesis and promotes hypertension in the rat. *Life Sci.* 1999;64:965-74.
110. Hinchliffe SA, Lynch MR, Sargent PH, Howard CV, Van VD. The effect of intrauterine growth retardation on the development of renal nephrons. *Br J Obstet Gynaecol.* 1992;99:296-301.
111. Manalich R, Reyes L, Herrera M, Melendi C, Fundora I. Relationship between weight at birth and the number and size of renal glomeruli in humans: a histomorphometric study. *Kidney Int.* 2000;58:770-73.

112. Goodfellow J, Bellamy MF, Gorman ST, Brownlee M, Ramsey MW, Lewis MJ, et al. Endothelial function is impaired in fit young adults of low birth weight. *Cardiovasc. Res.* 1998;40:600-6.

113. Leeson CP, Kattenhorn M, Morley R, Lucas A, Deanfield JE. Impact of low birth weight and cardiovascular risk factors on endothelial function in early adult life. *Circul.* 2001;103:1264-8.

114. Desai M, Khorram O, Gayle DA, Ross MG. Elevated endothelin-1 and hypertension in adult offspring of intrauterine growth restricted newborns. *Pediatric Research* 2005;58:1071-2.

115. Khorram O, Momeni M, Desai M, Ross MG. Vascular structural changes in hypertensive offspring of nutrient restricted pregnant rats. *Pediatric Research* 2005;58:1112.

116. Burns SP, Desai M, Cohen RD, Hales CN, Iles RA, Germain JP, et al. Gluconeogenesis, glucose handling, and structural changes in livers of the adult offspring of rats partially deprived of protein during pregnancy and lactation. *J Clin. Invest.* 1997;100:1768-74.

117. Ross MG, DeSai M. Gestational programming: population survival effects of drought and famine during pregnancy. *Am J Physiol Regul Integr Comp Physiol.* 2005;288:R25-33.

CHAPTER 2

NUTRITIONAL STATUS AND BEHAVIORS OF PREGNANT WOMEN IN THE U.S.

1. Subcommittee on Nutritional Status and Weight Gain During Pregnancy, Food and Nutrition Board, U.S. Institute of Medicine/ National Academy of Sciences. *Nutrition During Pregnancy.* Washington, DC: National Academy Press, 1990.

2. WHO Collaborative Study. Maternal anthropometry and pregnancy outcomes. *Bulletin of the World Health Organization* 1995; 73(Suppl.):1-98.

3. Kramer MS, Seguin L, Lydon J, Goulet L. Socio-economic disparities in pregnancy outcome: why do the poor fare so poorly? *Paediatr Perinat Epidemiol.* 2000 Jul;14(3):194-210.

4. Hendler I, Goldenberg RL, Mercer BM, Iams JD, Meis PJ, Moawad AH, MacPherson CA, Caritis SN, Miodovnik M, Menard KM, Thurnau GR, Sorokin Y. The Preterm Prediction Study: association between maternal body mass index and spontaneous and indicated preterm birth. *Am J Obstet Gynecol.* 2005 Mar;192(3):882-6.

5. Robinson HE, O'Connell CM, Joseph KS, McLeod NL. Maternal outcomes in pregnancies complicated by obesity. *Obstet Gynecol.* 2005 Dec;106(6):1357-64.

6. Rosenberg TJ, Garbers S, Lipkind H, Chiasson MA. Maternal obesity and diabetes as risk factors for adverse pregnancy outcomes: differences among 4 racial/ethnic groups. *Am J Public Health* 2005 Sep;95(9):1545-51.

7. Dietl J. Maternal obesity and complications during pregnancy. *J Perinat Med.* 2005;33(2):100-5.

8. Siega-Riz AM, Evenson KR, Dole N. Pregnancy-related weight gain—a link to obesity? *Nutr Rev.* 2004 Jul;62(7 Pt 2):S105-11.

9. Werler MM, Louik C, Shapiro S, Mitchell AA. Prepregnant weight in relation to risk of neural tube defects. *JAMA* 1996 Apr 10;275(14):1089-92.

10. Shaw GM, Velie EM, Schaffer D. Risk of neural tube defect-affected pregnancies among obese women. *JAMA* 1996 Apr 10;275(14):1093-6.

11. Nohr EA, Bech BH, Davies MJ, Frydenberg M, Henriksen TB, Olsen J. Prepregnancy obesity and fetal death: a study within the Danish National Birth Cohort. *Obstet Gynecol.* 2005 Aug;106(2):250-9.

12. See <http://www.cdc.gov/pednss/index.htm>.

13. Wells CS, Schwalberg R, Noonan G, Gabor V. Factors influencing inadequate and excessive weight gain in pregnancy: Colorado, 2000-2002. *Matern Child Health J.* 2006 Jan;10(1):55-62.

14. Rasmussen K. Is There a Causal Relationship between Iron Deficiency or Iron-Deficiency Anemia and Weight at Birth, Length of Gestation and Perinatal Mortality? *J Nutr.* 2001 Feb;131(2S-2):590S-601S.

15. Gabbe SG, Graves CR. Management of diabetes mellitus complicating pregnancy. *Obstet Gynecol.* 2003;102:857-68.

16. Korenbrot CC, Steinberg A, Bender C, Newberry S. Preconception care: a systematic review. *Matern Child Health J.* 2002;6:75-88.

17. Aerts L, Van Assche FA. Intra-uterine transmission of disease. *Placenta* 2003;24:905-11.

18. Martin JA, Hamilton BE, Sutton PD, Ventura SJ, Menacker F, Munson ML. Births: final data for 2002. *Natl Vital Stat Rep.* 2003 Dec 8;52(10):1-114.

19. Rodgers BD, Rodgers DE. Efficacy of preconception care of diabetic women in a community setting. *J Reprod Med.* 1996 Jun;41(6):422-6.

20. Siega-Riz AM, Bodnar LM, Savitz DA. What are pregnant women eating? Nutrient and food group differences by race. *Am J Obstet Gynecol.* 2002 Mar;186(3):480-6.
21. See <http://www.cpc.unc.edu/projects/pin/publications/index.html>.
22. Institute of Medicine. *Dietary reference intakes for energy, carbohydrate, fiber, fat, fatty acids, cholesterol, protein, and amino acids.* Washington DC: National Academies Press, 2005.
23. Butte NF, Wong WW, Treuth MS, Ellis KJ, O'Brian Smith E. Energy requirements during pregnancy based on total energy expenditure and energy deposition. *Am J Clin Nutr.* 2004 Jun;79(6):1078-87.
24. See <http://www.fao.org/DOCREP/MEETING/004/M2833E/M2833E00.HTM>.
25. International Society for the Study of Fatty Acids and Lipids. Workshop on the Essentiality of and Recommended Dietary Intakes for Omega-6 and Omega-3 Fatty Acids. April 7-9, 1999. <http://www.issfal.org.uk/Welcome/AdequateIntakes.asp>.
26. McGregor JA, Allen KG, Harris MA, Reece M, Wheeler M, French JJ, Morrison J. The omega-3 story: nutritional prevention of preterm birth and other adverse pregnancy outcomes. *Obstet Gynecol Surv.* 2001 May;56(5 Suppl. 1):S1-13.
27. Ervin RB, Wright JD, Wang CY, Kennedy-Stephenson J. Dietary intake of fats and fatty acids for the United States population: 1999-2000. *Adv Data* 2004 Nov 8;(348):1-6.
28. Institute of Medicine. *Dietary reference intakes for Vitamin A, Vitamin K, Arsenic, Boron, Chromium, Copper, Iodine, Iron, Manganese, Molybdenum, Nickel, Silicon, Vanadium, and Zinc.* Washington DC: National Academies Press. 2000.
29. Institute of Medicine. *Dietary reference intakes Vitamin C, Vitamin E, Selenium, and Carotenoids.* Washington DC: National Academies Press, 2000.
30. Institute of Medicine. *Dietary reference intakes for Thiamin, Riboflavin, Niacin, Vitamin B6, Folate, Vitamin B12, Pantothenic Acid, Biotin, and Choline.* Washington DC: National Academies Press, 1998.
31. Institute of Medicine. *Dietary reference intakes for Calcium, Phosphorus, Magnesium, Vitamin D, and Fluoride.* Washington DC: National Academies Press, 1997.
32. Cohen GR, Curet LB, Levine RJ, Ewell MG, Morris CD, Catalano PM, Clokey D, Klebanoff MA. Ethnicity, nutrition, and birth outcomes in nulliparous women. *Am J Obstet Gynecol.* 2001 Sep;185(3):660-7.
33. Harville EW, Schramm M, Watt-Morse M, Chantala K, Anderson JJ, Hertz-Picciotto I. Calcium intake during pregnancy among white and African-American pregnant women in the United States. *J Am Coll Nutr.* 2004 Feb;23(1):43-50.
34. Yu SM, Keppel KG, Singh GK, Kessel W. Preconceptional and prenatal multivitamin-mineral supplement use in the 1988 National Maternal and Infant Health Survey. *Am J Public Health* 1996 Feb;86(2):240-2.
35. Vahratian A, Siega-Riz AM, Savitz DA, Thorp JM Jr. Multivitamin use and the risk of preterm birth. *Am J Epidemiol.* 2004 Nov 1;160(9):886-92.
36. Jasti S, Siega-Riz AM, Cogswell ME, Hartzema AG, Bentley ME. Pill count adherence to prenatal multivitamin/mineral supplement use among low-income women. *J Nutr.* 2005 May;135(5):1093-101.
37. Popkin BM, Siega-Riz AM, Haines PS. A comparison of dietary trends among racial and socioeconomic groups in the United States. *N Engl J Med.* 1996 Sep 5;335(10):716-20.
38. Subcommittee on Nutritional Status and Weight Gain During Pregnancy, Food and Nutrition Board, U.S. Institute of Medicine/ National Academy of Sciences. *Nutrition During Pregnancy.* Washington, DC: National Academy Press, 1990: 45.
39. Siega-Riz AM, Herrmann TS, Savitz DA, Thorp JM. Frequency of eating during pregnancy and its effect on preterm delivery. *Am J Epidemiol.* 2001 Apr 1;153(7):647-52.
40. Herrmann TS, Siega-Riz AM, Hobel CJ, Aurora C, Dunkel-Schetter C. Prolonged periods without food intake during pregnancy increase risk for elevated maternal corticotropin-releasing hormone concentrations. *Am J Obstet Gynecol.* 2001 Aug;185(2):403-12.
41. Satia JA, Galanko JA, Siega-Riz AM. Eating at fast-food restaurants is associated with dietary intake, demographic, psychosocial and behavioral factors among African Americans in North Carolina. *Public Health Nutr.* 2004 Dec;7(8):1089-96.
42. U.S. Department of Agriculture (USDA). USDA Continuing Survey of Food Intakes by Individuals, 1994-1996. Washington, DC: USDA Economic Research Service, 1997.
43. McCrory MA, Fuss PJ, Hays NP, Vinken AG, Greenberg AS, Roberts SB. Overeating in America: association between restaurant food consumption and body fatness in healthy adult men and women ages 19 to 80. *Obesity Research* 1999;7:564-71.
44. Ma Y, Bertone ER, Stanek EJ III, Reed GW, Hebert JR, Cohen NL. Association between eating patterns and obesity in a free-living U.S. adult population. *American Journal of Epidemiology* 2003;158:85-92.

45. Jeffery RW, French SA. Epidemic obesity in the United States: are fast foods and television viewing contributing? *American Journal of Public Health* 1998;88(2):277-80.
46. Lin B, Frazão E, Guthrie J. Away-From-Home-Foods Increasingly Important to Quality of American Diet. *Agriculture Information Bulletin* No. 749. Washington, DC: Economic Research Service, U.S. Department of Agriculture, 1999.
47. Clemens LHE, Slawson DK, Klesges RC. The effect of eating out on quality of diet in premenopausal women. *Journal of the American Dietetic Association* 1999;99:442-4.
48. Haines PS, Hungerford DW, Popkin BM, Guilkey DK. Eating patterns and energy and nutrient intakes of U.S. women. *Journal of the American Dietetic Association* 1992;92:698-704, 707.
49. Guenther PM, Perloff BP. *Effects of Procedural Differences between 1977 and 1987 in the Nationwide Food Consumption Survey on Estimates of Food and Nutrient Intakes: Results from the USDA 1988 Bridging Study*. NFCS Report No. 87-M-1. Hyattsville, MD: U.S. Department of Agriculture, 1990.
50. Shannon BM, Parks SC. Fast foods: a perspective on their nutritional impact. *Journal of the American Dietetic Association* 1980;76:242-7.
51. Nielsen SJ, Siega-Riz AM, Popkin BM. Trends in food locations and sources among adolescents and young adults. *Prev Med*. 2002 Aug;35(2):107-13.
52. Corbett RW, Ryan C, Weinrich SP. Pica in pregnancy: does it affect pregnancy outcomes? *MCN Am J Matern Child Nurs*. 2003 May-Jun;28(3):183-9.
53. Rose EA, Porcerelli JH, Neale AV. Pica: common but commonly missed. *J Am Board Fam Pract*. 2000 Sep-Oct;13(5):353-8.
54. Horner RD, Lackey CJ, Kolasa K, Warren K. Pica practices of pregnant women. *J Am Diet Assoc*. 1991 Jan;91(1):34-8.
55. Edwards CH, Johnson AA, Knight EM, Oyemade UJ, Cole OJ, Westney OE, Jones S, Laryea H, Westney LS. Pica in an urban environment. *J Nutr*. 1994 Jun;124(6 Suppl.):954S-62S.
56. Smulian JC, Motiwala S, Sigman RK. Pica in a rural obstetric population. *South Med J*. 1995 Dec;88(12):1236-40.
57. Simpson E, Mull JD, Longley E, East J. Pica during pregnancy in low-income women born in Mexico. *West J Med*. 2000 Jul;173(1):20-4.
58. Corbett RW, Ryan C, Weinrich SP. Pica in pregnancy: does it affect pregnancy outcomes? *MCN Am J Matern Child Nurs*. 2003 May-Jun;28(3):183-9.
59. Cooksey NR. Pica and olfactory craving of pregnancy: how deep are the secrets? *Birth* 1995 Sep;22(3):129-37.

CHAPTER 3

PRENATAL NUTRITIONAL INTERVENTIONS: EVIDENCE OF EFFECTIVENESS

1. Villar J, Merialdi M, Gulmezoglu AM, Abalos E, Carroli G, Kulier R, de Onis M. Nutritional interventions during pregnancy for the prevention or treatment of maternal morbidity and preterm delivery: an overview of randomized controlled trials. *J Nutr*. 2003 May;133(5 Suppl. 2):1606S-25S.
2. Kramer MS. Nutritional advice in pregnancy. *Cochrane Database Syst Rev*. 2000;(2):CD000149.
3. Kramer MS. Balanced protein/energy supplementation in pregnancy. *Cochrane Database Syst. Rev*. 2000;(2):CD000032.
4. Kramer MS. Isocaloric balanced protein supplementation in pregnancy. *Cochrane Database Syst. Rev*. 2000;(2):CD000118.
5. Kramer MS. High protein supplementation in pregnancy. *Cochrane Database Syst. Rev*. 2000;(2):CD000105.
6. Kramer MS. Energy/protein restriction for high weight-for-height or weight gain during pregnancy. *Cochrane Database Syst. Rev*. 2000;(2):CD000080.
7. Duley L, Henderson-Smart D. Reduced salt intake compared to normal dietary salt, or high intake, in pregnancy. *Cochrane Database Syst. Rev*. 2000;(2):CD001687.
8. Hofmeyr GJ, Atallah A, Duley L. Calcium supplementation during pregnancy for preventing hypertensive disorders and related problems (Cochrane Review). In *The Cochrane Library*, Issue 4: CD001059. Update Software, Oxford, 2002.
9. Mahomed K. Iron supplementation in pregnancy. *Cochrane Database Syst. Rev*. 2000;(2):CD000117.
10. Mahomed, K. Folate supplementation in pregnancy. *Cochrane Database Syst. Rev*. 2000;(2):CD000183.
11. Mahomed, K. Iron and folate supplementation in pregnancy. *Cochrane Database Syst. Rev*. 2000;(2):CD001135.
12. Makrides M, Crowther CA. Magnesium supplementation in pregnancy. *Cochrane Database Syst. Rev*. 2000;(2):CD000937.
13. Duley L. Prophylactic fish oil in pregnancy. In *Pregnancy and Childbirth Module of the Cochrane Database of Systematic Reviews*, eds. MW Enkin, MJ Keirse, MJ Renfrew, and JP Neilson. London, UK: British Medical Journal Publishing Group, 1995.

14. Mahomed K. Zinc supplementation in pregnancy. *Cochrane Database Syst. Rev.* 2000;(2):CD000230.
15. Merialdi M, Carroli G, Villar J, Abalos E, Gulmezoglu AM, Kulier R, de Onis M. Nutritional interventions during pregnancy for the prevention or treatment of impaired fetal growth: an overview of randomized controlled trials. *J Nutr.* 2003 May;133(5 Suppl. 2):1626S-31S.
16. Mahomed K, Gulmezoglu AM. Vitamin D supplementation in pregnancy. *Cochrane Database Syst. Rev.* 2002;(2):CD000228.
17. Chappell LC, Seed PT, Briley AL, Kelly FJ, Lee R, Hunt BJ, Parmar K, Bewley SJ, Shennan AH, Steer PJ, Poston L. Effect of antioxidants on the occurrence of preeclampsia in women at increased risk: a randomised trial. *Lancet* 1999;354:810-816.
18. Lumley J, Watson L, Watson M, Bower C. Periconceptional supplementation with folate and/or multivitamins for preventing neural tube defects. *Cochrane Database Syst. Rev.* 2001;(3):CD001056.
19. MRC Vitamin Study Research Group. Prevention of neural tube defects: results of the Medical Research Council Vitamin Study. *Lancet* 1991;338:131-137.
20. Laurence KM, James N, Miller MH, Tennant GB, Campbell H. Double-blind randomised controlled trial of folate treatment before conception to prevent recurrence of neural-tube defects. *Br Med J.* 1981;282:1509-1511.
21. Kirke PN, Daly LE, Elwood JH for the Irish Vitamin Study Group. A randomised trial of low dose folic acid to prevent neural tube defects. *Arch Dis Child* 1992;67:1442-1446.
22. Czeizel AE, Dudás I. Prevention of the first occurrence of neural tube defects by periconceptional vitamin supplementation. *New Engl J Med.* 1992;327:1832-1835.
23. Gabbe SG, Graves CR. Management of diabetes mellitus complicating pregnancy. *Obstet Gynecol.* 2003;102:857-68.
24. Korenbrot CC, Steinberg A, Bender C, Newberry S. Preconception care: a systematic review. *Matern Child Health J.* 2002 Jun;6(2):75-88.
25. Levine RJ, Hauth JC, Curet LB, Sibai BM, Catalano PM, Morris CD, DerSimonian R, Esterlitz JR, Raymond EG, Bild DE, Clemens JD, Cutler JA. Trial of calcium to prevent preeclampsia. *N. Engl. J. Med.* 1997;337:69-76.
26. West KP Jr, Katz J, Khattry SK, LeClerq SC, Pradhan EK, Shrestha SR, Connor PB, Dali SM, Christian P, Pokhrel RP, Sommer A. Double blind, cluster randomised trial of low dose supplementation with vitamin A or beta carotene on mortality related to pregnancy in Nepal. The NNIPS-2 Study Group. *BMJ* 1999;318:570-5.
27. Suharno D, West CE, Muhilal, Karyadi D, Hautvast JG. Indonesia. *Lancet* 1993;342:1325-8.
28. Semba RD, Kumwenda N, Taha TE, Mtimavalyen L, Broadhead R, Garrett E, Miotti PG, Chipangwi JD. Impact of vitamin A supplementation on anaemia and plasma erythropoietin concentrations in pregnant women: a controlled clinical trial. *Eur. J. Haematol.* 2001;66:389-95.
29. Minihane AM, Fairweather-Tait SJ. Effect of calcium supplementation on daily nonheme-iron absorption and long-term iron status. *Am. J. Clin. Nutr.* 1998;68:96-102.
30. Mahomed K, James DK, Golding J, McCabe R. Zinc supplementation during pregnancy: a double blind randomised controlled trial. *BMJ* 1989;299:826-30.
31. Preziosi P, Prual A, Galan P, Daouda H, Boureima H, Hercberg S. Effect of iron supplementation on the iron status of pregnant women: consequences for newborns. *Am J Clin Nutr.* 1997;66:1178-82.
32. Hemminki E, Rimpela U. A randomized comparison of routine versus selective iron supplementation during pregnancy. *J Am Coll Nutr.* 1991;10:3-10.
33. Schmidt MK, Muslimatun S, Schultink W, West CE, Hautvast JG. Randomised double-blind trial of the effect of vitamin A supplementation of Indonesian pregnant women on morbidity and growth of their infants during the first year of life. *Eur J Clin Nutr.* 2002;56:338-46.
34. Conlisk AJ, Barnhart HX, Martorell R, Grajeda R, Stein AD. Maternal and child nutritional supplementation are inversely associated with fasting plasma glucose concentration in young Guatemalan adults. *J Nutr.* 2004;134:890-7.
35. Joos SK, Pollitt E, Mueller WH, Albright DL. The Bacon Chow study: maternal nutritional supplementation and infant behavioral development. *Child Dev.* 1983;54:669-76.
36. Schmidt MK, Muslimatun S, West CE, Schultink W, Hautvast JG. Mental and psychomotor development in Indonesian infants of mothers supplemented with vitamin A in addition to iron during pregnancy. *Br J Nutr.* 2004;91:279-86.
37. Tamura T, Goldenberg RL, Ramey SL, Nelson KG, Chapman VR. Effect of zinc supplementation of pregnant women on the mental and psychomotor development of their children at 5 y of age. *Am J Clin Nutr.* 2003;77:1512-6.
38. Helland IB, Smith L, Saarem K, Saugstad OD, Drevon CA. Maternal supplementation with very-long-chain n-3 fatty acids during pregnancy and lactation augments children's IQ at 4 years of age. *Pediatrics* 2003;111:e39-44.

39. Abrams B. Preventing low birth weight: does WIC work? A review of evaluations of the special supplemental food program for women, infants, and children. *Ann NY Acad Sci.* 1993 Mar 15;678:306-16.
40. See <http://www.fns.usda.gov/wic/>.
41. Institute of Medicine. *WIC food packages: Time for change.* Washington DC: National Academies Press, 2005.
42. Besharov DJ, Germanis P. Evaluating WIC. *Eval Rev.* 2000 Apr;24(2):123-90.
43. Gordon A, Nelson L. *Characteristics and outcomes of WIC participants and non-participants: Analysis of the 1988 National Maternal and Infant Health Survey.* Alexandria, VA: U.S. Department of Agriculture, Food and Nutrition Service, 1995.
44. Brien MJ, Swann CA. "Prenatal WIC participation and infant health: Selection and maternal fixed effects." Discussion paper 295; Thomas Jefferson Center, University of Virginia, 1997.
45. Rossi PH. *Feeding the poor: Assessing federal food aid.* Washington DC: American Enterprise Institute, 1988.
7. Carlson M, McLanahan S, England P. Union formation in fragile families. *Demography* 2004;41:237-61.
8. Zeitlin JA, Saurel-Cubizolles MJ, Ancel PY, EUROPOP Group. Marital status, cohabitation, and risk of preterm birth in Europe: where births outside marriage are common and uncommon. *Paediatr Perinat Epidemiol.* 2002;16(2):124-30.
9. Luo ZC, Wilkins R, Kramer MS, Fetal and Infant Health Study Group of the Canadian Perinatal Surveillance System. Disparities in pregnancy outcomes according to marital and cohabitation status. *Obstet Gynecol.* 2004;103(6):1300-7.
10. Putnam RD, Leonardi R, Nanetti RY. *Making Democracy Work: Civic Traditions in Modern Italy.* Princeton, NJ: Princeton University Press, 1993.
11. Kawachi I, Kennedy BP, Lochner K, Prothrow-Stith D. Social capital, income inequality, and mortality. *Am J Public Health* 1997;87:1491-8.
12. Buka SL, Brennan RT, Rich-Edwards JW, Raudenbush SW, Earls F. Neighborhood support and the birth weight of urban infants. *Am J Epidemiol.* 2003 Jan 1;157(1):1-8.

CHAPTER 4

RETHINKING NUTRITION AND INFANT MORTALITY: THE CONTEXT OF RELATIONALITY OVER THE LIFE COURSE

1. David R. *The Case for Relationality: The Historical Framework of Policy and Practice on Infant Mortality.* Washington DC: Joint Center for Political and Economic Studies, 2007.
2. Mathews TJ, Menacker F, MacDorman MF; Centers for Disease Control and Prevention, National Center for Health Statistics. Infant mortality statistics from the 2002 period: linked birth/infant death data set. *Natl Vital Stat Rep.* 2004 Nov 24;53(10):1-29.
3. Raatikainen K, Heiskanen N, Heinonen S. Marriage still protects pregnancy. *BJOG* 2005 Oct;112(10):1411-6.
4. Waldron I, Hughes ME, Brooks TL. Marriage protection and marriage selection – prospective evidence for reciprocal effects of marital status and health. *Soc Sci Med.* 1996;43:113-23.
5. Bennett T. Marital status and infant health outcomes. *Soc Sci Med.* 1992 Nov;35(9):1179-87.
6. McLanahan S, Garfinkel I, Brooks-Gunn J, Zhao H, Johnson W Jr, Rich L, et al. "Unwed fathers and fragile families." Working paper # 98-12. Princeton NJ: Center for Research on Child Wellbeing, 1998. <http://crcw.princeton.edu/workingpapers/wp98-12-FF-McLanahan.pdf>.
13. Collins JW Jr, David RJ, Handler A, Wall S, Andes S. Very low birthweight in African American infants: the role of maternal exposure to interpersonal racial discrimination. *Am J Public Health* 2004;94:2132-8.
14. Mustillo S, Krieger N, Gunderson EP, Sidney S, McCreath H, Kiefe CI. Self-reported experiences of racial discrimination and Black-White differences in preterm and low-birthweight deliveries: the CARDIA Study. *Am J Public Health* 2004;94:2125-31.
15. Rosenberg L, Palmer JR, Wise LA, Horton NJ, Corwin MJ. Perceptions of racial discrimination and the risk of preterm birth. *Epidemiology* 2002;13:646-52.
16. Collins JW Jr, David RJ, Symons R, Handler A, Wall SN, Dwyer L. Low-income African-American mothers' perception of exposure to racial discrimination and infant birth weight. *Epidemiology* 2000;11:337-9.
17. Jones CP. Levels of racism: A theoretic framework and a gardener's tale. *Am J Public Health* 2000;90:1212-5.
18. Polednak AP. Trends in urban black infant mortality, by degree of residential segregation. *Am J Public Health* 1996;86:723-6.
19. LaVeist TA. Segregation, poverty, and empowerment: Health consequences for African Americans. *Milbank Q.* 1993;71:41-64.
20. Lobel M, Dunkel-Schetter C, Scrimshaw SCM. Prenatal maternal stress and prematurity: a prospective study of socio-economically disadvantaged women. *Health Psychol.* 1992;11:32-40.

21. Hedegaard M, Henriksen TB, Sabroe S, Secher NJ. Psychological distress in pregnancy and preterm delivery. *BMJ* 1993;307:234-9.
22. Paarlberg KM, Vingerhoets JJM, Passchier J, Dekker GA, Van Geijn HP. Psychosocial factors and pregnancy outcome: A review with emphasis on methodological issues. *J Psychosomatic Res.* 1995;39:653-95.
23. Copper RL, Goldenberg RL, Das A, Elder N, Swain M, Norman G, Ramsey R, Cotroneo P, Collin BA, Johnson F. The preterm prediction study: maternal stress is associated with spontaneous preterm birth at less than 35 weeks' gestation. *Am J Obstet Gynecol.* 1996;175:1286-92.
24. Chrousos GP, Gold PW. The concepts of stress and stress system disorders. Overview of physical and behavioral homeostasis. *JAMA* 1992;267:1244-52.
25. Black PH, Garbutt LD. Stress, inflammation and cardiovascular disease. *J Psychosom Res.* 2002;52:1-23.
26. Lockwood CJ, Kuczynski E. Risk stratification and pathological mechanisms in preterm delivery. *Paediatr Perinat Epidemiol.* 2001 Jul;15(Suppl. 2):78-89.
27. Challis JRG. Mechanism of Parturition and Preterm Labor. *Obstet Gynecol Survey* 2000;55(10):650-60.
28. Hobel CJ, Dunkel-Schetter C, Roesch S. Maternal stress as a signal to the fetus. *Prenat Neonat Med.* 1998;3:116-20.
29. Dudley DJ. Hormonal pathways of preterm birth. *Am J Obstet Gynecol.* 1999;180:S251-6.
30. Wadhwa PD, Culhane JF, Rauh V, Barve SS, Hogan V, Sandman CA, Hobel CJ, Chicz-DeMet A, Dunkel Schetter C, Garite TJ, Glynn L. Stress, infection, and preterm birth: a biobehavioural perspective. *Paed Perinat Epidemiol.* 2001;15:17-29.
31. McLean M, Bisits A, Davies J, Walters W, Hackshaw A, De Voss K, Smith R. Predicting risk of preterm delivery by second-trimester measurement of maternal plasma corticotropin-releasing hormone and alpha-fetoprotein concentrations. *Am J Obstet Gynecol.* 1999 Jul;181(1):207-15.
32. Leung TN, Chung TK, Madsen G, McLean M, Chang AM, Smith R. Elevated mid-trimester maternal corticotrophin-releasing hormone levels in pregnancies that delivered before 34 weeks. *Br J Obstet Gynaecol.* 1999 Oct;106(10):1041-6.
33. Holzman C, Jetton J, Siler-Khodr T, Fisher R, Rip T. Second trimester corticotropin-releasing hormone levels in relation to preterm delivery and ethnicity. *Obstet Gynecol.* 2001 May;97(5 Pt 1):657-63.
34. Hobel CJ, Dunkel-Schetter C, Roesch SC, Castro LC, Arora CP. Maternal plasma corticotropin-releasing hormone associated with stress at 20 weeks' gestation in pregnancies ending in preterm delivery. *Am J Obstet Gynecol.* 1999 Jan;180(1 Pt 3):S257-63.
35. Sandman CA, Glynn L, Schetter CD, Wadhwa P, Garite T, Chicz-Demet A, Hobel C. Elevated maternal cortisol early in pregnancy predicts third trimester levels of placental corticotropin releasing hormone (CRH): Priming the placental clock. *Peptides* 2005 Nov 21; [Epub ahead of print].
36. Wadhwa PD, Culhane JF, Rauh V, et al. Stress and preterm birth: neuroendocrine, immune/inflammatory, and vascular mechanisms. *Matern Child Health J.* 2001;5:119-25.
37. Culhane JF, Rauh V, McCollum KF, Hogan VK, Agnew K, Wadhwa PD. Maternal stress is associated with bacterial vaginosis in human pregnancy. *Matern Child Health J.* 2001 Jun;5(2):127-34.
38. Culhane JF, Rauh V, McCollum KF, Elo IT, Hogan V. Exposure to chronic stress and ethnic differences in rates of bacterial vaginosis among pregnant women. *Am J Obstet Gynecol.* 2002 Nov;187(5):1272-6.
39. McCubbin JA, Lawson EJ, Cox S, Sherman JJ, Norton JA, Read JA. Prenatal maternal blood pressure response to stress predicts birth weight and gestational age: a preliminary study. *Am J Obstet Gynecol.* 1996;175:706-12.
40. Lu MC, Halfon N. Racial and ethnic disparities in birth outcomes: a life-course perspective. *Matern Child Health J.* 2003 Mar;7(1):13-30.
41. Hofferth S. "Race/ethnic differences in father involvement with young children: A conceptual framework and empirical test in two-parent families." Paper presented at the Urban Seminar on Fatherhood. Harvard University, Cambridge, MA, April 1999.
42. Cabrera N, Tamis-LeMonda CS, Bradley R, Hofferth S, Lamb ME. Fatherhood in the twenty-first century. *Child Development* 2000;71:127-36.
43. Jaffee SR, Caspi A, Moffitt TE, Taylor A, Dickson N. Predicting early fatherhood and whether young fathers live with their children: Prospective findings and policy reconsiderations. *J Child Psychol Psychiat.* 2001;42:803-15.
44. Aronson RE, Whitehead TL, Baber WL. Challenges to masculine transformation among urban low-income African American males. *Am J Public Health* 2003;93:732-41.
45. Barker DJP. Fetal and infant origins of adult disease. *Br Med J.* 1990;301:1111.

46. Powers C, Hertzman C. Social and biological pathways linking early life and adult disease. *Brit Med Bull.* 1997;53:210-21.
47. Soumi SJ. Early determinants of behaviour: evidence from primate studies. *Br Med Bull.* 1997;53:170-84.
48. Meaney MJ, Aitken S, Sharma S, Viau V, Sarrieau A. Postnatal handling increases hippocampal type II glucocorticoid receptors and enhances adrenocortical negative-feedback efficacy in the rat. *J Neuroendocrinol.* 1989;5:597-604.
49. Seckl JR. Physiologic programming of the fetus. *Emerging Concepts in Perinatal Endocrinology* 1998;25:939-62.
50. Sapolsky RM. Social subordination as a marker of hypercortisolism: Some unexpected subtleties. *Ann NY Acad Sci.* 1995;771:626-39.
51. Gunnar MR, Nelson CA. Event-related potentials in year-old infants: relations with emotionality and cortisol. *Child Dev.* 1994;65:80-94.
52. Heim C, Newport DJ, Heit S, Graham YP, Wilcox M, Bonsall R, Miller AH, et al. Pituitary-adrenal and autonomic responses to stress in women after sexual and physical abuse in childhood. *JAMA* 2000;284:592-7.
53. Coe CL. Psychosocial factors and psychoneuroimmunology within a lifespan perspective. In *Developmental health and the wealth of nations: social, biological and educational dynamics*, eds. DP Keating and C Hertzman. New York: Guilford Press, 1999: 201-19.
54. McEwen BS. Protective and damaging effects of stress mediators. *N Eng J Med.* 1998;338:171-9
55. Marmot MG, Davey Smith G, Stansfeld S, Patel C, North F, Head J, et al. Inequalities in health twenty years on: the Whitehall II study of British Civil Servants. *Lancet* 1991;337:1387-94.
56. Power C, Matthews S. Origins of health inequalities in a national population sample. *Lancet* 1997b;350:1584-9.
57. Blane D. The life course, the social gradient, and health. In *Social determinants of health*, eds. M Marmot and RG Wilkinson. Oxford: Oxford University Press, 2000: 44-63.
58. Brunner EJ. Toward a new social biology. In *Social epidemiology*, eds. LF Berkman and I Kawachi. Oxford: Oxford University Press, 2000: 306-31.
59. Sapolsky RM. Social subordination as a marker of hypercortisolism: Some unexpected subtleties. *Ann NY Acad Sci.* 1995;771:626-39.
60. Sapolsky RM, Krey P, McEwen B. Glucocorticoid-sensitive hippocampal neurons are involved in terminating the adrenocortical stress-response. *Proc Natl Acad Sci.* 1984;81:6174-77.
61. Chrousos GP. Stress response and immune function: Clinical implications. *Ann NY Acad Sci.* 2000;917:38-67.
62. Geronimus AT. Black/white differences in the relationship of maternal age to birthweight: a population-based test of the weathering hypothesis. *Soc Sci Med.* 1996;42:589-97.
63. American Academy of Pediatrics Work Group on Breast-feeding. Breastfeeding and the use of human milk. *Pediatrics* 1997;100(6):1035-37.
64. U.S. Department of Health and Human Services. *Healthy People 2010*. 2nd ed. With Understanding and Improving Health and Objectives for Improving Health. 2 vols. Washington, DC: U.S. Government Printing Office, November 2000.
65. Philipp B, Jean-Marie S. *African American Women and Breast-feeding*. Washington DC: Joint Center for Political and Economic Studies, 2007.
66. Lu MC, Lange L, Slusser W, Hamilton J, Halfon N. Provider encouragement of breast-feeding: evidence from a national survey. *Obstet Gynecol.* 2001 Feb;97(2):290-5.
67. Fulkerson JA, Neumark-Sztainer D, Story M. Adolescent and parent views of family meals. *J Am Diet Assoc.* 2006 Apr;106(4):526-32.
68. Collins WA. Relationships and development Family adaptation to individual change. In *Close Relationships and Socioemotional Development*, ed. S Shulman. Norwood, NH: Ablex, 1995: 128-54.
69. Steinberg L. Interdependence in the family: Autonomy, conflict and harmony in the parent-adolescent relationship. In *At the Threshold: The Developing Adolescent*, eds. SS Feldman and GL Elliott. Cambridge, MA: Harvard University Press, 1990: 255-76.
70. Davis JA. Family meals: A thing of the past. *Arch Dis Child* 1995;3:356.
71. MacKenzie M. Is the family meal disappearing? *J Gastronomy* 1993;7:34-45.
72. Wolin SJ, Bennett LA. Family rituals. *Fam Proc.* 1984;23:401-20.
73. Gillman MW, Rifas-Shiman SL, Frazier L, Rockett HRH, Camargo CA, Field AE, et al. Family dinner and diet quality among older children and adolescents. *Arch Fam Med.* 2000;9:235-40.

74. Neumark-Sztainer D, Hannan PJ, Story M, Croll J, Perry CL. Family meal patterns Associations with sociodemographic characteristics and improved dietary intake among adolescents. *J Am Diet Assoc.* 2003;103:317-22.
75. Videon TM, Manning CK. Influences on adolescent eating patterns: The importance of family meals. *J Adolesc Health* 2003;32:365-73.
76. Neumark-Sztainer D, Wall M, Story M, Fulkerson JA. Are family meal patterns associated with disordered eating behaviors among adolescents? *J Adolesc Health* 2004;35:350-9.
77. Nicklas TA, Morales M, Linares A, Yang S, Baranowski T, de Moor C, et al. Children's meal patterns have changed over a 21-year period. The Bogalusa Heart Study. *J Am Diet Assoc.* 2004;104:753-61.
78. Nielsen SJ, Siega-Riz AM, Popkin BM. Trends in food locations and sources among adolescents and young adults. *Prev Med.* 2002 Aug;35(2):107-13.
79. Neumark-Sztainer D, Hannan PJ, Story M, Croll J, Perry C. Family meal patterns: associations with sociodemographic characteristics and improved dietary intake among adolescents. *J Am Diet Assoc.* 2003 Mar;103(3):317-22.
80. Perry C, Kelder S and Komro K. The social world of adolescents Family, peers, schools and the community. In *Promoting the Health of Adolescents New Directions for the 21st Century*, eds. S Millstein, A Peterson, and E Nightengale. New York, NY: Oxford University Press, 1993: 73-96.
81. McGinnis JM, Gootman JA, Kraak VI, eds. Institute of Medicine Food and Nutrition Board. *Food marketing to children: Threat or opportunity?* Washington DC: National Academies Press. 2006.
82. See <http://www.cspinet.org/new/200311101.html>.
83. See http://medialit.med.sc.edu/marketing_food_to_kids.htm.
84. See <http://www.fas.usda.gov/itp/Policy/tradeFAQ.htm#4>.
85. Nord M, Andrews M, Carlson S. *Household food security in the United States, 2003*. Economic Research Service, Food Assistance and Nutrition Research Report No. (FANRR42). Washington DC: U.S. Department of Agriculture, 2004.
86. Anderson SA, ed. Core indicators of nutritional state for difficult-to-sample populations. *J Nutr.* 1990;120:1559S-1600.
87. Rose D, Oliveira V. Nutrient intakes of individuals from food-insufficient households in the United States. *Am J Public Health* 1997;87:1856-61.
88. Townsend MS, Peerson J, Love B, Achterberg C, Murphy SP. Food insecurity is positively related to overweight in women. *J Nutr.* 2001;131:1738-45.
89. Olson CM. Nutrition and health outcomes associated with food insecurity and hunger. *J Nutr.* 1999;129:521S-4.
90. Olson CM, Strawderman MS, Hinton PS, Pearson TA. Gestational weight gain and postpartum behaviors associated with weight change from early pregnancy to 1 y postpartum. *Int J Obes Relat Metab Disord.* 2003;27:117-27.
91. Laraia BA, Siega-Riz AM, Gunderson C, Dole N. Psychosocial factors and socioeconomic indicators are associated with household food insecurity among pregnant women. *J Nutr.* 2006 Jan;136(1):177-82.
92. Nelson K, Brown ME, Lurie N. Hunger in an adult patient population. *JAMA* 1998;279:1211-14.
93. Alaimo K, Briefel RR, Frongillo EA, Olson CM. Food insufficiency exists in the United States: results from the third National Health and Nutrition Examination Survey (NHANES III). *Am J Public Health* 1998;88:419-26
94. See <http://www.healthpolicy.ucla.edu/pubs/publication.asp?pubID=143>.
95. Dallman MF, Pecoraro NC, la Fleur SE. Chronic stress and comfort foods: self-medication and abdominal obesity. *Brain Behav Immun.* 2005 Jul;19(4):275-80.
96. Oliver G, Wardle J. Perceived effects of stress on food choice. *Physiol. Behav.* 1999;66:511-15.
97. Freeman LM, Gil KM. Daily stress, coping and dietary restraint in binge eating. *Int. J. Eat. Disord.* 2004;36:204-12.
98. Schoemaker C, Smit F, Bijl RV, Vollebergh WAM. Bulimia nervosa following psychological and multiple child abuse: support for the self-medication hypothesis in a population-based cohort study. *Int. J. Eat. Disord.* 2002;32:381-8.
99. Epel E, Lapidus R, McEwen B, Brownell K. Stress may add bite to appetite in women: a laboratory study of stress-induced cortisol and eating behavior. *Psychoneuroendocrinology* 2001;26:37-49.
100. Sorensen E, Mincy R, Halpern A. *Redirecting welfare policy toward building stronger families*. Washington DC: Urban Institute, 2000.
101. Lu MC, Jones L, Bond M, Pumpuang M, Maidenberg M, Jones D, et al. Where is the F in MCH? Father involvement in African American families. *Ethn Dis*. In press.

102. Hewlett SA, West C. *The war against parents: What we can do for America's beleaguered moms and dads*. Boston: Houghton Mifflin, 1998.
103. Morehouse Conference on African American Fathers. "Turning the corner on father absence in Black America." Atlanta: Morehouse Research Institute & Institute for American Values, 1999.
104. Wilson WJ. *When work disappears: The world of the new urban poor*. New York: Knopf, 1996.
105. McLanahan S, Sandefur G. *Growing up with a single parent*. Cambridge, MA: Harvard University Press, 1994.
106. Bronfenbrenner U. *Ecology of Human Development: Experiments by Nature and Design*. Cambridge, MA: Harvard University Press, 1979.
107. Bloom D, Sherwood K. "Matching opportunities to obligations: Lessons for child support reform from the Parents' Fair Share Pilot Phase." <http://fatherhood.hhs.gov/pfs94>.
108. Sorensen E. *Obligating dads: Helping low-income noncustodial fathers do more for their children*. Washington DC: Urban Institute, 1999.
109. Wheaton L. *Low-income families and the marriage tax*. Washington DC: Urban Institute, 1998.
110. Jones L, Lu MC, Ferre C, Norris K, Lucas-Wright A, Maidenberg M, Dillon-Brown N, Broussard M. One hundred intentional acts of kindness toward a pregnant woman: Building reproductive social capital in Los Angeles. *Ethn Dis*. In press.
111. Minkler M. Using Participatory Action Research to Build Healthy Communities. *Public Health Rep*. 2000;115:191-7.
112. Lu MC, Kotelchuck M, Hogan V, Jones L, Jones C, Halfon N. Closing the Black-White gap in birth outcomes: A life-course approach. Accepted for publication in *Ethnicity and Disease*, 2005.
113. Smedley BD, Stith AY, Nelson AR, eds. *Unequal treatment: Confronting racial and ethnic disparities in health care*. Washington DC: The National Academies Press, 2003.

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