

ABSTRACT

Title of Dissertation: EXAMINING MATERNAL EXPERIENCES OF FOOD INSECURITY ON BIRTH AND EARLY CHILDHOOD OUTCOMES

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Food insecurity, or the inadequate access to nutritionally-acceptable food, has been increasingly recognized as a distal determinant of poor health outcomes. While 11.1% of U.S. households suffer from food insecurity, specific populations are disproportionately affected, including pregnant women and single mothers. From a life course perspective, growth trajectories may be influenced by both prenatal exposures and postpartum practices, such as breastfeeding. Since food insecurity is associated with stress and poor nutrition, when occurring during critical periods of development, the fetus may be programmed to have poor future health. Other research on developmental origins have identified patterns that exhibit sex differences, particularly around growth. Therefore, this investigation sought to examine maternal food insecurity on: (i) low birthweight and sex-specific differences; (ii) breastfeeding initiation and exclusive breastfeeding duration; and (iii) obesity at age two and sex-specific differences. In Aim 1, mothers experiencing prenatal food insecurity had a higher odds of delivering a low birthweight infant, though much of the association was explained by sociodemographic factors after adjustment (OR

1.10 [95%CI 0.98, 1.25]), with female infants showing OR 1.21 (95%CI 1.02, 1.43). For Aim 2, food insecurity did not appear to deter women from initiating breastfeeding (OR 1.41 [95%CI 0.58, 3.47]), and minimal differences in exclusive breastfeeding duration between food insecure and food secure women were found (TR 0.89 [95%CI 0.57, 1.39]). Finally, Aim 3 revealed that sociodemographic factors explained much of the association between maternal food insecurity and childhood obesity at 2 years (OR 1.15 [95%CI 0.75, 1.75]), though male offspring showed a stronger magnitude (OR 1.54 [95%CI 0.86, 2.74]). Maternal food insecurity is a multi-faceted exposure that is related to other covariates, which may interact in multiple ways to influence health outcomes through both biologic and behavioral pathways. This preliminary investigation highlights the importance of exploring exposures occurring during critical periods, including food insecurity, on birth and childhood outcomes, and how it may affect the later health of offspring differently by sex. Additional longitudinal data with accurate measures of food insecurity are needed to fully ascertain how it affects the health of mothers and children, and to identify possible mediating pathways.

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AND EARLY CHILDHOOD OUTCOMES

by

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Dedication

This dissertation is dedicated to my grandma, Anna. She has always been my biggest support and believed I would one day become a doctor. Although her advanced Alzheimer's makes it difficult for me to share this achievement, I know that she would be beaming with pride.

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List of Abbreviations

AFT – accelerated failure time

aOR – adjusted odds ratio

BMI – body mass index

CDC – Centers for Disease Control and Prevention

CI – confidence interval

CPS – current population survey

DAG – directed acyclic graph

DHHS – Department of Health and Human Services

DOHaD – Developmental Origins of Health and Disease

EBT – electronic benefit transfer

FAO – Food and Agriculture Organization

FPL – federal poverty level

HFSSM – Household food security survey module

IUGR – intrauterine growth restriction

LBW – low birthweight

MCH – maternal and child health

NH – non-Hispanic

NHANES – National Health and Nutrition Examination Survey

OR – odds ratio

PRAMS – Pregnancy Risk Assessment Monitoring System

SGA – small for gestational age

SNAP – Supplemental Nutrition Assistance Program

TANF – Temporary Assistance for Needy Families

TR – time ratio

USDA – U.S. Department of Agriculture

WIC – Special Supplemental Nutrition Program for Women, Infants, and Children

Chapter 1: Introduction

1.1. Background and Significance

Food insecurity refers to inadequate access to nutritionally-acceptable food, with or without hunger.¹ An important phenomenon in the last few decades, food insecurity is most commonly thought to affect nutrition of an individual; however, it is increasingly being recognized as a distal determinant of other poor health and behavioral outcomes.^{1,2} Food insecurity may be temporary or long term, and it may be influenced by various factors, including income, unemployment, race/ethnicity, or even structural factors and neighborhood conditions such as food deserts and lack of available food.³ As such, food insecurity may be an issue at the individual or household level, as well as the community-level.

Recent data from the U.S. Department of Agriculture (USDA) suggest that as of 2018, 11.1% of U.S. households are experiencing food insecurity, which is equivalent to the entire population of Canada, and further, 4.3% or 5.6 million of U.S. households were suffering from very low food security.⁴ Food insecurity primarily affects all low-income individuals, even if they are marginally food insecure, which refers to when the household has had problems or anxiety with accessing adequate food. However, more than a quarter of food insecure people also live above the poverty line.^{5,6} One of the objectives of Healthy People 2020 for social determinants of health (and in particular, the domain of economic stability) is to reduce U.S. household food insecurity to 6%.³ To reach this goal, the national rate must be reduced by nearly half, necessitating more work in order to be able to this lower rate.

Specific people in various developmental stages of life are more vulnerable to food insecurity, including growing children, the elderly, and women. In fact, food insecurity disproportionately affects women, particularly single mothers.⁴ For example, despite the national household average, for women living alone, 14.2% were food insecure; this figure is 27.8% among single women with children.⁴ Given the valuable role that women play in both the economy and the family, it is important to consider the consequences that food insecurity has on women, and to find novel ways to intervene and support them through relevant policies.

Of particular note, food insecurity may be even more vital for pregnant women, who are experiencing physiological changes in order to support new life.⁷ As such, pregnancy requires a greater nutritional demand for energy to sustain both the mom and the baby. Unfortunately, food insecure mothers may be unable to meet those needs, and thus engage in strategies to cope such as consuming a poorer quality of food with excess macronutrients (i.e., carbohydrates, fats) but minimal micronutrients.^{1,8-10} Studies have shown that food insecurity has been associated with poor pregnancy outcomes, including gestational weight gain and gestational diabetes, as well as postpartum outcomes such as maternal postpartum weight gain and psychosocial outcomes such as depression, and anxiety.^{7,11,12}

Although food insecurity during pregnancy may appear to only be affecting the mother, there is also an effect on the developing fetus, which in turn can alter the future health of the fetus in childhood and adulthood.^{11,12,13} At birth, food insecurity may increase the risk of the infant having low birthweight or macrosomia.¹¹ From a life course perspective, food insecurity is important as poor nutrition and stress can affect fetal

programming that is mediated by the placenta, and it can alter the metabolism of the fetus, leading to obesity later in life.^{14,15} As a result of deprived micronutrients in utero, if the infant is born into a mismatched environment whereby there is an abundance of poor nutrition and maladaptive feeding practices, then it may increase the risk of developing insulin resistance in the child.^{14,16,17} Because food insecurity may result in stress and poor nutrition, as well as inadequate access to healthy food options (and possible greater intake of unhealthier options), then it is plausible that the fetus may be programmed to have increased risk of disease in adulthood due to the mother's food insecure environment.¹⁸ As a result, chronic conditions such as obesity and diabetes that develop later in life can increase health care expenditures, which in turn can result in an inability to work, lower household income, and maintain the continuing food insecurity cycle.^{19,20}

Postpartum, or after the infant is born, food insecurity may also affect behaviors, such as infant feeding practices and other nutritional food sources.²¹⁻²³ Breastfeeding offers an affordable and natural way to feed infants and would intuitively be the option to choose in a food insecure household; as such, breastfeeding serves as a source of food *security* for infants.^{22,24} However, from a biologic standpoint, if a mother is nutritionally depleted or does not have an appropriate intake of micronutrients as a result of food insecurity, it may make it harder to initiate and sustain breastfeeding.²⁵⁻²⁷ Moreover, food insecurity is also a chronic stressor and appears to have a dose-response relationship with depressive and anxiety symptoms,²⁸ which may contribute to decreased breastfeeding initiation and affect the decision-making for infant feeding.²² Food insecurity may also increase obesogenic feeding practices including decreased exclusive breastfeeding or not

appropriately responding to infant feeding cues.^{22,29,30} Beyond this first year, food insecurity may also affect availability of food sources for the child.²

Social assistance programs such as the Special Supplemental Nutrition Program for Women, Infant, and Children (WIC) or the Supplemental Nutrition Assistance Program (SNAP) may help to alleviate food insecurity. SNAP is available to all individuals based on income and does not have a standardized food list, which may further enable purchasing foods of low nutritional value.^{31–33} In contrast, WIC focuses on pregnant women, infant, and children, and a major component of the WIC program is nutrition in the form of education and food packages designed to meet nutritional standards for pregnant women, infants, and children. This intervention helps mothers to supplement their food budgets to purchase healthier foods as well as breastfeeding promotion. For example, recent changes have improved the food packages for mothers who choose to breastfeed as an incentive to encourage more breastfeeding.^{34,35} Such a program aids in providing proper nutrition to the developing fetus during pregnancy and the infant after birth.¹⁷

As mentioned, using a life course perspective, the future growth trajectories of the child may be influenced by both prenatal exposures and postpartum behaviors, such as breastfeeding.³⁶ Given the biological plausibility for food insecurity associated with stress and poor nutrition, the interplay between maternal environment and biology may produce future poor health.³⁶ Additionally, other research on exposures during critical periods have identified sex-specific patterns, particularly around growth, as males and females appear to have different growth adaptations, with males being more vulnerable (“male disadvantage”).^{37–41} While the exact mechanisms are still unclear, it appears to be

related to how placental genes are regulated.⁴² Therefore, this investigation seeks to explore how individual maternal experiences of food insecurity may affect immediate birth outcomes, early infant feeding practices, and childhood outcomes in the context of the life course theory. Given that food insecurity is an important public health issue, examining the effects of food insecurity during pregnancy is also relevant to explore, especially if it influences later health. Additionally, assessing whether effects of growth differ by sex of the infant is of interest. As there is a paucity of studies that have explored whether maternal experiences of food insecurity during pregnancy are associated with birth and early pediatric outcomes, this investigation seeks to fill this research gap by establishing quantitative associations that can be used for further investigation, improve existing data sources, and inform program and advocacy efforts to support food insecure mothers to prevent such outcomes. Additionally, few data sources currently exist for examining longitudinal infant and child health outcomes.⁴³ This investigation is novel in that it uses data collected longitudinally to examine the association between maternal food insecurity during pregnancy and breastfeeding patterns and early pediatric obesity.

1.2. Specific Aims & Hypotheses

Recognizing the prevalence and extent of women impacted by food insecurity at the individual level, it is important to better delineate the implications of in utero exposure to food insecurity on the health trajectory of the infant and child, and whether this differs by biological sex of the child. Therefore, this dissertation will examine the association between prenatal food insecurity and key birth, infant, and child health outcomes using the following three specific aims. A conceptual model of the aims of this investigation is presented in Figure 1.1.

Aim 1: Assess the association between prenatal food insecurity and infant birthweight in a sample of pregnant women in eleven States, and whether these associations differ by infant sex.

- Examine the association of prenatal food insecurity and infant low birthweight overall
- Determine whether this association differs by infant sex.

The hypotheses for this aim are as follows:

1. Mothers experiencing prenatal food insecurity will be more likely to have a higher prevalence of low birthweight at delivery compared to women with no food insecurity;
2. Associations between prenatal food insecurity and low birthweight will differ for male and female infants, such that males will have an increased risk of low birthweight.

Aim 2: Examine the associations between prenatal food insecurity and subsequent initiation and continuation of exclusive breastfeeding in a representative sample of pregnant mothers in Oregon.

- Determine the effects of exposure to prenatal food insecurity on breastfeeding initiation and duration of exclusive breastfeeding.

The hypotheses for this aim are as follows:

1. Mothers experiencing prenatal food insecurity will be less likely to initiate breastfeeding than mothers who were food-secure during pregnancy;

2. Among mothers that breastfeed their infants, food-insecure mothers will be more likely to have a shorter duration of exclusive breastfeeding than food-secure mothers;

Aim 3: Investigate the association between maternal food insecurity and pediatric obesity (at age 2) in a representative sample of pregnant mothers in Oregon, and whether this association is modified by sex of the infant.

- Establish whether exposure to any maternal food insecurity in the prenatal and/or postpartum period is associated with pediatric obesity (at age 2), as measured via body mass index (BMI).
- Ascertain whether this relationship may have a sex-specific response.

The hypotheses for this aim are as follows:

1. Mothers experiencing any maternal food insecurity will be more likely to have an obese child at 2 years of age than mothers not experiencing food insecurity;
2. Associations between maternal food insecurity and early childhood obesity will differ between male and female offspring, such that males will have an increased risk of obesity.

1.3. Research Implications

This investigation aims to fill the gap that currently exists on food insecurity during critical periods, such as the pregnant and postpartum period, on child outcomes.⁴³ Given that this investigation is a preliminary exploration of associations, the results of this investigation may spur additional hypothesis-generating research. Building on the findings of this study and future research in this area may lead to possibly programmatic

and policy-relevant improvements, including maternity care and neighborhood planning, such as increasing the number of grocery stores and farmers' markets to increase access to healthier foods for food-insecure mothers. Furthermore, findings may inspire further research on family and maternal-child health (MCH) policies, such as if WIC is meeting the needs of food insecure mothers or if it should be enhanced. Findings may also have the potential to promote additional research on other assistance programs that address food insecurity, such as whether SNAP may be helpful to mothers, particularly given the finite eligibility period for WIC, so that nutrition prior to, during, and between pregnancies may be optimized to improve birth and infant health.

Chapter 2: Literature Review

2.1. *Food (in)security and its measurement*

Food insecurity can best be explained by understanding how food *security* surfaced as an important concept. Currently, food security is understood to be a multidimensional concept that involves dimensions of food availability, food accessibility, food utilization, and food stability.^{1,24} Before food security was a recognized concept, food availability was tracked and measured following the First World War and during the Second World War to ensure there was an appropriate food supply, particularly for people who were in regions afflicted by conflict.^{44,45} Therefore, when the concept of food security first arose in the 1970s at the World Food Summit, the term emphasized food supply.^{45,46} Around the same time, hunger was a focus in the United States, despite federal assistance programs that were established (see Section 2.3). Although the U.S. was considered a country of abundant food compared to other low- and middle-income countries with chronic food scarcity, it was increasingly recognized that hunger in the United States was a problem that was becoming more prevalent.⁴⁶ After a federal task force was charged with determining why hunger was still increasing, they concluded that hunger was a complex issue that also included elements of poverty and unemployment, and that those being served by the federal assistance programs were a heterogeneous population.⁴⁶ The task force recommended that researchers and government agencies develop measures to assess the severity and magnitude of hunger.

While the U.S. began to include new questions on access to food supplies and food sufficiency (i.e., adequacy of food supplies) in national surveys, the international world realized that food security included other dimensions, such as accessibility, which

refers to not only the physical access of food, but also economic means to access food.^{24,45,46} The conceptualization of food security continued to evolve because of concerns that food acquisition behaviors within households was important, rather than within nations, because the behaviors of household members to acquire food translated into food security.⁴⁵ In particular, women preferred to allocate money towards health, nutrition, and education of children in the household compared to men.⁴⁵ Additionally, the international research community began shifting focus from undernutrition to micronutrient undernutrition, due to concerns of poor diet quality. As such, in addition to food availability and accessibility, it was recognized that food utilization was important for ensuring food security.^{24,45} This domain referred to the differences in distribution of food in the household, the nutritional quality of the food, and how the body consumes nutrients in the food to make enough energy that determines an individual's nutritional status.⁴⁵ Stability, though less recognized, was the last and arguably most important dimension of food security, because food security of a household may vary over time; therefore, there may be periods of food insecurity that are temporary or it may be chronic. This dimension explains how the other three dimensions (food distribution, nutritional food quality, and consumption of nutrients) must be stable over time and this can determine a household or individual's food security status.^{24,45} Figure 2.1 presents a heuristic of the multidimensional concept of food insecurity. This current definition of food security was established by the Food and Agriculture Organization (FAO) of the United Nations in the 1996 World Food Summit.

In tandem, the last decade of the 20th century saw a convergence of national and international findings to conceptualize the current understanding of food security, food

insecurity, and hunger as it pertains to the U.S. setting.⁴⁶ While food insecurity initially became a hunger-related issue in the U.S., it expanded to include some of the other domains of food security, with hunger becoming a possible consequence, but not a necessity, of food insecurity. The U.S. Department of Agriculture (USDA) and Department of Health and Human Services (DHHS) held a national conference on food security measurement and research in order to review, clarify, and consider reliable national measures of household and individual-level hunger and food insecurity. The work of two seminal research studies understanding families' experiences of hunger and food insecurity^{47,48} helped in the development of the Household Food Security Survey Module (HFSSM) that was first included in the 1995 supplement of the Current Population Survey (CPS).⁴⁵ This module is an 18-item survey asking respondents about various indicators of food insecurity and hunger to get at the prevalence and severity. In particular, the questions ask about anxiety of household food supplies, perceptions of the adequacy of the quality and quantity of accessible food, reduced food intake by adults, and reduced food intake by children. This measure has resulted in a continuum of food security status, ranging from high food security to very low food security (see Table 1 for definitions).⁴⁹ It should be noted, however, that the measure of food insecurity is an 'ever' measure: that is, whether respondents experienced any of the conditions at any time during the previous 12 months.⁴ Nonetheless, this measure of food insecurity has been found to be valid, and has been adapted in other low- and middle-income countries.⁵⁰⁻⁵³ Nationally, the validated HFSSM was included in surveys such as National Health and Nutrition Examination Survey (NHANES), and the Early Childhood Longitudinal Survey (ECLS).^{46,54} An abbreviated 6-item version was also created,⁵⁴ and

more recently a 2-item measure, and have been shown to have high sensitivity and specificity compared to the 18-item gold standard.^{55,56} From the 6-item measure, one question was selected for inclusion in select states' Pregnancy Risk Assessment Monitoring System (PRAMS) questionnaire.⁵⁷

Since the introduction of the HFSSM, it has provided data to monitor trends in food insecurity, develop research priorities, and evaluate the performance of food assistance programs in the U.S over time. While these data in the CPS are analyzed at the aggregate-level (i.e., national or state) by the USDA with the household as the unit of analysis, this information is still limited because it does not capture an individual's experience within the household.^{4,58} An advantage of the PRAMS' single-item question is that it surveys mothers to ascertain an indicator of food insecurity at the individual level rather than the household level. Additionally, this measure can be linked to prenatal and postpartum experiences in order to distinguish when food insecurity affected the mother, and to birth and infant health outcomes that are also collected in PRAMS. However, it should be noted that this PRAMS question does not capture all indicators of food insecurity as measured by the six-item measure or specific nutritional content consumed. Although the six-item measure was not intended to be used individually, even responding affirmatively to this question renders one to be considered marginally food secure, and research has indicated that even marginally food secure persons have similar health outcomes to food insecure persons.⁵⁹ Nonetheless, it serves as a useful tool to ascertain maternal experiences of food insecurity during the pregnancy and postpartum period, and prior studies have used this same question to determine maternal food insecurity status.^{57,60–62}

2.2. Epidemiology of food insecurity in the U.S.

As of 2018, the prevalence of household food insecurity in the U.S. was 11.1% according to the USDA, which is similar to the level reflected before the recession in 2007.⁴ Despite this decrease, there are still nearly 40 million people living in households suffering from food insecurity;⁴ this figure is equivalent to the population of Canada. Additionally, 4.3% of U.S. households (5.6 million households) were suffering from very low food security (higher than pre-recession rates), including running out of food and meal cutting.⁴ The prevalence of food insecurity in 2018 significantly declined from the prevalence in 2017 (11.8%), and represents a continuous decline since its peak in 2011, when the prevalence of food insecurity was 14.9%.⁴ The prevalence is higher, however, among households with children under 18, where 13.9% were food insecure in 2018. Of current relevance, because the COVID-19 pandemic has resulted in high rates of unemployment, it is likely that there will be increases in food insecurity seen in 2020.

The prevalence of food insecurity differs widely for certain geographic regions and sociodemographic characteristics. For example, large urban cities have higher rates of food insecurity than suburban areas, but overall, rural nonmetropolitan counties are disproportionately most affected, particularly in the South.^{4,58} Additionally, states vary in their rates of food insecurity; New Mexico had high rates of food insecurity, while Louisiana and Alabama had high rates of very low food security.⁵⁸

In terms of demographic characteristics, food insecurity primarily affects low-income individuals; 35.3% of households with a household income less than the poverty line were food insecure, but more than a quarter of households reporting income at or above 185% of the poverty line also suffer from food insecurity.^{4,6} Additionally, rates of

food insecurity were below the national average for non-Hispanic White households, as well as married couples with children.⁴ However, food insecurity disproportionately affects minority racial groups and women. Non-Hispanic Black and Hispanic households had higher rates of food insecurity than the national average (21.2% and 16.2%, respectively), as well as single women (14.2%) and single women with children (27.8%).⁴ Both single women and single women with children also had higher rates of very low food security than the national average (6.5% and 9.4%, respectively).⁴

Although the 2018 prevalence of food insecurity declined from 2017 among specific subgroups, including households with children headed by single women, the rate increased, though not significantly, among single women.⁴ Unfortunately, households headed by single women make up nearly a fifth of the overall household food insecurity rate.⁴ This latter group may also include single women who are pregnant. Therefore, policymakers and practitioners should keep in mind populations that are vulnerable and harder to reach, so that communities that are more ill-equipped are better supported to meet the needs of these populations.

2.3. Food assistance programs in the U.S.

In the U.S., there are two main food assistance programs to help individuals with limited resources cope with food insecurity: The Special Nutrition Assistance Program (SNAP) and the Special Supplemental Nutrition Program for Women, Infants, and Children (WIC). Both programs are managed by the Food and Nutrition Service (FNS) of the USDA.⁴ SNAP, which was formerly the Food Stamp Program, serves nearly 46 million low-income participants to purchase food using monthly benefits at authorized stores.³¹ Eligibility for SNAP requires that participants either meet an income criteria or

they are automatically eligible based on receipt of other safety net benefits.⁶³

Nonfinancial criteria also must be met, such as residency and seeking for employment if unemployed.⁶³ SNAP benefits are based on net monthly income, and benefits are given in the form of electronic benefits transfer (EBT) cards that can be used like debit cards in stores to purchase a variety of food items, often unrestricted in price or size. This means that items such as red meat, seafood, soft drinks, junk foods, and desserts are eligible.⁶⁴ Such freedom in unhealthy food purchases have led to studies on the effects of consuming such foods on health outcomes, resulting in a push toward trying to limit the types of foods eligible for purchasing under SNAP.³¹ In contrast, WIC, which is only available to pregnant women and children, is much more stringent and foods must meet certain nutritional standards. Although pregnant mothers may receive SNAP, WIC is specifically designed to improve the health of pregnant women and children. A larger discussion of the WIC program is needed in order to determine how it may be able to better support mothers and children.

WIC is a federal assistance program that was first implemented in 1972, as an amendment to the Child Nutrition Act of 1966.^{65,66} Administered by the USDA Food and Nutrition Service, WIC provides benefits to nearly nine million women and children per year, of which a million are pregnant women.⁶⁵⁻⁶⁷ In 2016, 39.6% of women who gave birth in the U.S. received prenatal benefits from WIC.⁶⁸ The need for this program arose because the U.S. recognized that many low-income Americans, and particularly mothers and children, were suffering from malnutrition.⁶⁵ Media attention gained traction because of the hunger that Americans were facing, which led to a conference on food, nutrition,

and health in 1970 and a report that recommended attention be given to the nutritional needs of low-income pregnant women and preschool children.⁶⁶

Although a proto-WIC program was established in 1969, it was recognized that this program in addition to SNAP was not meeting the needs of pregnant women and infants.⁶⁶ This ultimately led to the beginning of food banks, and women would be prescribed food that they could use at the food banks. These ‘prescriptions’ were the first vouchers, eventually establishing voucher programs that began appearing in several communities.⁶⁶ Ultimately, this led to the establishment of WIC as a permanent program in 1975.^{65,69}

In its current form, the program is eligible to women who are pregnant or postpartum within 6 months, or if breastfeeding, up to one year postpartum. In its initial years, non-breastfeeding postpartum women were not eligible. Children must be under the age of five. Participants must meet an income-based eligibility or be adjunctively eligible. The income requirement is for the family income to be at or below 185% of the federal poverty level, although this cut-off was not initially defined, and some states may choose to have variations in income eligibility.^{66,70} To streamline eligibility processes, adjunctive eligibility allows women and children to be automatically enrolled in WIC if they are enrolled in other safety-net programs such as the Supplemental Nutrition Assistance Program (SNAP), Medicaid, and the Temporary Aid for Needy Families (TANF).^{70,71} This eligibility is especially useful in states that have higher income eligibilities in these safety-net programs. Although this type of eligibility may put into question the equity of the WIC program if some WIC applicants above the 185% criteria

are eligible, data indicate that less than 2% of WIC participants have incomes above 185%.⁷²

Additionally, eligibility for WIC must include a determination of being nutritionally at risk. This determination may be made through biochemical (e.g., anemia levels) or anthropometric (e.g., body mass index) means, or other nutritionally-related conditions whether they are caused by deficiencies, dietary patterns, self-induced, or life changes.⁶⁶ Because WIC is a discretionary program and not an entitlement program, the number of individuals served by the program is limited by the funding that is established annually.⁷⁰ Therefore, a priority system is set up so that those individuals who are at the most nutritional risk receive the benefits of the program.⁶⁶ Thus, medically-based nutrition risks are prioritized over dietary-based nutrition risks, and pregnant women and breastfeeding mothers are prioritized over infants. Infants are prioritized over children, who are prioritized over non-breastfeeding postpartum women.⁶⁶ However, nationally representative data have indicated that this priority system enables at least 97% of income-eligible women, infants, and children to participate.⁷¹

The WIC program has three main components to the program. The first component is nutrition, and promotes healthy eating in order to improve fetal development and reduce low birthweight, which is one of the major causes of infant mortality. Participants are given a supplemental food package that contain nutrients that might be lacking in their diets.⁶⁶ Food packages include a wide variety of foods, and depending on the individual (i.e., pregnant mother, breastfeeding mother, or infant and child), they receive specific items such as milk, cheese, eggs, cereal, juice, beans, peanut butter, tuna, and infant formula. Greater flexibility in the foods chosen help States appeal

to the ethnic and cultural groups in their population.⁷⁰ Given that this food is supplemental, it is not intended to replace their complete diets or meet total nutritional needs. Infant formula is also provided as a benefit, particularly to mothers who cannot breastfeed. In addition, since 2009, women and children receive \$11 and \$8 vouchers, respectively, per month for fresh food and vegetables for use at authorized WIC vendors.⁷⁰

The second component is improved education about nutrition, including breastfeeding promotion and support, although the education component was not part of the program in its conception. This education component in WIC is unique compared to other federal assistance programs, and is an important benefit that sets WIC apart from other programs.⁶⁵ The goal of this education component is to stress the importance of proper nutrition and good health and to promote breastfeeding during pregnancy, to encourage initiation, and postpartum, to encourage continuation, in order to improve infant development, because the first year of life is very important for a child, and breastmilk serves as a source of food security for the infant.⁷³ Additionally, as an incentive to encourage breastfeeding over infant formula, fully breastfeeding mothers receive a larger food package with more variety if they choose not to receive infant formula from WIC.⁷⁴

Lastly, the WIC program also allows for referrals to other social and health services.^{65,70} These referrals are important in order to maintain good health during critical periods of growth and development. Local WIC agencies provide assistance to participants to obtain alternate services, such as SNAP, Medicaid, or immunizations.

WIC counsellors may also refer participants to other counselling services as needed to improve their mental health.

2.4. *Food insecurity and health outcomes*

Food insecurity is hypothesized to influence health throughout the life course through two kinds of pathways: nutrition pathways and non-nutrition pathways.⁷⁵ The nutrition pathway includes responses to the food intake as a result of food insecurity, whereas the non-nutrition pathway includes responses to actual or anticipated characteristics of the food supply.⁷⁵ The effects of food insecurity on child health has been studied extensively primarily using a nutrition pathway. For example, food insecurity is associated with anemia,^{76,77} a lower intake of nutrients,⁷⁸ cognitive, behavioral, and developmental issues,^{79–83} poor mental health,^{80,84,85} asthma⁸⁶, and weight status^{87–89} in children. For seniors, a few studies have explored the effects of food security status on health. Those studies that have been conducted found that food insecure seniors are more likely to have a lower intake of nutrients and overall poor general health,^{90–92} as well as poor mental health.^{90,93}

Similarly, there have not been many studies conducted on the adult population. In adult populations, food insecurity has been shown to be associated with an increase in chronic conditions such as hypertension and diabetes.^{94,95} Recent studies have also found food insecurity to be associated with health behaviors, such as alcohol use and smoking.⁹⁶ Using a family lens, food insecurity has implications in terms of family roles using the non-nutrition pathway, specifically the family stress theory. This theory indicates that a pile-up of stressors creates a strain, challenging resources and capabilities, which in turn compromises health.⁹⁷ However, families are usually able to develop coping mechanisms

to face these challenges.⁹⁷ The theory emphasizes the amount of stress that the family faces and how that stress may affect family functioning. Food insecurity may be one kind of stressor that can be introduced to the family and affect functioning. For example, one of the main functions of the family is to provide emotional support and nurturing, and therefore emphasizes the caregiving role that families serve.⁹⁸ Food insecurity is associated with poor mental health and depressive symptoms, especially among women.^{7,80,99,100} Because of these poor mental health outcomes, food insecurity may undermine a mother's role of being able to provide as caregivers. Some mothers feel shame and embarrassment that they are not able to care for their child, and worry that others may think that they are neglecting their child.¹⁰¹ These findings have reiterated the need for social and emotional support. Both cross-sectional and longitudinal data suggest that social and emotional support modifies the relationship between food insecurity and poor mental health among women.⁹⁹

Furthermore, food insecurity may be stressful because it threatens the economic stability of a family, which is another main function of family.⁹⁸ It is already recognized that food security includes the economic means to access food, and it falls under the domain of economic stability as a social determinant of health.³ Food insecurity could be brought on due to changes to the economy or individual financing and income. For families, including single mothers, there is often a feeling of helplessness and tension regarding family finances.¹⁰¹ Because of the financial burdens of other expenses, such as mortgages, car payments, health payments, and debts, families are forced to choose between which areas to prioritize in order to move forward.¹⁰¹ This often leads families

and single mothers to work two or more jobs in order to pay bills, and can even cause tension in the household.¹⁰¹

Specific focus on the effects of food insecurity in pregnant mothers warrant attention. Food insecurity coupled with pregnancy marks a particularly vulnerable time. Pregnancy is already a stressful period and can be taxing for the mother, because nutrient demands are higher to support the pregnancy.^{1,7} Additionally, food preparation may require more effort during pregnancy, and pregnant women may be required to take leave near the end of their pregnancy or if there are complications, which can lead to financial burden and further stress.^{1,7} These reasons may contribute to food insecurity during pregnancy. A population-based survey in California (Maternal and Infant Health Assessment) revealed that the majority of poor women reported food insecurity around the time of pregnancy compared to women of higher income groups.¹³ Food insecurity has also been found to increase poor health and pregnancy outcomes, including pre-pregnancy obesity, gestational diabetes, and increased gestational weight gain.¹¹

As already discussed, household food insecurity can have a profound impact on the health trajectory of children. However, food insecurity may also impact fetal and infant health if experienced prenatally or postnatally, and there is a paucity of information about these important periods of development. Maternal experiences during pregnancy, such as low or poor nutrition, may have consequences to the developing fetus in utero, which in turn, may impact the child's health at birth and into adulthood.¹⁰² For example, food insecurity during pregnancy may increase the likelihood of certain birth defects or result in low birthweight.^{11,103,104} Additionally, maternal food insecurity may also affect

infant feeding practices, including the decision to breastfeed.²² Mechanisms for how food insecurity may influence these outcomes are described below.

2.5. *Mechanism of associations between maternal food insecurity and birth and early childhood outcomes*

The importance of critical periods of development may explain the mechanism that links food insecurity and birth outcomes, such as low birthweight. Critical periods include the first 1,000 days of life from conception to the 2nd birthday, and these periods coincide with rapid cell differentiation and development of energy intake regulation.^{29,88,102} Therefore, when insults occur as a result of maternal environmental conditions, such as food insecurity and the poor nutrition and stress that may arise from it, the switching on or off of genes at the cellular level is likely to occur.¹⁰⁵ Several epidemiological studies reported an inverse relationship between birthweight and death risk from cardiovascular disease.¹⁰⁵ This unexpected discovery led to further studies confirming this association, which cannot be explained by confounding alone.¹⁰⁶ This was the foundation of the Developmental Origins of Health and Disease (DOHaD), which explains that later poor health originates from ‘programming’ as a result of developmental plasticity.¹⁰⁵ Developmental plasticity is the property of a genotype to produce several different phenotypes in response to environmental conditions. This phenomenon has been confirmed by biologists studying undernourished caterpillars, and this hypothesis has been extended for human development.¹⁰⁵ Therefore, variations in nutrient availability that occur in utero may influence gene expression, leading to fetal adaptations.¹⁰⁵

The recognition of this association led to the thrifty phenotype hypothesis, proposed by Hales and Barker. This hypothesis suggests that malnutrition during fetal and neonatal periods causes hypoplasia of the pancreas and hyposecretion of insulin as an adaptive response to deprived nutrients.¹⁰⁷ This reduction of insulin increases insulin resistance, directing more glucose to important organs, such as the brain and heart, and less to skeletal muscles.¹⁰⁸ As such, this response saves energy and is ‘thrifty’, resulting in a lowered fetal metabolism so that the growth of key organs are optimized in order to promote survival of the fetus at birth.¹⁰⁸ The trade-off in phenotypic changes ensures the short-term survival at the expense of a long-term advantage; in this case, intrauterine growth restriction (IUGR) is a consequence, bringing with it a high rate of later morbidity.^{109–111} Many mechanisms by which a fetus is programmed have been proposed; however, because a main determinant of intrauterine growth is placental function, evidence suggests that the placenta plays a central role in fetal programming of adaptation to maternal experiences, such as malnutrition or stress from food insecurity.^{106,112}

The placenta appears to act as a nutrient sensor, which co-ordinates transport functions with maternal availability of nutrients.¹¹² Therefore, this hypothesis is another way of suggesting that the maternal nutrient supply regulates key nutrient transporters, and the placenta makes changes in proportion to that supply in order to match the fetal growth rate.¹¹² In the setting of low or poor nutrition, this has implications for fetal growth and, therefore, programming. Thus, instead of upregulating placental nutrient transporters, when the placenta senses malnutrition, key transporters may be downregulated to decrease fetal growth while still protecting major organs.¹¹²

In addition, prenatal stress, which may include nutritional or psychosocial stress, is also implicated in fetal programming of growth.^{113,114} Increased levels of stress during pregnancy exposes the fetus to stress biomarkers, such as cortisol and pro-inflammatory cytokines, which can alter the placental physiology and function in providing nutrients to the developing fetus.^{39,115,116} Since food insecurity is associated with chronic stress,¹² it may also be possible that there may be an increase in exposure to stress biomarkers that ultimately result in placental dysfunction. As such, this may also increase the risk of oxidative stress, or the inability to remove reactive oxygen from aerobic metabolism.¹¹⁵ Because food insecurity may involve a deficiency of micronutrients, many of which may be antioxidants, this oxidative stress may lead to compromised fetal growth, programming outcomes such as low birthweight at birth, and morbidity in the future, including obesity.¹¹⁵

The pathway by which food insecurity may affect breastfeeding is likely from a behavioral standpoint, and has to do with how food insecurity affects parenting practices, threatens the caregiving role, and increased stress.^{73,117,118} Research has indicated that parents who suffer from food insecurity may be more likely to have less positive and more inconsistent parenting.^{119–121} Less positive parenting is associated with poorer infant feeding practices, such as adhering to breastfeeding recommendations and timing of when to introduce solid foods.¹²² Paradoxically, it may be expected that food insecure mothers would breastfeed because it is a free source of nourishment for the infant; however, many food insecure mothers also live in poverty, and they are the least likely to breastfeed.¹¹⁷ Qualitative studies have indicated that women who are food insecure perceive their breastmilk to be of poor quality due to their poor diets.^{117,123} Additionally,

because food insecurity is a stressor, it has been associated with increased depressive symptoms in women, which may contribute to decreased breastfeeding initiation and duration.^{121,124} Studies have also found that increased stress, including stress experienced during the prenatal period, can impair breastmilk production and lower oxytocin needed for the contraction of the breast for feeding, thereby decreasing initiation and continuation of breastfeeding.^{118,125–127}

Similarly, the mechanisms from maternal food insecurity and pediatric obesity are related to programming during critical periods and infant feeding practices. For example, the insulin resistance that may have been programmed in utero due to poor maternal nutrition (including total energy intake, and macronutrient and micronutrient composition) can result in epigenetic changes by the switching on and off of genes, eventually leading to glucose intolerance and other metabolic-related diseases later in life.^{14,128–131} In terms of infant feeding practices, breastfeeding has been consistently found to be protective against weight gain,^{128,132–134} and the introduction of solid foods earlier than recommended has been shown to be associated with increased adiposity and BMI.^{128,135,136} Food-insecure mothers have been shown to cease exclusive breastfeeding sooner,²² have non-responsive feeding styles in which mothers do not respond to infant hunger and satiety cues,^{23,29} or have increased controlling feeding styles out of fear that their infant would gain weight,^{21,121} all of which contribute to the child not being able to regulate their own hunger and satiety.¹²⁸ Additionally, breastfeeding has been shown to improve gut microbiota that is beneficial, as breastmilk contains *Bifidobacteria* species,^{128,137} and the composition of breastmilk contains nutrients that proliferate the species.¹³⁸ Studies have shown a causal link between appropriate development of gut

microbiota and protection against obesity.^{138,139} The recognition of both pathways are important to understand prenatal food insecurity and fetal susceptibility of obesity and diseases in later life.

2.6. *Potential sex-specific response to early life exposures*

In general, obesity is more prevalent among females than men globally.^{140–142} While the physiology of the sex differences continue to be investigated, most sex differences have been attributed to genetics. A recent genomic study has found that several genes associated with the weight-to-hip ratio in adulthood, which is an indicator of obesity, is differentially expressed in males than females.¹⁴³ Additionally, the genes on the X chromosomes have also been implicated in regulating body weight independently from sex hormones.^{38,144,145} Because females have two X chromosomes, manipulated animal models suggest that the second chromosome may contribute to a dosage response that influences body weight, though the Y chromosome in males is also not completely absolved.^{144,145}

Beyond genetics, and relevant to this investigation, studies and animal models have increasingly suggested that sexual dimorphism in growth trajectories begin at conception, and that there appears to be sex-specific responses to stress and changes in the prenatal and postnatal nutritional environment that may contribute to birthweight and obesity later in life.^{37,42,146} Additionally, there may be sex-specific sensitivities in response to metabolic hormones that maintain glucose homeostasis, such as leptin and insulin.³⁸ Postnatally, animal models also suggest that composition of maternal milk is sex-specific, and specific fatty acids and metabolic hormones that circulate in breastmilk as a result of

poor nutrition may elicit sex-specific programming of glucose homeostasis, appetite, and eating patterns in offspring.^{147–150}

It is suggested that the differing responses to stresses occurring during pregnancy may be due to how placental genes are regulated differently by sex.^{42,151} The placenta has long been assumed to have no sex attached to it; that is, it had been considered to be a sexless organ. However, given that the placenta originates from the fetus itself, the placenta does share the same genetic information as the fetus, and may likely be responsible for sex-specific differences and expressing of genes that influence growth.^{37,41,152} In terms of fetal growth, males and females adapt differently to nutritional or other stressors in order to promote survival, further contributing to sex differences in growth.^{39,42,151} Studies examining prenatal exposures and fetal programming have suggested that females appear to be more responsive to prenatal insults such as nutrient supply, environmental pollutants, and stress compared to males.^{39,153} As males invest in rapid fetal growth compared to females, when a nutritional insult occurs, the placental surface expands to maintain the rapid growth of male infants, while the placenta upregulates certain receptors and adapts the growth rate for females to reduce adverse outcomes.^{39,151,152} This difference in placental adaptation appears to confer a survival advantage in suboptimal conditions in utero for female fetuses, while males may be vulnerable to additional stresses, such as food supply, and adverse outcomes, such as preterm birth and neonatal mortality.^{39,42,151} The effects of programming may carry on postnatally, with animal models suggesting males to be vulnerable to programming of adiposity and body weight later in life from exposure to poor nutrition in pregnancy and during breastfeeding compared with females.^{154–156} Ultimately, these different

compensatory survival mechanisms may translate to adverse birth outcomes and poor health outcomes.⁴² At birth, this may mean that females may be born low birthweight due to the risky strategy that males employ,⁴² while males may be predisposed to develop early childhood obesity. Therefore, it is important to examine how exposures during critical periods may result in differences by biologic sex. The framework for how these differences are programmed is described below.

2.7. Conceptual framework: Life Course Health Development

The theme of the mechanisms between maternal food insecurity and infant and toddler outcomes and possible sex-specific responses described in Section 2.5 and 2.6 is best conceptualized using the life course framework, as depicted in Figure 1.1. Using this framework, the maternal environment (such as food insecurity) and stress may affect programming of the fetus through epigenetic changes and oxidative stress as carried out by the sex-specific placenta, which in turn influences how genes are expressed. The downstream effects of these changes may eventually lead to low birthweight (Aim 1), and obesity (Aim 3) in later life, exhibiting sex-specific differences. Additionally, food insecurity may lead to stress that affects infant feeding behaviors such as breastfeeding (Aim 2), which may also mediate postnatal growth. To further understand this conceptual model, a discussion of the life course framework follows.

In brief, the life course framework burgeoned out of work from the biopsychosocial model of health based on biology, sociology, and psychology, as well as health perspectives and theories emerging in epigenetics, developmental psychology, and the DOHaD hypothesis.³⁶ Shifting from the more mechanistic biomedical model, the life course theory hypothesizes that an individual's health trajectory is determined by

multiple interactions of biology, sociology, psychology, and environmental factors that occur at different life stages from gestation to adulthood.^{14,97}

As discussed, the main pathway for the associations in this investigation is through critical periods of development. Thus, explicit in this framework is that timing and sequence of events is a key component of health development, as adversity occurring during critical periods of development, can alter future life course, resulting in changes and adaptations to promote health, and any mismatches in the interaction between the biological and environmental contexts produce disease.³⁶ In the context of this investigation, this chain of risks as a result of maternal food insecurity may affect metabolic processes and poor eating habits in childhood. Coupled with environmental and social factors, such as poverty and living in food deserts or lack of green space, it can further facilitate obesity throughout adolescence and into adulthood.

That is not to mean that the future health trajectory is set in stone, but that the interactions between biological, social, and environmental exposures result in dynamic pathways of health development, another mechanism of the life course theory.^{36,157} For example, it is important to take into consideration how the social, economic, and policy environments interact with individual-level behaviors, physiology, and epigenetics across the life course.¹⁵⁸ Nutrition supports such as WIC have the potential to mitigate risks that may occur from food insecurity. Finally, exposures can be cumulative across an individual's life and pass down across generations, thereby affecting the future health of offspring. For example, if maternal food insecurity is associated with increased risks of developing metabolic disorders later in life, this can increase health care expenditures,

which in turn can result in possible unemployment, lower household income, and continuance of the food insecurity cycle.¹⁹

In sum, examining the effects of maternal food insecurity is important from a life course perspective. Current research is inadequate to determine how deleterious the effects of maternal food insecurity are and therefore warrants further attention. Using this information can inform additional research, improve existing policies, such as WIC, support advocacy initiatives, or implement other interventions to promote health among women and infants exposed to food insecurity.

Chapter 3: Methods

3.1. Aim 1: The association between food insecurity and low birthweight

3.1.1. Data Sources & Population

The Pregnancy Risk Assessment Monitoring System (hereafter referred to as CDC PRAMS) is an ongoing population-based surveillance system managed by the Centers for Disease Control and Prevention (CDC) since 1987.¹⁵⁹ The CDC PRAMS surveillance system was created in order to reduce infant mortality, low birthweight, and promote safe motherhood, and evaluates maternal attitudes and experiences before, during, and after the pregnancy across the U.S.¹⁶⁰ Currently, forty-seven states, the District of Columbia, New York City, Puerto Rico, and a tribe participating in CDC PRAMS, covering 83% of births.¹⁶⁰ CDC PRAMS surveys mothers within two to six months postpartum, with core questions asked of all states, such as prenatal care, obstetric history, and social support.¹⁶⁰ Because CDC PRAMS is managed at the state-level, there may be additional standard questions that meet the needs of specific states, such as food security.¹⁵⁹ Thus, it makes it unique in exploring and analyzing the mother-child dyad.

State birth certificate records are used as a sampling frame for CDC PRAMS, using a stratified, random sample to identify resident women who had a live birth 2-6 months prior.^{159–161} States may oversample based on specific characteristics, including maternal race/ethnicity, low birthweight infants (<2,500 grams), women in high-risk geographic areas, and sociodemographic factors, such as WIC participants.¹⁶⁰ Weighting accounts for the different sampling rates of the various subpopulations. Therefore, data

are representative of women in the states that participants reside. PRAMS collects data via mail, and uses telephone to follow-up with non-respondents.

3.1.2. Analytic Sample

For this investigation, CDC PRAMS data from 2009-2017 was utilized. Because only select states include a question on food insecurity, the following states were included: Colorado, Iowa, Kansas, Maine, New Mexico, Oregon, Pennsylvania, Tennessee, Wisconsin, and Wyoming. The analytic sample included all resident women of these states with a singleton birth who responded to CDC PRAMS during this time period.

3.1.3. Variables

Exposure: prenatal food insecurity. The exposure variable for this study was food insecurity during pregnancy. As previously mentioned, this was determined using the food security question in the above-mentioned states participating in CDC PRAMS. This question asked about the mother's food insecurity status with the following: "During the past 12 months before your new baby was born, did you ever eat less than you felt you should because there wasn't enough money to buy food?". Responses to this question on CDC PRAMS were dichotomized as 'yes' (food insecure) or 'no' (food secure). This question was adapted by a similar question from the USDA's six-item household measure.

Outcomes. The main outcome variables for this study was low birthweight. Low birthweight was defined as a delivery of an infant <2,500 grams. Birthweight is documented in CDC PRAMS using the birth certificate. This variable was dichotomized as either low birthweight (<2,500 grams) or not ($\geq$ 2,500 grams). Although dichotomizing

birthweight may have the potential to lose some information, examining changes in mean birthweight in a linear fashion may not be as meaningful than the categorical indicator from a clinical standpoint.^{162,163} This outcome was also explored restricting the sample to term births as a proxy for intrauterine growth restriction. Additionally, a sensitivity analysis examining birthweight as a categorical variable (low birthweight, normal birthweight [2,500-3,999 grams], macrosomia [≥ 4000 grams]) was also conducted. Because the public-use PRAMS data provides only categories of birthweight and gestational age, we were unable to measure birthweight that was small-for-gestational age.

Covariates. Individual-level maternal health behaviors and sociodemographic covariates known to be associated with low birthweight and food insecurity was considered (see Figure 3.1).^{164,165} Potential covariates such as maternal age, maternal education, maternal race/ethnicity, marital status, pre-pregnancy maternal BMI, number of previous livebirths, and income at or below the federal poverty level (FPL).^{164,166} Age was analyzed as a categorical variable due to the possible U-shaped association with low birthweight and preterm birth.^{167,168} The following categories were used: <20, 20-24, 35-39, 30-34, 35-39, 40+. Maternal educational attainment was analyzed categorically, with 'less than high school', 'high school', 'some college', and 'college degree'. Maternal race/ethnicity was examined categorically: NH-White, NH-Black, Hispanic, and Other. Marital status at birth was dichotomized as 'married' or 'not married'. Pre-pregnancy maternal BMI was derived from the birth certificate, and categorized as underweight (<18.5 kg/m²), normal (18.5-24.9 kg/m²), overweight (25-29.9 kg/m²), and obese (≥ 30 kg/m²). Number of children was determined by the number of previous

livebirths that the mother reports, and is also derived from the birth certificate. This variable was categorized as 0, 1, 2, and 3+. Income at or below the FPL was calculated using questions on household income in the previous year and the number of people in the household that relied on the household income for each survey year. FPL was examined categorically for descriptive purposes (0-99%, 100-185%, $\geq 186\%$), but continuously for model purposes. Survey year and state were also examined categorically to account for differences by year and contributions by state.

Prenatal WIC participation and smoking before and/or during pregnancy were also examined as possible covariates; however, because we did not have information on the timing of these covariates in relation to the experiences of food insecurity, these covariates could also act as potential mediators. Therefore, we examined them separately in analytic models described below. Prenatal WIC participation is derived from the birth certificate, and was categorized as ‘yes’ or ‘no’. Maternal smoking was also derived from the birth certificate, determined by the number of cigarettes smoked before and during pregnancy. Mothers who did not report smoking any cigarettes were classified as ‘non-smokers’. Mothers who responded to smoking any amount before pregnancy and not smoking during pregnancy were classified as ‘pre-pregnancy smoking only’. Mothers who smoked any amount before and during pregnancy were classified as ‘smoking before and during pregnancy’. Because gestational age may lie on the pathway between prenatal food insecurity and low birthweight, we also examined term births separately. For this reason, gestational age was dichotomized as ‘preterm (<37 weeks)’ or ‘term (37+ weeks)’.

Effect modifiers. Given the evidence of possible sex-specific patterns, infant's biological sex was assessed as a possible moderator according to the hypotheses of Aim 1. On PRAMS, infant sex was determined by the birth certificate. As such, it was dichotomously presented as 'male' or 'female'. For Aim 1, infant sex was tested as an interaction with prenatal food insecurity. This interaction would assess how food insecurity may differentially impact male offspring compared to female offspring. Given a priori recommendations to consider infant sex in prenatal exposure research,^{39,151} results were presented overall and stratified by sex.

3.1.4. Analytic Plan

Descriptive analysis were conducted for the exposure, outcome, and covariate variables overall and by infant sex. This included presenting means and standard deviations for continuous variables, and proportions for categorical variables. Additionally, the analyses included examining the distribution of maternal and infant characteristics, including the prevalence of low birthweight within the categories of prenatal food insecurity. Bivariate analyses compared the prevalence of low birthweight across maternal and infant characteristics using chi-square tests. All statistical analyses were be conducted using Stata 15 and 16 using appropriate survey commands for weighting.

Multivariable logistic regression was used to model the relationship between prenatal food insecurity and low birthweight. Covariates were included in the model using a tiered approach (i.e., examining sets of covariates based on their distal or proximal relationships or timing of occurrence with food insecurity and birth outcomes). Six models were examined: (1) unadjusted model of prenatal food insecurity and low

birthweight with no covariates; (2) adjusted model for sociodemographic covariates (maternal age, maternal race/ethnicity, maternal education, marital status, race/ethnicity, maternal pre-pregnancy BMI), survey year, and state; (3) Model 2 with previous livebirths; (4) Model 3 with income according to the FPL; (5) Model 4 with prenatal WIC participation; and (6) Model 5 with smoking before/during pregnancy. As previously mentioned, because gestational age may lie on the causal pathway between food insecurity and low birthweight, findings will also be restricted to term births (37+ weeks) to ensure that low birthweight is not due to the timing of delivery.

3.2. Aims 2 & 3: The association of food insecurity on breastfeeding and childhood obesity

3.2.1. Data Source & Population

For Aims 2 & 3 data specifically from Oregon PRAMS and PRAMS-2, an ongoing population-based surveillance system, were utilized. Oregon PRAMS and PRAMS-2 are managed by the Oregon Public Health Division, and supported by the CDC, as part of the national PRAMS surveillance system, which has been in place since 1987.¹⁵⁹ The PRAMS surveillance system was created in order to reduce infant mortality, low birthweight, and promote safe motherhood, and evaluate maternal attitudes and experiences before, during, and after the pregnancy across the U.S.¹⁶⁰ PRAMS surveys mothers within two to six months postpartum, with core questions asked of all states, such as prenatal care, obstetric history, and social support.¹⁶⁰ Because PRAMS is managed at the state-level, there are additional questions added that meet the needs of the Oregon Department of Human Services (DHS), including a question on food security.¹⁵⁹ Since 2006, PRAMS-2 is a follow-up survey of mothers who had previously responded to

PRAMS two years prior. Thus, using a longitudinal design, the Oregon Office of Family Health re-surveyed mothers when their child turned two. PRAMS-2 explores questions related to childcare, breastfeeding, nutrition, and child development.

Like CDC PRAMS, birth certificate records serve as a sampling frame for Oregon PRAMS, using a stratified, random sample to identify resident women who had a live birth.^{159–161} American Indian/Alaska Native, Asian/Pacific Islander, Hispanic, and Non-Hispanic (NH) Black women are oversampled, as well as white low birthweight infants (<2,500 grams).¹⁶¹ Low birthweight infants of other racial categories are unable to be oversampled due to the racial composition of the population in Oregon.¹⁵⁹ However, the weighting variables account for this oversampling, and therefore, data are representative of Oregon women. PRAMS collects data via mail, and uses telephone to follow-up with non-respondents. PRAMS-2 follows the same data collection procedure. All women who completed PRAMS are followed-up in PRAMS-2, unless they indicated to not be contacted again or if their infants died.¹⁶⁹ Because of loss to follow-up that may occur between PRAMS and PRAMS-2, PRAMS-2 is re-weighted to account for this so that data are still representative of the original PRAMS sampling frame. Given the longitudinal nature of PRAMS and PRAMS-2 when linked, this dataset makes it unique in establishing a temporal association, if one exists, between maternal food insecurity and BMI.

3.2.2. Analytic Sample

This investigation used Oregon PRAMS from 2008-2015, and PRAMS-2 from 2010- 2017, representing births from 2008-2015. It should be noted that data were not fully collected during 2012-13 due to changes in CDC collection protocol, and therefore

were excluded from this sample (including its follow-up in 2014-15). The eligible sample included all women with a singleton birth who responded to both PRAMS (2008-11 and 2014-15) and PRAMS-2 (2010-13 and 2016-17).

3.2.3. *Variables*

Exposure (Aim 2): prenatal food insecurity. In order to measure the exposure variable, prenatal food insecurity, the food security question in Oregon PRAMS was used for Aim 2. Similar to CDC PRAMS, this question asked about the mother's food insecurity status with the following: "During the past 12 months before your new baby was born, did you ever eat less than you felt you should because there wasn't enough money to buy food?", which was based on a question from the USDA's 6-item abbreviated food security module.^{54,60} Responses to this question on Oregon PRAMS were dichotomized as 'yes' (food insecure) or 'no' (food secure). This question is the same question used across other state-specific PRAMS surveys used in Aim 1. An additional analysis exploring food insecurity categorically (prenatal food insecurity only, persistent food insecurity) for the outcome of exclusive breastfeeding duration (described below) was also conducted. Women who responded 'yes' to only the question in PRAMS were considered to be prenatal food insecure, while women who reported having food insecurity in both PRAMS and PRAMS-2 follow-up were considered to have persistent food insecurity (food insecurity that carried into the postpartum period).

Exposure (Aim 3): any maternal food insecurity. In order to measure the exposure variable, any maternal food insecurity, the food security question in both Oregon PRAMS and PRAMS-2 was used for Aim 3. This question asked about the mother's food insecurity status with the following: "During the past 12 months before your new baby

was born, did you ever eat less than you felt you should because there wasn't enough money to buy food?" (as in Aim 1 and 2) and "In the past 12 months, did you ever eat less than you felt you should because there wasn't enough money to buy food?" (PRAMS-2), which would have been surveyed around the time the child was age 2 on average. Respondents who answered 'yes' to either of these two questions were dichotomized as 'any food insecurity' or 'food secure'. In additional sensitivity analyses, food insecurity was examined categorically by the phase of pregnancy experienced: prenatal food insecurity only, postpartum food insecurity only, and persistent food insecurity (experienced in both the prenatal and postpartum period). Women who responded 'yes' to only the PRAMS question and 'no' to the follow-up in PRAMS-2 were classified as having 'prenatal food insecurity'. Women who responded 'yes' to only the PRAMS-2 question were classified as having 'postpartum food insecurity'. Women who responded 'yes' to both questions were classified as having 'persistent food insecurity'. Finally, women who responded 'no' to both questions were considered to be food secure.

Outcomes (Aim 2). There were two main dependent variables for Aim 2: breastfeeding initiation and duration of exclusive breastfeeding. Initiation of breastfeeding was determined by the following question on Oregon PRAMS: "Did you ever breastfeed or pump breast milk to feed your new baby, even for a short period of time?" Responses to this question were dichotomized as 'yes' or 'no'. Because Oregon PRAMS only samples women two to six months postpartum, and to account for women who breastfed beyond this period, duration of exclusive breastfeeding required the linked PRAMS-2 dataset, which asked mothers to recall their total breastfeeding length and

introduction of other foods at the time that they were followed-up when their child was two years old. Therefore, exclusive breastfeeding duration was determined by taking the minimum age of the infant at which external liquids and/or solids were introduced, as reported by the mother. As such, this variable was recorded continuously.

Outcome (Aim 3). The dependent variable for Aim 3 was childhood obesity at 2 years of age, defined as having a BMI at or greater than the 95th percentile as per accepted pediatric charts by the CDC.¹⁷⁰ BMI was calculated using the height and weight variables as reported by the mother in PRAMS-2. This variable was dichotomized as ‘yes’ (obese) or ‘no’.

Covariates (Aim 2). For Aim 2, covariates that have been previously shown to be associated with food insecurity and infant feeding practices were considered, such as sociodemographic characteristics (maternal age, maternal education, marital status, race/ethnicity, number of children in the household, income at or below the federal poverty level, prenatal WIC participation), pregnancy and health behaviors (prenatal depression, maternal smoking), and survey year (Figure 3.2).^{22,171–174} Maternal age, maternal education, marital status, and race/ethnicity are derived from the birth certificate in PRAMS. Maternal age was categorized using the following: <20, 20-24, 25-29, 30-34, 35-39, 40+. Maternal educational attainment was analyzed categorically, with ‘less than high school’, ‘high school’, ‘some college’, and ‘college degree’. Maternal race/ethnicity was also examined categorically: NH-White, NH-Black, Hispanic, and Other. Marital status was dichotomized as ‘married’ or ‘not married’. Pre-pregnancy maternal BMI was derived from the birth certificate, and categorized as underweight (<18.5 kg/m²), normal (18.5-24.9 kg/m²), overweight (25-29.9 kg/m²), and obese (≥30 kg/m²). Number of

children was determined by the number of previous livebirths that the mother reports, and is also derived from the birth certificate. This variable was measured categorically as 0, 1, 2, and 3+. Like CDC PRAMS, the federal poverty level (FPL) at the time of pregnancy was calculated using questions on household income in the previous year and the number of people in the household that relied on the household income for each survey year. The FPL was also examined categorically for descriptive purposes as in Aim 1, but continuously for model purposes.

Prenatal WIC participation, prenatal depression, and smoking before and/or during pregnancy were also examined as possible covariates; however, because the timing of these covariates could not be differentiated from the timing of food insecurity, these covariates could also act as potential mediators. Additionally, longitudinal studies suggest that there may be a bidirectional relationship between depression and food insecurity.²⁸ Thus, we examined these covariates separately in analytic models described below. Prenatal WIC participation was examined using responses to the following question in PRAMS: “During your most recent pregnancy, were you on WIC (the Special Supplemental Nutrition Program for Women, Infants, and Children)?” Responses were dichotomized as ‘yes’ or ‘no’. Prenatal depression was derived based on responses to the following question in PRAMS: “During your most recent pregnancy, how often did you feel down, depressed, or hopeless?” Mothers who responded ‘almost always’ or ‘always’ were considered to have prenatal depression, while mothers who responded ‘never’, ‘rarely’, or ‘sometimes’ were considered to not have prenatal depression. Because postpartum depression would lie in the causal pathway between prenatal food insecurity and infant feeding practices, this was not adjusted for in models. Maternal smoking was

examined using responses to the following questions in PRAMS: “In the 3 months before you got pregnant, how many cigarettes did you smoke on an average day?” and “In the last 3 months of your pregnancy, how many cigarettes did you smoke on an average day?”. Mothers who responded as ‘I didn’t smoke then’ were classified as ‘non-smokers’. Mothers who responded to smoking any amount before pregnancy and not smoking during pregnancy were classified as ‘pre-pregnancy smoking only’. Mothers who smoked any amount before and during pregnancy were classified as ‘smoking before and during pregnancy’. Finally, the survey year was examined categorically.

Covariates (Aim 3). For Aim 3, potential covariates that were considered for the association between maternal food insecurity and child obesity included maternal age, maternal education, maternal race/ethnicity, pre-pregnancy maternal BMI, marital status, income at or below the federal poverty level, prenatal WIC participation, prenatal depression, maternal smoking before/during pregnancy, and survey year (Figure 3.3). Given the same data sources, these variables were analyzed similarly to what was described above for Aim 2. While the pathway from maternal food insecurity to child obesity status at 2 years of age may continue through breastfeeding and infant food insecurity status, these variables would act as potential mediators. While understanding the mechanism is important, this aim sought to establish the total effects as a first step. Therefore, breastfeeding and infant food insecurity status were not adjusted for.

Effect modifier (Aim 3). Infant sex was assessed as a possible moderator, according to the hypotheses of Aim 3. On PRAMS, infant sex is determined by the birth certificate. As such, it was dichotomously presented as ‘male’ or ‘female’. For Aim 3, infant sex was tested as an interaction with maternal food insecurity. This interaction

assesses how food insecurity may differentially impact male offspring compared to female offspring. Similar to Aim 1, the decision to present results overall and stratified by sex was made a priori given the potential for sex-specific differences.

3.2.4. Analytic Plan

Aim 2. First, descriptive statistics were conducted to examine the exposure, outcome, and covariate variables. The analyses included examining the distribution of maternal and infant characteristics within the categories of prenatal food insecurity. Bivariable associations with breastfeeding initiation were conducted, using chi-square tests. All analyses were conducted using Stata 15 or 16 survey data analysis command, using weights to account for the sampling design of PRAMS-2.

Multivariable logistic regression was used to examine the relationship between prenatal food insecurity and breastfeeding initiation in a tiered approach, that is, covariates were introduced as sets based on their distal or proximal relationships or timing of occurrence with food insecurity and birth outcomes. Six models were specified: (1) unadjusted model; (2); Model 1 adjusted for the demographic covariates (maternal age, maternal education, maternal race/ethnicity, maternal BMI, marital status), survey year; (3) Models 2 and 3 adjusted for socioeconomic covariates (number of live births, federal poverty level); (4) Models 3-5 adjusted for health behaviors that may be potential mediators (prenatal WIC participation, prenatal depression, and smoking status). Models were restricted to term births to remove the potential effect of gestational age and account for preterm births, which may affect these associations due to the differences in growth and management of preterm children.

For the second outcome, a Kaplan-Meier curve was constructed to visually differentiate exclusive breastfeeding rates among mothers who experienced prenatal food insecurity with mothers who did not. Accelerated failure time (AFT) models was used to examine the relationship between prenatal food insecurity and exclusive breastfeeding duration among the subset of women who reported breastfeeding their infant, using adjusted Wald tests and residuals to determine the appropriate distributional fit. Given the national and international recommendations to exclusively breastfeed for six months, women were right-censored at 6 months' duration. Similar to breastfeeding initiation, six AFT models were specified (1) unadjusted model; (2) Model 1 adjusted for demographic covariates and survey year; (3) Models 2 and 3 adjusted for socioeconomic covariates; (4) Model 3-5 adjusted for pregnancy and health behaviors. Similar to the outcome of breastfeeding initiation, models were restricted to term births. As described, food insecurity was also explored as a categorical variable to determine whether women who had persistent food insecurity (as a proxy measure for possibly severe or chronic food insecurity) influenced exclusive breastfeeding duration.

Aim 3. Similar to the previous two aims, descriptive statistics of the exposure, outcome, and covariate variables were explored. The distribution of maternal and infant characteristics within the categories of maternal food insecurity was assessed. Bivariable associations with obesity at 2 years was conducted overall and by infant sex using chi-square tests where appropriate. All analyses were conducted using Stata 15 or 16 using survey commands to account for the weighting in PRAMS-2.

Multivariable logistic regression was used to examine the relationship between maternal food insecurity and obesity at age 2. Similarly, six models were specified: (1)

unadjusted model; (2) Model 1 adjusted for the demographic covariates (maternal age, maternal education, maternal race/ethnicity, pre-pregnancy BMI, marital status) and survey year; (3) Models 2 and 3 adjusted for socioeconomic covariates (number of previous livebirths, federal poverty level); (4) Models 3 and 4 adjusted for pregnancy and health behaviors (prenatal depression, maternal smoking); and (5) Model 5 adjusted for prenatal WIC participation. As previously mentioned, models were additionally restricted to term births. because food insecurity may be a temporary or chronic phenomenon, a sensitivity analysis was also conducted classifying maternal food insecurity by phase of pregnancy in which it occurred: prenatal food insecurity only, postpartum food insecurity only, and persistent food insecurity, which included mothers who experienced food insecurity in both the prenatal and postpartum periods.

Chapter 4: Aim 1—Infant sex-specific associations between prenatal food insecurity and low birthweight: A multi-state analysis

4.1. Introduction

The birth size of a newborn is an important indicator of survival, health, and well-being. In particular, low birthweight (LBW), defined as <2500 g, accounts for 60-80% of neonatal mortality globally, and among survivors, is associated with increased risk of adverse outcomes, such as developmental delays and poor health later in life.^{1,2} Because maternal sociodemographic factors do not explain all of the risk in low birthweight, other risk factors occurring during pregnancy, such as stress and poor nutrition, should be explored. During pregnancy, poor nutrition has the potential to influence fetal programming of poor health.^{3,4} From a Life Course Health Development framework and the Developmental Origins of Health and Disease (DOHaD) hypothesis, any exposures or stressors occurring during critical periods of growth and development (the first 1,000 days since conception) may result in alterations to the growth trajectory of the child in response to the in utero environment.^{5,6} These changes are carried out at the cellular level by the placenta, diverting resources to protect the brain and heart at the expense of skeletal muscle to increase survival odds.⁶ As such, phenotypic changes, such as low birthweight, may be a consequence, with the expense of having a high rate of morbidity later in life.^{6,7}

Food insecurity, which is the inadequate access to nutritionally-acceptable food, is an example of how a mother's nutritional status may be compromised.⁸⁻¹⁰ Within the U.S. context, food insecurity affects 11.1% of households. However, the prevalence may vary by state; in New Mexico, 16.8% suffer from food insecurity, while 8.9% suffer from food

insecurity in Wisconsin.¹¹ Food insecurity disproportionately affects minority populations, as well as pregnant and single mothers.^{11,12} For the latter group, this distinction is important, as women require increased nutritional needs to support fetal development, and any changes that impact those needs may result in fetal programming of poor health.¹³ Additionally, from a non-nutrition pathway, food insecurity is an added stressor, and stress has been shown to influence birthweight due to altered placental function from exposure to stress biomarkers.¹⁴⁻¹⁶ Studies also suggest that there may be a sexually dimorphic response to changes in the prenatal environment, due to genetics or to sex-specific responses to stress signals such as cortisol and metabolic hormones responsible for glucose homeostasis.¹⁷⁻²² Specifically, sex differentials in fetal growth have been documented, with males investing in growth more than females; however, because males need to sustain growth, they may be at a disadvantage, while females can reduce their growth to prenatal stresses, such as poor nutrition, to reduce adverse outcomes and promote survival.^{18,22}

Previous studies have found an association between household food insecurity on birthweight, such as low birthweight and macrosomia (≥ 4000 g).^{16,23-25} However, few studies have explored the individual experience of food insecurity on birth outcomes beyond the household level and in a high-income country setting, such as the U.S. This limitation is due, in part, to the paucity of data sources available on maternal experiences of food insecurity and birth outcomes. Therefore, this investigation sought to explore the association between prenatal food insecurity and low birthweight, and whether or not there may be a sex-specific response using a multi-state survey.

4.2. Methods

Data source and analytic sample

This analysis was conducted using the Pregnancy Risk Assessment Monitoring System (PRAMS) data of livebirths from 2009-2017 from the following states that collected information on prenatal food insecurity: Colorado, Iowa, Kansas, Maine, Missouri, New Mexico, Oregon, Pennsylvania, Tennessee, Wisconsin, and Wyoming. PRAMS is managed by the Centers for Disease Control and Prevention (CDC) and state health departments, and consists of core and standard questions that states may opt to include, such as food insecurity. PRAMS uses state birth certificate records as a sampling frame, using a stratified, random sample to identify resident women who had a live birth within the previous 2-6 months. The analytic sample consisted of women with a singleton birth between 2009-2017 (N=50,915). Responses were weighted for oversampling of certain groups, non-response, and non-coverage to reflect the populations of the respective states. Additional details about PRAMS methodology have been described elsewhere.²⁶ This study received an exempt determination from the University of Maryland's Institutional Review Board, and was approved by the central CDC PRAMS and the respective states.

Measures

The exposure variable was prenatal food insecurity, which was measured using the single-item food insecurity question from the states that opted to ask women in PRAMS ["During the past 12 months before your new baby was born, did you ever eat less than you felt you should because there wasn't enough money to buy food?"]. This food insecurity question was based on certain aspects of the USDA's 6-item abbreviated

food security module, which include other domains of food insecurity, such as food availability, sufficiency, and resources.¹¹ Respondents who answered ‘yes’ to the question were dichotomized as ‘food insecure’ or ‘food secure’ if they responded ‘no’. The outcome variable was low birthweight, which was defined as having an infant weighing <2500 grams. This variable was derived directly from the birth certificate and categorized dichotomously.

For the association between prenatal food insecurity and low birthweight, we included maternal age, maternal education, maternal race/ethnicity, marital status, and pre-pregnancy BMI as potential covariates and were derived from the birth certificate. Age was analyzed as a categorical variable with the following categories: <20, 20-24, 35-39, 30-34, 35-39, 40+. Maternal educational attainment was categorized as: ‘less than high school’, ‘high school’, ‘some college’, and ‘college degree’. Maternal race/ethnicity was examined as: NH-White, NH-Black, Hispanic, and Other. Marital status at birth was dichotomized as ‘married’ or ‘not married’. Pre-pregnancy maternal BMI was categorized as underweight (<18.5 kg/m²), normal (18.5-24.9 kg/m²), overweight (25-29.9 kg/m²), and obese (≥30 kg/m²).

Furthermore, we included variables that were related to the socioeconomic status of the mother, such as previous livebirths, and income according to the federal poverty level (FPL). Previous livebirths was categorized as 0, 1, 2, and 3+, and the FPL was calculated using questions on household income in the previous year and the number of people in the household that relied on the household income for each survey year.

Prenatal WIC participation and maternal smoking before and/or during pregnancy were also considered as possible covariates that may influence the association. Prenatal

WIC participation is derived from the birth certificate, and was categorized as ‘yes’ or ‘no’. Maternal smoking was also derived from the birth certificate, determined by the number of cigarettes smoked before and during pregnancy. Mothers who did not report smoking any cigarettes were classified as ‘non-smokers’. Mothers who responded to smoking any amount before pregnancy and not smoking during pregnancy were classified as ‘pre-pregnancy smoking only’. Mothers who smoked any amount before and during pregnancy were classified as ‘smoking before and during pregnancy’. Finally, survey year and state were also considered to adjust for any differences by year and contribution by state. As we were interested in sex-specific differences, infant sex was also tested as a potential moderator.

Analysis plan

The distribution of maternal and infant characteristics within categories of prenatal food insecurity and sex of the child were examined. The prevalence of low birthweight by maternal characteristics was also examined in each respective group. Odds ratios (OR) and 95% confidence intervals (CI) for the association between prenatal food insecurity and low birthweight were estimated using multivariable logistic regression, and we used a tiered approach to covariates. First, Model 1 included demographic characteristics, such as maternal age, maternal education, marital status, race/ethnicity, maternal pre-pregnancy BMI, survey year, and state. Second, the number of previous livebirths was added to the model in addition to the covariates included in Model 1. Model 3 included income according to the FPL in addition to covariates included in Model 2, and was considered to be the main model. Because prenatal WIC participation and smoking may be behavioral factors that are influenced by the experience of food

insecurity, we added these separately in Models 4 and 5, respectively. Given the design of the survey, we are unable to determine the timing of these variables in relation to the mother's experience of food insecurity. Furthermore, we further restricted models to term births, given that preterm birth (<37 weeks) may affect infant growth and result in low birthweight. This restriction would help to identify low birthweight that may be more likely due to intrauterine growth restriction (IUGR), rather than short gestational age, that may be occurring in term births. Variance inflation factor (VIF) for multicollinearity was examined for models. Given the theoretical knowledge that there may be a sexually-dimorphic response to the prenatal nutritional environment,^{11,27} we stratified results by sex of child a priori.

Because of the potential for food insecurity to be associated with macrosomia,²⁸ a sensitivity analysis using multinomial regression was performed categorizing birthweight to include normal weight (2500-3999 grams) and macrosomia (≥ 4000 grams). All analyses were conducted using Stata 16 survey design command, with weights to account for the complex sampling design of PRAMS.

4.3. Results

The final unweighted analytic sample included N=50,915 women from the eleven states during 2009-2017. The weighted prevalence of prenatal food insecurity overall was 9.1% (unweighted N=5,138) and ranged from 7.9% to 11.8% across included states. Maternal and infant characteristics by prenatal food security status and sex of the child are presented in Table 4.1. Overall, the majority of women were between 24-34, non-Hispanic white, and delivered at term (37+ weeks). Food insecure women had a higher proportion of younger women (≤ 24 years) and minority women, were more likely to not

be married, have a high school education or less, were under 100% of the FPL, were underweight or obese pre-pregnancy, participated in prenatal WIC, and were smokers compared to food secure mothers. Food insecure mothers additionally appeared to have a higher proportion of babies born with low birthweight.

Among mothers who had a male offspring, food insecure women showed similar distributions to the overall food insecure population compared with their food secure counterparts. Similar distributions were also seen among food insecure mothers who had a female offspring. When comparing differences by sex of the child, food insecure women with a male child had a lower proportion of women who did not complete high school, and a lower proportion of babies who were born preterm and with low birthweight compared with food insecure women who had a female child. Food secure women had similar patterns in the distribution of characteristics by sex of child.

The prevalence of low birthweight by maternal characteristics and prenatal food insecurity are presented overall and by sex of the child in Table 4.2. Food insecure mothers generally had a higher prevalence of low birthweight, and this was more apparent among women who had a female offspring. Additionally, younger and older women, non-Hispanic black women, unmarried women, below 100% of the FPL, did not seek prenatal care, were underweight, were smokers, and had a history of depression had a higher prevalence of having a low birthweight infant, with the prevalence slightly more pronounced among food insecure women who had a female offspring.

Unadjusted and adjusted ORs for the association between prenatal food insecurity and low birthweight are presented in Table 4.3. Unadjusted results revealed that compared to mothers who were food secure, prenatal food insecurity was associated with

a statistically significant increased odds of delivering a low birthweight baby (OR 1.38 [95%CI 1.25, 1.53]). Increased odds of a low birthweight infant remained until the addition of federal poverty level. Addition of maternal smoking (Model 5) diluted results further, which appeared to explain most of the association.

Although the moderator for infant sex was not significant, stratified analysis suggested a stronger magnitude of effect among food insecure women that gave birth to females (unadjusted OR 1.48 [95%CI 1.28, 1.72] and remained significant after adjustment of covariates until the addition of maternal smoking (Model 5), which moved the estimate towards the null [aOR 1.13 [95%CI 0.94, 1.35]. Among food insecure women who gave birth to males, unadjusted results showed an increased odds of low birthweight (unadjusted OR 1.27 [95%CI 1.11, 1.47]); however, addition of sociodemographic variables appeared to explain the association. Similar patterns were seen when restricting the sample to term births. Figure 4.1 illustrates a visual representation of the marginal predicted mean low birthweight by sex of child and prenatal food security status for all births and term births, using Model 3. Girls born to food insecure mothers appeared to have a higher predicted mean of being born with low birthweight. VIF results did not reveal any multicollinearity between confounders. Sensitivity analyses found no difference in the adjusted odds of macrosomia overall or by sex of the child compared to normal birthweight infants, though it was suggestive of a decreased odds (aOR 0.81 [95%CI 0.65, 1.02]). Furthermore, patterns still held for the outcome of low birthweight compared with normal birthweight infants, and there were no substantive changes compared to our main analyses (Supplementary Table 4.1).

4.4. Discussion

In this study, the prevalence of prenatal food insecurity and low birthweight were 9.1% and 6.3%, respectively during this time period, and varied by state. Previous national estimates (1999-2008) using the USDA measure suggest that 14% of pregnant women are food insecure.¹² During this specific time period, national prevalence rates of food insecurity among single mothers or women were estimated to be between 20-30%.¹¹ Additionally, national estimates of low birthweight during this time period are around 8%.²⁹ These estimates may be lower due to the measure used for the exposure, and the states included. Our findings suggest that maternal prenatal food insecurity is associated with an increased odds of a low birthweight baby, particularly for female offspring, whereas this association was not associated after adjusting for sociodemographic differences. Findings were consistent when restricting to term births.

Our finding that food insecurity increases risk of low birthweight is consistent with previous studies exploring household food insecurity and birth outcomes, though primarily among low- and middle-income countries.^{16,23,25} However, unlike prior studies, we found that this association was most pronounced among food insecure women who gave birth to females.

In the context of early life programming models, nutrition pathways suggest that compromised nutrition during fetal development results in hyposecretion of insulin as an adaptive survival response to deprived nutrients.³⁰ As such, organs such as the brain and heart are protected, resulting in lowered fetal metabolism and decreased growth to promote infant survival.^{3,5} Additionally, from a non-nutrition pathway, food insecurity is a chronic stressor especially during pregnancy, and studies have shown that chronic stress

can influence birthweight.^{14,15,24} Since stress during pregnancy may increase the fetus' exposure to stress-related biomarkers, such as cortisol, these factors have also been theorized to affect the placenta's fetal programming of growth and development.²⁷ However, and consistent with our findings, previous research has indicated that there may be sex-specific programming in response to maternal under- or overnutrition and stress during critical periods of development.²⁰⁻²² In terms of fetal growth, males and females rely on different strategies to increase their chances of survival during times of nutritional or other stressors, resulting in differences in growth.^{18,21,22} Studies examining fetal programming have suggested that females appear to be more responsive to prenatal insults such as nutrient supply and stress compared to males.²¹ Additionally, females may be more vulnerable to a mother's lifetime nutrition and metabolism, while males may be vulnerable to a mother's current diet.¹⁸ Because of this vulnerability and the fact that males invest in rapid fetal growth compared to females, when a nutritional insult occurs, the placenta shifts to maintain the rapid growth of male infants, while the placenta adapts the growth rate for female that may be reduced with fewer adverse outcomes.^{21,22} This difference in placental adaptation appears to increase the ability for a female fetus to survive in suboptimal conditions in utero, while males may be vulnerable to additional stresses.^{21,22} Thus, future research should explore whether food insecurity is associated with decreased infant survival and whether it differs by sex.

It should be noted that there was a higher proportion of females born preterm. However, even when restricted to term births, our findings revealed the effect of prenatal food insecurity on low birthweight became stronger among females. Since preterm birth is associated with an increased risk of a low birthweight baby and affects infant growth

differently,³¹ restriction to term births enabled us to focus on low birthweight driven by intrauterine growth restriction and possibly the length of time that the mother may have been at risk for food insecurity.

It is interesting to note that the food insecure women in this sample had a higher proportion of women who were underweight and obese compared to food secure mothers. Food insecurity has been shown to affect maternal weight gain during pregnancy, which may in turn affect the growth of the infant.^{28,32} While the association was still significant independent of pre-pregnancy BMI, it is uncertain whether maternal weight gain, for which we were unable to ascertain in these data, may be a possible mediator in this association and may require further research. Additionally, food insecurity may also increase the likelihood of smoking due to the stress, and because smoking is a risk factor for low birthweight,³³ it appeared to explain much of the risk of low birthweight. However, because we could not distinguish the timing of when smoking occurred in relation to prenatal food insecurity in these data, and the fact that smoking may be exacerbated by food insecurity,³⁴ further study may be warranted on how health behaviors, such as smoking, may mediate the association.

Strengths and Limitations

This analysis has some strengths to note. First, this study is, to our knowledge, the first to have explored the individual experience of maternal prenatal food insecurity on low birthweight and assess this association by infant sex. PRAMS is also one of the few datasets that includes a question on food insecurity that can be linked to prenatal and postpartum experiences. Second, we included multiple states in the analysis, with a fairly large sample size. Given that PRAMS are state-level population-based data, these data

are generalizable to the women in the states included due to the sampling and weighting of the data.

This analysis also has some limitations to take into consideration when interpreting our data. The most important limitation was that only a single-item food insecurity question was used. The single-item question, though adapted from USDA's six-item measure, does not capture the entire construct of food insecurity that longer modules measure, including aspects of affordability, availability, and sufficiency of food. Although, this measure is unable to discern the true extent of prenatal food insecurity or the nutritional impact (i.e., whether a mother experienced under- or overnutrition), the fact remains that women may cope by eating a poorer quality of food at a lower cost and/or engaging in poor health behaviors such as smoking. Future surveys and/or studies should consider incorporating the full food insecurity module or even a 2-item measure that has been validated particularly in clinical settings.^{35,36} Compared to the PRAMS question, which asks about meal-cutting, the 2-item measure is derived from two questions from the full 18-item measure related to stress from sufficiency and affordability of food.

Second, low birthweight may be a poor proxy of fetal growth, and birthweight according to gestational age, such as small for gestational age (SGA), may be more informative of fetal growth because it takes into account gestational age.³⁷ Unfortunately, we were unable to determine whether the infant was born SGA within this dataset due to variables being provided at the aggregate level to prevent identification. Third, beyond the WIC program, there may be other safety-net benefits such as the Supplemental Nutrition Assistance Program (SNAP) that may also be beneficial in improving food

security status or diet quality and we were unable to ascertain the effects of SNAP using these data; however, SNAP has not been shown to be nutritionally advantageous in the same way as WIC.³⁸ We accounted for WIC, which may likely capture potentially improved diet quality. However, further research may be warranted on how these safety-net nutrition programs may be leveraged to increase healthy options and consider possible sex-specific effects from food insecurity during pregnancy.

Conclusion

In summary, prenatal food insecurity is associated with an increased risk of low birthweight, and findings suggest a potential sex-specific association, with the overall effect largely driven by the effect on female offspring, even after adjustment for sociodemographic characteristics and WIC status. Future studies should use improved measures of food insecurity and continue to consider possible sex-specific effects of food insecurity. These future investigations may help to further elucidate the association and determine whether interventions should be developed or improved to protect future health of infants exposed to prenatal food insecurity.

Table 4.1. Weighted percent distribution of selected maternal and infant characteristics by food insecurity status overall and by sex of the infant: PRAMS data, 2009-2017

	Total n (unweighted)	Food insecure ¹			Food secure ¹		
		Overall	Male	Female	Overall	Male	Female
Maternal characteristics at time of birth							
Maternal age at birth (years)	n=50,915	n=5,138	n=2,590	n=2,548	n=45,777	n=22,975	n=22,799
< 20	3479	10.2	10.8	9.7	5.8	6.1	5.5
20-24	10934	35.6	34.2	37.1	18.8	19.1	18.5
25-29	15057	28.5	30.5	26.5	30.2	30.0	30.4
30-34	13613	16.7	14.9	18.6	29.2	28.9	29.5
35-39	6386	7.1	7.9	6.2	13.2	13.2	13.2
≥40	1446	1.8	1.7	1.9	2.8	2.7	2.9
Maternal race/ethnicity	n=50,453	n=5,088	n=2,564	n=2,524	n=45,365	n=22,746	n=22,616
non-Hispanic white	28180	58.7	58.8	58.7	68.1	68.1	68.0
non-Hispanic black	4249	12.2	12.9	11.5	7.6	7.4	7.7
Hispanic	10891	20.8	19.7	22.1	16.1	16.1	16.0
Other ²	7133	8.2	8.7	7.7	8.3	8.4	8.2
Marital status	n=50,832	n=5,121	n=2,581	n=2,540	n=45,711	n=22,937	n=22,771
Not married	19149	62.6	61.7	63.6	33.7	33.9	33.4
Married	31683	37.4	38.3	36.4	66.3	66.1	66.6
Maternal educational attainment	n=50,521	n=5,076	n=2,555	n=2,521	n=45,445	n=22,806	n=22,636
No high school diploma	7349	19.7	17.8	21.8	12.8	12.9	12.6
High school diploma	12174	37.0	38.8	35.0	22.8	22.5	23.2
Some college	15144	35.6	35.7	35.4	27.3	27.3	27.2
Bachelor's degree or higher	15854	7.7	7.7	7.8	37.1	37.4	36.9
Median federal poverty level (%)	205 [87, 369]	75 [46, 135]	87 [48, 140]	68 [40, 132]	222 [100, 396]	218 [99, 395]	223 [102, 396]

	Total n (unweighted)	Food insecure ¹			Food secure ¹		
		Overall	Male	Female	Overall	Male	Female
Federal poverty level categories							
0-99%	n=47,194 15327	n=4,770 58.1	n=2,401 56.3	n=2,369 60.0	n=42,424 25.0	n=21,303 25.4	n=21,119 24.6
100-185%	9942	29.4	30.7	28.1	19.2	19.0	19.5
≥186%	21925	12.5	13.0	11.9	55.8	55.6	55.9
Payment source at delivery	n=45,227	n=4,519	n=2,262	n=2,267	n=40,708	n=20,346	n=20,359
Medicaid	20348	68.7	69.4	68.0	35.6	35.7	35.5
Private	21433	25.6	25.6	25.5	57.2	56.9	57.5
Self-pay	1529	2.9	2.8	3.1	3.7	3.9	3.5
Other	1917	2.8	2.2	3.4	3.5	3.5	3.5
Prenatal WIC participation	n=4,092	n=4,438	n=2,234	n=2,204	n=39,464	n=19,749	n=19,712
Not eligible (FPL>185)	17475	8.1	8.9	7.2	49.0	49.1	48.9
Eligible but no use (FPL≤185)	7303	23.5	23.3	23.8	14.5	14.5	14.4
Yes	19124	68.4	67.8	69.0	36.5	36.4	36.7
Prenatal care sought	n=49,776	n=4,997	n=2,514	n=2,483	n=44,779	n=22,456	n=22,320
No	375	1.3	1.4	1.2	0.7	0.7	0.7
Yes	49401	98.7	98.6	98.8	99.3	99.3	99.3
Pre-pregnancy body mass index	n=48,744	n=4,874	n=2,458	n=2,416	n=43,870	n=22,040	n=21,828
Underweight	2069	5.8	5.9	5.8	3.7	3.9	3.5
Normal	23783	41.5	41.1	41.9	50.4	50.5	50.2
Overweight	11760	24.0	23.7	24.2	24.2	24.4	24.1
Obese	11132	28.7	29.3	28.1	21.7	21.2	22.2
Smoking before/during pregnancy	n=46,116	n=4,596	n=2,303	n=2,293	n=41,520	n=20,775	n=20,742
Never	40197	69.2	69.4	68.9	87.9	87.6	88.1
Before only	1289	4.2	4.1	4.3	3.3	3.3	3.2
Before and/or during	4630	26.6	26.4	26.8	8.8	9	8.6

	Total n (unweighted)	Food insecure ¹			Food secure ¹		
		Overall	Male	Female	Overall	Male	Female
Pre-pregnancy depression	n=50,267	n=5,063	n=2,559	n=2,504	n=45,204	n=22,678	n=22,523
No	43224	71.6	73.0	70.0	88.2	88.3	88.2
Yes	7043	28.4	27.0	30.0	11.8	11.7	11.8
Route of Delivery	n=49,594	n=4,988	n=2,516	n=2,472	n=44,606	n=22,383	n=22,221
Spontaneous vaginal	33365	68.9	68.9	69.0	68.9	67.4	70.5
Assisted vaginal	1574	2.9	3.4	2.5	3.6	4.1	3.2
Cesarean section	14655	28.2	27.8	28.6	27.4	28.5	26.3
Infant characteristics at birth							
Previous livebirths	n=50,791	n=5,120	n=2,585	n=2,535	n=45,671	n=22,915	n=22,753
None	21473	39	38.6	39.4	40.9	40.8	41
1	15564	29.4	29.0	29.9	33.1	33.3	32.9
2	8060	17.6	18.3	16.9	15.7	15.7	15.8
≥3	5694	13.9	14.1	13.8	10.3	10.3	10.3
Gestational week at birth	n=50,832	n=5,131	n=2,589	n=2,542	n=45,701	n=22,929	n=22,769
<37 weeks (preterm)	8153	8.9	7.9	10	7	7.2	6.8
≥37 weeks	42679	91.1	92.1	90.0	93.0	92.8	92.2
Sex	n=50,912	n=5,138	n=2,590	n=2,548	n=45,774	n=22,975	n=22,799
Male	25565	51.2	-	-	50.8	-	-
Female	25347	48.8	-	-	49.2	-	-
Birthweight categories	n=50,915	n=5,138	n=2,590	n=2,548	n=45,777	n=22,975	n=22,799
Low birthweight (<2500g)	11268	8.2	7.2	9.3	6.1	5.7	6.5
Normal birthweight	36206	85.6	85.4	85.7	85.5	83.4	87.5
Macrosomia (≥4000g)	3441	6.2	7.4	5.0	8.5	10.8	6.1

1 – weighted proportion

2 – includes Asian/Pacific Islander, American Indian or Alaska Native, Other, and Hispanic origin unknown or not stated.

Table 4.2. Weighted prevalence of low birthweight by maternal characteristics and exposure overall and by sex of the infant: PRAMS data, 2009-2017

Total number of births Exposure	Low birthweight					
	Overall			Male		
	n	Percent ¹	p-value	n	Percent ¹	p-value
Maternal demographics						
Prenatal food insecurity						
No	9896	6.1	<0.001	4639	5.7	<0.001
Yes	1372	8.2		645	7.2	
					6.5	<0.001
					9.3	
Maternal age at birth (years)						
Under 20	874	8.5		451	8.1	
20-24	2509	6.9		1139	6.3	
25-29	3166	5.9	<0.001	1478	5.5	<0.001
30-34	2848	5.5		1338	5.2	
35-39	1456	6.1		684	5.6	
40+	415	9.0		194	8.4	
Maternal race/ethnicity						
non-Hispanic white	7323	5.3		3435	5.0	
non-Hispanic black	1037	11.7	<0.001	454	10.6	<0.001
Hispanic	1812	6.7		870	6.2	
Other ²	984	7.4		472	6.9	
Marital status						
Not married	4568	8.4	<0.001	2140	7.8	<0.001
Married	6669	5.1		3126	4.7	
Maternal educational attainment						
No high school diploma or GED	1595	7.8	<0.001	775	7.5	<0.001
					8.1	<0.001

Total number of births	Low birthweight							
	Overall				Male			
	n	Percent ¹	p-value	n	Percent ¹	p-value	n	Percent ¹ p-value
High school diploma or GED	3041	7.6		1414	7.0		1626	8.1
Some college	3363	6.4		1536	6.1		1827	6.9
Bachelor's degree or higher	3149	4.6		1504	4.2		1644	5.0
Federal poverty level								
0-99%	3565	8.2		1633	7.4		1902	9.0
100-185%	2101	6.4	<0.001	980	6.2	<0.001	1121	6.7
≥186%	4654	5.0		2197	4.7		2456	5.3
Prenatal WIC participation								
Not eligible (FPL>185)	1792	8.1		818	7.5		974	8.7
Eligible but no use (FPL≤185)	3565	4.8	<0.001	1671	4.5	<0.001	1893	5.1
Yes	3869	7.2		1811	6.7		2057	7.8
Prenatal care sought								
No	132	13.8		64	14.0		68	13.5
Yes	10885	6.2	<0.001	5098	5.7	<0.001	5785	6.7
Pre-pregnancy body mass index								
Underweight	691	10.6		314	9.4		377	12.0
Normal	5344	6.0		2468	5.4		2876	6.7
Overweight	2332	5.6	<0.001	1123	5.4	<0.001	1209	5.8
Obese	2404	6.8		1140	6.6		1263	6.9
Smoking before/during pregnancy								
Never	7786	5.7		3650	5.3		4134	6.0
Before only	260	6.3		116	5.1		144	7.6
Before and/or during	1654	11.2	<0.001	746	10.4	<0.01	908	12.2
Pre-pregnancy history of depression								
No	9125	5.9	<0.001	4292	5.5	<0.001	4831	6.3
Yes	2003	8.6		925	8.0		1078	9.3

1 – weighted proportion

2 – includes Asian/Pacific Islander, American Indian or Alaska Native, Other, and Hispanic origin unknown or not stated.

Table 4.3. Unadjusted and adjusted OR of prenatal food insecurity and low birthweight overall and by infant sex: PRAMS data, 2009-2017

	All births			Term births		
	Overall OR (95% CI)	Male OR (95% CI)	Female OR (95% CI)	Overall OR (95% CI)	Male OR (95% CI)	Female OR (95% CI)
Unadjusted	1.38 (1.24, 1.53)	1.27 (1.11, 1.47)	1.48 (1.28, 1.72)	1.47 (1.26, 1.72)	1.31 (1.05, 1.63)	1.62 (1.30, 2.01)
Model 1	1.13 (1.01, 1.27)	1.01 (0.85, 1.18)	1.26 (1.07, 1.49)	1.17 (0.99, 1.39)	0.98 (0.77, 1.25)	1.35 (1.07, 1.71)
Model 2	1.15 (1.03, 1.29)	1.03 (0.88, 1.22)	1.28 (1.08, 1.51)	1.20 (1.01, 1.43)	1.01 (0.79, 1.30)	1.38 (1.09, 1.75)
Model 3	1.10 (0.98, 1.25)	1.01 (0.85, 1.20)	1.21 (1.02, 1.43)	1.15 (0.96, 1.37)	0.98 (0.76, 1.28)	1.29 (1.02, 1.64)
Model 4	1.12 (0.99, 1.27)	1.02 (0.85, 1.22)	1.23 (1.03, 1.47)	1.15 (0.96, 1.38)	0.97 (0.74, 1.28)	1.31 (1.02, 1.68)
Model 5	1.04 (0.91, 1.18)	0.96 (0.80, 1.15)	1.13 (0.94, 1.35)	1.06 (0.88, 1.27)	0.90 (0.68, 1.19)	1.19 (0.92, 1.52)

Model 1 – adjusted for maternal age, maternal education, maternal race/ethnicity, maternal BMI, marital status, survey year, state

Model 2 – Model 1 + number of previous livebirths

Model 3 – Model 2 + federal poverty level

Model 4 – Model 3 + WIC use

Model 5 – Model 4 + maternal smoking

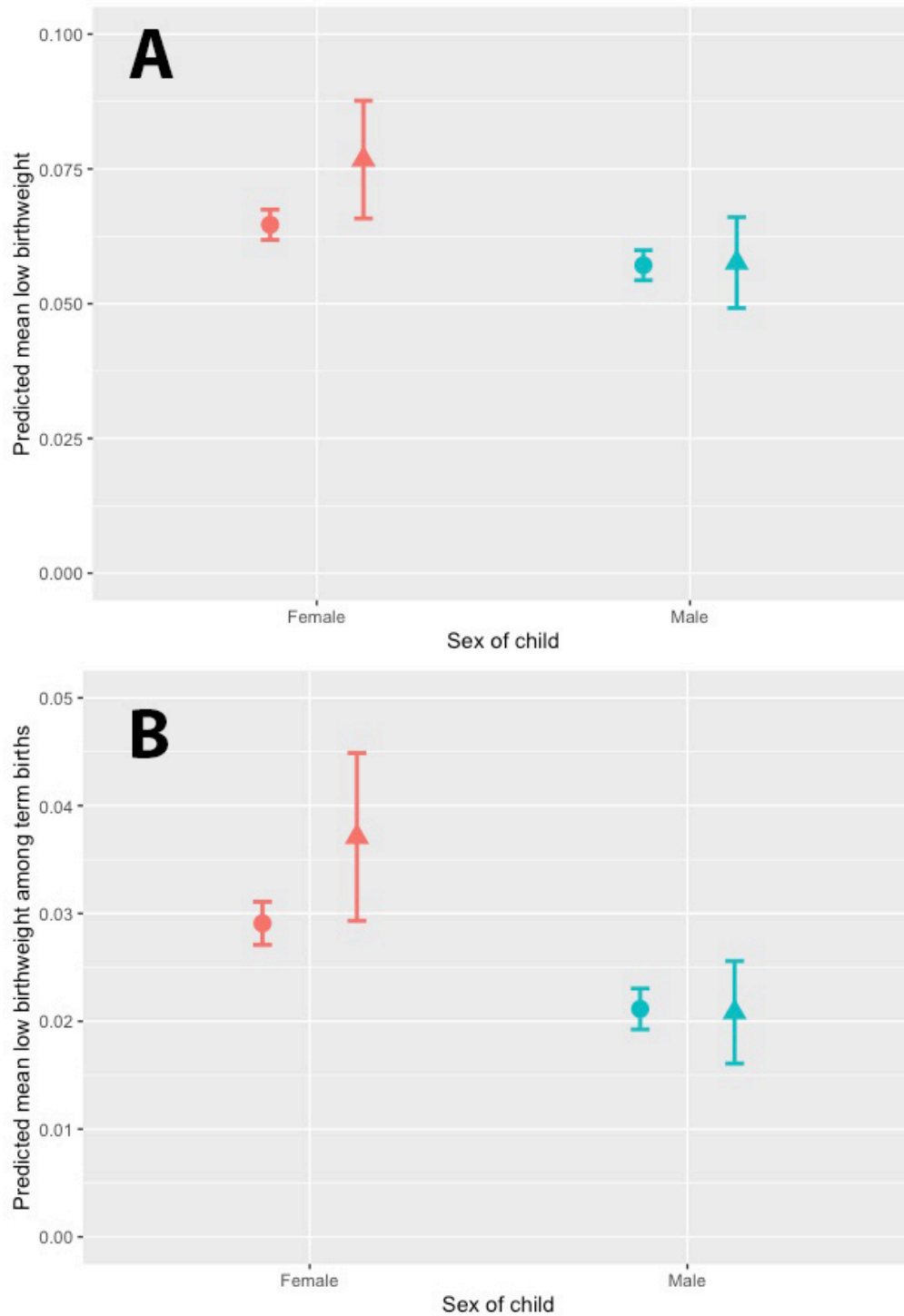


Figure 4.1. Predicted mean birthweight among A) all births and B) term births. Circles indicate food secure mothers, and triangles indicate food insecure mothers.

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SUPPLEMENTAL APPENDIX

Supplementary Table 4.1. Multinomial logistic regression of unadjusted and adjusted OR of prenatal food insecurity and low birthweight or macrosomia overall and by infant sex: PRAMS data, 2009-2017.

	All births			Term births		
	Overall OR (95% CI)	Male OR (95% CI)	Female OR (95% CI)	Overall OR (95% CI)	Male OR (95% CI)	Female OR (95% CI)
Unadjusted						
Low birthweight	1.35 (1.21, 1.49)	1.23 (1.06, 1.42)	1.47 (1.26, 1.70)	1.44 (1.23, 1.68)	1.26 (1.01, 1.56)	1.60 (1.29, 1.99)
Macrosomia	0.73 (0.60, 0.90)	0.67 (0.51, 0.88)	0.84 (0.62, 1.13)	0.74 (0.60, 0.91)	0.67 (0.61, 0.88)	0.85 (0.63, 1.16)
Model 1						
Low birthweight	1.12 (0.99, 1.25)	0.98 (0.84, 1.16)	1.26 (1.07, 1.48)	1.16 (0.98, 1.38)	0.96 (0.75, 1.22)	1.35 (1.06, 1.71)
Macrosomia	0.85 (0.68, 1.05)	0.79 (0.59, 1.05)	0.93 (0.67, 1.29)	0.86 (0.68, 1.07)	0.78 (0.58, 1.05)	0.96 (0.69, 1.33)
Model 2						
Low birthweight	1.14 (1.01, 1.28)	1.01 (0.86, 1.19)	1.27 (1.08, 1.50)	1.19 (1.00, 1.41)	0.99 (0.77, 1.27)	1.37 (1.08, 1.75)
Macrosomia	0.83 (0.66, 1.03)	0.76 (0.57, 1.02)	0.92 (0.67, 1.28)	0.84 (0.67, 1.05)	0.76 (0.57, 1.02)	0.95 (0.68, 1.32)
Model 3						
Low birthweight	1.09 (0.96, 1.23)	0.99 (0.83, 1.17)	1.19 (1.01, 1.42)	1.13 (0.95, 1.35)	0.96 (0.74, 1.25)	1.28 (1.01, 1.62)
Macrosomia	0.81 (0.65, 1.02)	0.78 (0.58, 1.06)	0.83 (0.59, 1.16)	0.82 (0.65, 1.03)	0.78 (0.58, 1.05)	0.85 (0.60, 1.19)
Model 4						
Low birthweight	1.10 (0.97, 1.25)	0.99 (0.82, 1.19)	1.22 (1.02, 1.45)	1.13 (0.94, 1.36)	0.95 (0.72, 1.25)	1.30 (1.01, 1.66)
Macrosomia	0.81 (0.63, 1.03)	0.78 (0.57, 1.07)	0.82 (0.58, 1.17)	0.81 (0.64, 1.04)	0.77 (0.56, 1.06)	0.83 (0.59, 1.20)
Model 5						
Low birthweight	1.03 (0.90, 1.17)	0.94 (0.78, 1.14)	1.12 (0.93, 1.34)	1.05 (0.87, 1.26)	0.88 (0.67, 1.17)	1.18 (0.92, 1.51)
Macrosomia	0.87 (0.68, 1.11)	0.84 (0.61, 1.16)	0.88 (0.62, 1.25)	0.88 (0.69, 1.12)	0.83 (0.60, 1.14)	0.90 (0.62, 1.29)

Model 1 – adjusted for maternal age, maternal education, maternal race/ethnicity, maternal BMI, marital status, survey year, state
Model 2 – Model 1 + number of previous livebirths
Model 3 – Model 2 + federal poverty level
Model 4 – Model 3 + WIC use
Model 5 – Model 4 + maternal smoking

Chapter 5: Aim 2—The association between prenatal food insecurity and breastfeeding initiation and exclusive breastfeeding duration

5.1. Introduction

Food insecurity, which is inadequate access to nutritionally-acceptable food, has been increasingly recognized as a determinant of poor health, including physical and mental health, and pregnancy outcomes such as gestational diabetes and low birthweight.¹⁻⁷ Recent national estimates suggest that 11.1% of U.S. households experience food insecurity, although the rate has been declining since peaking during the Great Recession in 2007.⁸ However, this prevalence still varies by state; for example, the average prevalence of food insecurity from 2016-18 was 16.8% in New Mexico, while New Hampshire had an average prevalence of 7.8%.⁸ Minority populations tend to be disproportionately affected, as well as pregnant and single mothers.^{8,9}

During pregnancy, food insecurity has the potential to influence fetal programming of poor health as a result of poor nutrition.^{10,11} In the postpartum period, food insecurity may affect infant feeding practices.^{12,13} From a biologic standpoint, if a mother is nutritionally depleted or does not have an appropriate intake of micronutrients (as a result of food insecurity), breastmilk quality may be affected or it may make it difficult for a mother to breastfeed for a longer duration.¹⁴⁻¹⁶ From a behavioral standpoint, food insecurity may be a chronic stressor and is suggested to be associated with prenatal and postpartum depressive and anxiety symptoms, which may contribute to decreased breastfeeding initiation and alter decision-making for infant feeding.¹⁷ Because breastfeeding is a source of food security for infants, it is important to support mothers

who may be suffering from food insecurity so that an infant's food supply is not compromised.

Current guidelines recommend that infants be exclusively breastfed for 6 months; whereas 40% of infants globally are breastfed exclusively through six months, this figure is only 24.9% in the U.S.^{18–20} Improving the proportion of infants who are breastfed and exclusively breastfed has remained an objective of Healthy People 2020.²⁰ Few studies have explored how maternal food insecurity may affect breastfeeding. Qualitative studies appear to suggest that household food insecurity may be associated with a shorter duration of breastfeeding. Additionally, an empirical study in Canada found that household food insecurity was associated with ceasing exclusive breastfeeding sooner.²¹ Limited studies in the U.S. have explored this association, particularly maternal experiences of food insecurity during pregnancy. Therefore, this investigation sought to determine whether or not mothers who experienced food insecurity during pregnancy were less likely to initiate breastfeeding, and have a shorter duration of exclusive breastfeeding.

5.2. Methods

Data source and analytic sample

This study was conducted using Oregon Pregnancy Risk Assessment Monitoring System (PRAMS) data of live births from 2008–2015 and the Oregon PRAMS-2 data follow-up survey of mothers re-interviewed two years later. Birth certificate records serve as a sampling frame for Oregon PRAMS, using a stratified, random sample to identify resident women who had a live birth. Women of racial/ethnic minorities and non-Hispanic white women who deliver a low birthweight infant are oversampled. The

analytic sample consisted of women with a singleton birth between 2008-2011 and 2014-2015, and who were followed-up in PRAMS-2 from 2011-2013 and 2016-2017. Due to changes in the PRAMS protocol, data were not sufficiently collected for live births from 2012-2013 (and their follow-up in 2014-2015) and were excluded from analysis. To reflect Oregon's population of mothers who deliver a live birth, responses were weighted for oversampling, nonresponse, and non-coverage. Additional details about Oregon PRAMS and PRAMS-2 methodology have been described elsewhere.^{22,23} This study received an exempt determination from the University of Maryland's Institutional Review Board.

Measures

The two main outcome variables were breastfeeding initiation and duration of exclusive breastfeeding. Breastfeeding initiation was determined by the question in PRAMS ("Did you ever breastfeed or pump breast milk to feed your new baby, even for a short period of time?"), and were dichotomized as 'yes' or 'no'. Duration of exclusive breastfeeding was calculated using both datasets, using the breastfeeding questions asking mothers to recall their total breastfeeding length and the age at which other liquids and/or solids were introduced. Exclusive breastfeeding duration was then determined by the minimum age of the infant when external liquids and/or solids were introduced.

The exposure variable, any prenatal food insecurity, was measured using the single food security question in Oregon PRAMS ("During the past 12 months before your new baby was born, did you ever eat less than you felt you should because there wasn't enough money to buy food?"). This single-item question was based on the USDA's 6-item abbreviated food security module.⁸ Responses to this question were dichotomized as

‘yes’ (food insecure) or ‘no’ (food secure). Because food insecurity may persist into the postpartum period, we additionally examined the effect of temporary (prenatal only) and prolonged food insecurity (women experiencing food insecurity in both the perinatal and postpartum period, using the same question in the follow-up PRAMS-2 as a proxy) specifically on the outcome of exclusive breastfeeding duration. Women who responded ‘yes’ to only the question in Oregon PRAMS were categorized as having experienced food insecurity in the prenatal period only, while women who responded ‘yes’ to both questions in Oregon PRAMS and PRAMS-2 were categorized as having prolonged food insecurity. Women who responded ‘no’ to both questions were categorized as food secure.

Potential covariates that were considered for the associations with breastfeeding initiation and exclusive duration were maternal age, maternal education, maternal race/ethnicity, marital status, and pre-pregnancy BMI, which were derived from the birth certificate. Age was analyzed as a categorical variable with the following categories: <20, 20-24, 25-29, 30-34, 35-39, 40+. Maternal educational attainment was categorized as: ‘less than high school’, ‘high school’, ‘some college’, and ‘college degree’. Maternal race/ethnicity was examined as: NH-White, NH-Black, Hispanic, and Other. Marital status at birth was dichotomized as ‘married’ or ‘not married’. Pre-pregnancy maternal BMI was categorized as underweight (<18.5 kg/m²), normal (18.5-24.9 kg/m²), overweight (25-29.9 kg/m²), and obese (≥30 kg/m²).

Furthermore, we included variables that were related to the socioeconomic status of the mother, such as previous livebirths, and income according to the federal poverty level (FPL). Previous livebirths was categorized as 0, 1, 2, and 3+, and the FPL was

calculated using questions on household income in the previous year and the number of people in the household that relied on the household income for each survey year.

Prenatal WIC participation and behavioral covariates, such as prenatal depression, and maternal smoking before and/or during pregnancy, were also considered in the association. Prenatal WIC participation was determined by the question in PRAMS, and was categorized as ‘yes’ or ‘no’. Prenatal depression was determined by the question in PRAMS on how often the mother felt down, depressed, or hopeless during their pregnancy. Mothers who responded ‘almost always’ or ‘always’ were considered to have prenatal depression, while mothers who responded ‘never’, ‘rarely’, or ‘sometimes’ were considered to not have prenatal depression. Maternal smoking was examined using responses to the following questions in PRAMS: “In the 3 months before you got pregnant, how many cigarettes did you smoke on an average day?” and “In the last 3 months of your pregnancy, how many cigarettes did you smoke on an average day?”. Mothers who responded as ‘I didn’t smoke then’ were classified as ‘non-smokers’. Mothers who responded to smoking any amount before pregnancy and not smoking during pregnancy were classified as ‘pre-pregnancy smoking only’. Mothers who smoked any amount before and during pregnancy were classified as ‘smoking before and during pregnancy’. Finally, survey year was also examined to adjust for any differences by year.

Analysis plan

The distribution of maternal and infant characteristics within the categories of prenatal food insecurity was examined. The prevalence of breastfeeding initiation by maternal characteristics was also examined. To examine the relationship between prenatal food insecurity and breastfeeding initiation, multivariable logistic regression was

conducted using a tiered approach to covariates. First, confounder-adjusted models were examined. Model 1a included demographic characteristics, such as maternal age, maternal education, marital status, race/ethnicity, maternal pre-pregnancy BMI, and survey year. Second, variables related to poverty were adjusted in a tiered fashion. Model 1b included number of previous live births in addition to the covariates included in Model 1a. Model 1c adjusted for income according to the FPL in addition to covariates in Model 1b. Potential behavioral mediators that may be a result of food insecurity were examined in separate models, including prenatal WIC participation, prenatal depression, and maternal smoking. Separate models were implemented because the design of the survey could not allow us to determine whether these factors occurred before food insecurity, in which case they would be confounders, or after, in which they would be considered mediators. Lastly, because preterm birth (before 37 weeks' gestation) may dictate the method of infant feeding in order to meet their nutritional needs,^{24,25} all models of the main analyses were also restricted to term births. Odds ratios (OR) and 95% confidence intervals (CI) for ever breastfed (vs. never breastfed) were estimated for all models.

For the outcome of exclusive breastfeeding duration, which was restricted to those women who initiated breastfeeding, Kaplan-Meier curves were constructed to visually inspect the differences between food insecure and food secure women. Accelerated failure time models (AFT) were used to examine the relationship between prenatal food insecurity and duration of exclusive breastfeeding. Because of the survey structure of the data, the appropriate distribution for the model was selected based on adjusted Wald tests fit on a generalized gamma distribution model. Women were right-censored at 6 months' (26.07 weeks) duration, given the national and international recommendations to

exclusively breastfeed for six months.^{18,19} Covariates were added in a tiered approach similar to the breastfeeding initiation analysis. With AFT models, time ratios (TR) and 95% confidence intervals (CI) were estimated. A time ratio >1 was interpreted as a longer duration of exclusive breastfeeding, while a time ratio <1 indicated a shorter duration of exclusive breastfeeding. Similar procedures were done when examining food insecurity categorically (prenatal only and prolonged food insecurity). All analyses were conducted using Stata 15 survey data analysis command, using weights to account for the complex sampling design of PRAMS-2.

5.3. Results

A final unweighted analytic sample of N=3,624 women from 2008-2015 was included for analyses. The weighted prevalence of prenatal food insecurity was 9.8% (unweighted n=332). Table 5.1 presents select maternal and infant characteristics by maternal food security status, weighted to represent the population of Oregon mothers during this time period. The majority of women were between 24-34, non-Hispanic white, and delivered at term. Food insecure women were more likely to be younger, be of a minority population, have a high school education or less, were under 100% of the FPL, have a delivery paid for by Medicaid, and participate in prenatal WIC compared to food secure mothers. Additionally, food insecure mothers had similar breastfeeding initiation, but appeared to have a lower median exclusive breastfeeding duration compared to food secure mothers.

Table 5.2 presents the unweighted N and weighted prevalence of breastfeeding by maternal characteristics and exposure to prenatal food insecurity. Breastfeeding initiation was very similar across maternal characteristics; however, mothers who were non-

Hispanic black, were not married, had less than a high school education, were below 100% of the FPL, participated in prenatal WIC, and who smoked had a lower prevalence of initiating breastfeeding.

Unadjusted and adjusted ORs for the relationship of prenatal food insecurity and breastfeeding initiation are presented in Table 5.3. Magnitudes of the unadjusted model results revealed that compared to mothers who were food secure during pregnancy, prenatal food insecurity was associated with decreased breastfeeding initiation, though this was non-significant (OR 0.88 [95%CI 0.39, 1.99]). Upon adjustment for sociodemographic variables, the direction of the effect reversed, indicating that food insecurity was associated with an increased odds of initiating breastfeeding (aOR 1.46 [95%CI 0.60, 3.60]). When looking at the covariates separately, maternal education and marital status were primarily responsible for reversing the effect of prenatal food insecurity from less likely to initiate breastfeeding to more likely to initiating breastfeed. The magnitude slightly increased among addition of confounders and potential behavioral mediators (i.e., WIC participation, prenatal depression and maternal smoking). When restricted to term births, similar findings were seen, though the magnitudes were slightly smaller.

Figure 5.1 depicts the Kaplan-Meier curve of exclusive breastfeeding duration by food insecurity status. Food insecure mothers appeared to have a lower duration of exclusive breastfeeding compared to food secure mothers. Unadjusted and adjusted TRs modeling the relationship of prenatal food insecurity and exclusive breastfeeding duration are presented in Table 5.4. Adjusted Wald tests indicated that the log normal distribution was the most appropriate fit, consistent with previous studies using AFT models for

breastfeeding duration.^{26,27} Additional model fit curves, including a plot of the hazard against the Kaplan-Meier are provided in Appendix B. Unadjusted results indicated that compared to mothers who were food secure during pregnancy, mothers who experienced food insecurity were associated with a shorter exclusive breastfeeding duration, though non-significant (TR 0.76 [95%CI 0.50, 1.17]). Adjusted models moved estimates closer to the null, but TR estimates did not change upon addition of each confounder and potential mediators. Similar patterns were also seen when restricted to term births; however, adjusted models appeared to show no difference in exclusive breastfeeding duration.

Additional analysis examining the effect of women who experienced food insecurity prenatally only and women experiencing food insecurity that persisted into the postpartum period on exclusive breastfeeding duration is presented in Table 5.5, and the Kaplan-Meier curve in Supplementary Figure 5.1. Women who only experienced prenatal food insecurity were more likely to have a shorter exclusive breastfeeding duration, though non-significant, and did not vary upon addition of sociodemographic covariates and potential mediators compared to food secure mothers. However, among women who experienced food insecurity that persisted into the postpartum period, magnitudes reversed in adjusted models and appeared to have a slightly longer duration of exclusive breastfeeding compared to food secure mothers. Similar patterns were seen when restricted to term births, though the magnitudes were slightly stronger among women experiencing persistent food insecurity. We additionally verified the sensitivity of our findings to the length of exclusive breastfeeding by using an earlier cessation of breastfeeding, given that the Kaplan-Meier curve (Supplementary Figure 5.1) crossed.

We therefore censored women at 2 months. Patterns were consistent with the analyses (results not shown).

5.4. Discussion

In this pregnant population, there were 9.8% of women experiencing food insecurity. During the time period of this analysis, national prevalence rates of household food insecurity ranged from 12.6 to 14.6%, while Oregon estimates ranged from 13.7 to 16.1%; however, estimates are widely different among couples with children (around 10%) vs. single mothers or single women (20 to 30%), highlighting the difference in this sample and the measurement of food insecurity.^{8,28} Unadjusted analyses from this study suggested that women experiencing prenatal food insecurity were less likely to initiate and sustain exclusive breastfeeding; however, much of this was explained by other underlying factors related to food insecurity, particularly maternal education and marital status. After adjustment, food insecure mothers were more likely to initiate breastfeeding than food secure mothers, but this was not significant. We found minimal differences in the duration of exclusive breastfeeding between food insecure and food secure mothers after adjustment. However, when examining food insecurity by its persistence, findings suggested an increased duration of exclusive breastfeeding among mothers experiencing food insecurity in both the prenatal and postpartum period compared with food secure mothers, while findings suggested an earlier cessation among mothers experiencing temporary food insecurity during pregnancy only.

Our finding of increased magnitudes of breastfeeding initiation among food insecure mothers appears to be consistent with the limited evidence from other quantitative and qualitative studies exploring food insecurity and breastfeeding. A

nationally-representative study conducted in Canada found that mothers living in food insecure households had similar likelihoods of initiating breastfeeding compared to food secure households.²¹ Within the U.S. context, a recently published multi-state cross-sectional analysis using PRAMS data, which also included Oregon, found that after adjusting for sociodemographic characteristics, food insecure women had a non-significant increased magnitude of breastfeeding, a reversal that was similar to our findings.²⁹ Since maternal education and marital status appeared to be responsible for the reversed effect in our analysis, and given their association with both food insecurity and breastfeeding,^{8,30} future studies should consider possible moderating effects.

Other evidence on breastfeeding initiation remains inconsistent. One study, primarily among Hispanic WIC participants found no difference in breastfeeding among food insecure households, whereas another study among WIC participants in Massachusetts found that prenatal household food insecurity was associated with a lower breastfeeding initiation.^{13,31} Similarly, another study using NHANES data found that household food insecurity was associated with a lower breastfeeding initiation.³² An additional qualitative study also found that mothers were less likely to breastfeed due to their perceived poor diet.¹² It should be noted, however, that these studies did not measure food insecurity at the individual-level like the current analysis, and therefore may not capture the individual experience of food insecurity; nonetheless, caution should be taken with these interpretations, as our estimates were imprecise due to the small sample size of mothers experiencing prenatal food insecurity (unweighted n=332). Furthermore, other research has also indicated that in times of household food insecurity, vulnerable populations such as children may be shielded from food insecurity by other household

members.³³ It is unclear whether pregnant women may also be similarly protected by other members in the household, and this analysis is unable to test such a hypothesis.⁹

In terms of exclusive breastfeeding duration, we found that any prenatal food insecurity (either temporary or persistent) appeared to have no influence on breastfeeding duration. However, when examining the categories separately, women experiencing food insecurity only during the prenatal period had a magnitude indicating a shorter duration of exclusive breastfeeding on average compared to food secure mothers, whereas mothers experiencing persistent food insecurity had a magnitude indicating a longer duration of exclusive breastfeeding on average. These findings were consistent when censoring women to an earlier exclusive breastfeeding cessation, as the proportion of women who remain breastfeeding longer becomes smaller. In addition, our finding that the magnitudes of exclusive breastfeeding duration attenuated when restricting to term births may be due to the specialized nutritional needs that some preterm infants require for optimal growth,^{24,25,34} or may be related to having a longer duration for mothers to report an experience of prenatal food insecurity.

Existing evidence is somewhat mixed regarding breastfeeding duration. In the previous multi-state PRAMS study, authors explored early breastfeeding cessation and found that food insecure women were likely to cease breastfeeding by the 4-6 week mark compared to women who breastfed ≥ 10 weeks.²⁹ However, authors used a multinomial analysis and did not have a longitudinal component like our study, nor did they explore exclusive breastfeeding duration. In another previously mentioned study among women participating in WIC in Massachusetts, authors found no association between household prenatal food insecurity during pregnancy and breastfeeding duration; however,

household food insecurity postpartum was associated with breastfeeding at three and six months compared to food secure mothers.³¹ In Canada, mothers with household food insecurity was associated with ceasing exclusive breastfeeding earlier, but household food insecurity may not represent individual experiences within the household, and there may be differences across populations on how food insecurity impacts breastfeeding.²¹

Paradoxically, breastfeeding offers a sense of food security for families, as resources do not need to be spent on infant formula.³⁴ Although supports from programs such as WIC may be helpful for food insecure mothers, and especially the program's breastfeeding education, we found that women who participated in prenatal WIC had a lower prevalence of breastfeeding. As such, the addition of WIC appeared to strengthen the magnitudes seen for breastfeeding initiation, and it may be possible that women who go on to participate in WIC intend not to breastfeed, possibly due to the benefit of infant formula that the program offers. Alternatively, breastfeeding may contribute to further food insecurity as it prevents mothers from (re)entering the workforce if they are trying to adhere to recommendations, particularly in settings where maternity leave is unavailable or where workplace policies do not provide lactation supports.³⁵ Therefore, it is unclear whether those experiencing prolonged food insecurity were breastfeeding longer because they could not return to work and reported continued food insecurity despite consuming fewer resources, or whether women experiencing prenatal food insecurity only had a shorter duration because they returned to work sooner and did not report continued food insecurity in the postpartum period.

Family-centered policies have the potential to help food insecure women overcome any breastfeeding barriers. Because WIC includes a breastfeeding education

component, it uniquely serves food insecure mothers, and should be leveraged to help improve breastfeeding rates. Additionally, continued insurance provisions, such as the Affordable Care Act's requirement to cover of breastfeeding supplies at no cost may also help to decrease the financial burden of having to purchase additional supports.³⁶ Finally, workplace and family leave policies should be examined, such as increasing lactation rooms and expanding current family leave laws to provide paid leave.³⁶ For example, California's paid maternity leave has shown to increase duration of breastfeeding, and few states have recently expanded paid medical leave, including Oregon.^{37,38} Given the important benefits of breastfeeding to both mother and infant,^{39,40} further research, including qualitative research, may be warranted on how such policies and interventions could be beneficial for mothers experiencing food insecurity.

Strengths & Limitations

Strengths of this analysis should be noted. To our knowledge, this is the first study to have explored individual-level experiences of maternal food security status (as opposed to household food security status) on breastfeeding outcomes. Additionally, Oregon PRAMS and PRAMS-2 are state-level population-based data, and given the longitudinal nature of the linked datasets, it allows us to establish temporality between exposure and outcome. These data are also generalizable to mothers in Oregon, due to the sampling structure and weighting of the data to represent Oregon women.

There are also some limitations in this analysis to note. First, and most importantly, there was only one question used to measure food insecurity. This definition does not capture all dimensions of food insecurity, such as affordability and availability of nutritious meals, as well as sufficiency of groceries and food. Therefore, we are unable

to determine the true extent of the food insecurity that the mothers are experiencing compared to using accepted food security modules. However, PRAMS is one of the few datasets that includes information on food insecurity, and other studies have also used this 1-item question.^{29,41} However, future surveys and/or studies should consider incorporating the full food insecurity module or even a 2-item measure that has been validated in both interview and survey methods.^{42,43} Compared to the PRAMS question, which asks about cutting meals, the 2-item measure is derived from two specific questions from the full 18-item measure related to stress experienced from sufficiency and affordability of food.

Second, the sample size for the exposure was small, and may account for the imprecise confidence intervals seen in the findings. Given the retrospective nature of the breastfeeding duration question, there may be the possibility of social desirability and recall bias, in which women may exaggerate the length of breastfeeding; however, evidence suggests that maternal recall of breastfeeding is usually accurate within three years of birth.^{44,45} Fourth, although benefits such as WIC and other safety-net benefits, such as the Supplemental Nutrition Assistance Program (SNAP), may also be beneficial in improving food security status or diet quality, we did not have enough numbers to meaningfully examine the effects of WIC, nor were we able to ascertain the effects of SNAP using this data. Additional research on how these policies support, and in particular the breastfeeding education that is part of WIC, may be improved for food insecure mothers is warranted. Finally, Oregon has a different racial and ethnic make-up and has been reported to have higher rates of breastfeeding compared to the rest of the country.²⁰ As racial and ethnic minorities are disproportionately affected by food

insecurity and may also differ in breastfeeding rates, additional data across the country are needed to fully ascertain the association of maternal food security status during pregnancy on breastfeeding outcomes. As such, our findings may not be generalizable beyond the state of Oregon.

Conclusion

In sum, this study suggests that food insecurity does not appear to deter women from breastfeeding or ceasing exclusive breastfeeding earlier particularly if it persists; however, there may be other confounding factors that complicate this relationship, such as maternal education and marital status. Further studies including a more representative sample and improved precision of food insecurity may be helpful in fully describing this association, and whether there may be moderating effects by specific factors. Further research on policies that consider the economic and caregiving responsibilities of food-insecure mothers so that they are supported at home may also be warranted. This information can be used to further understand barriers and facilitators of breastfeeding.

Table 5.1. Weighted percent distribution of selected maternal and infant characteristics by prenatal food insecurity status: Oregon PRAMS data, 2008-2015*

Maternal and infant characteristics	Total N (unweighted)	Food insecure¹	Food secure¹
Maternal characteristics at time of birth			
Maternal age at birth (years)	N=3624	N=332	N=3292
< 20	145	7.6	3.6
20-24	599	34.9	17.5
25-29	1043	30.5	32.4
30-34	1107	18.3	28.2
35-39	596	6.6	14.7
≥40	134	2.1	3.6
Maternal race/ethnicity	N=3612	N=331	N=3281
non-Hispanic white	1068	62.3	68.7
non-Hispanic black	256	2.4	1.8
Hispanic	815	26.9	18.8
Other ²	1473	8.3	10.6
Marital status	N=3621	N=332	N=3289
Not married	998	56.9	28.5
Married	2623	43.1	71.5
Maternal educational attainment	N=3611	N=330	N=3281
No high school diploma	522	21.7	13.9
High school diploma	653	40.8	22.5
Some college	1060	30.9	27.1
Bachelor's degree or higher	1376	6.5	36.5
Median federal poverty level (%)	201 [87, 349]	82 [48, 128]	214 [99, 396]
Federal poverty level	N=3466	N=316	N=3150
0-99%	991	62.4	25.2
100-185%	679	28.1	19.6
≥186%	1796	9.4	55.2
Insurance pre-pregnancy	N=3490	N=329	N=3161
Medicaid	561	20.2	16.3
Other	2128	30.5	61.8
Self-pay	801	49.3	22.0
Payment source at delivery	N=3606	N=330	N=3276
Medicaid	1450	73.4	37.9
Private	2034	22.1	59.5
Other	51	2.1	1.4
Self-pay	71	2.4	1.1
Prenatal WIC participation	N=3562	N=325	N=3188
Not eligible (FPL>185)	373	15.6	10.8
Eligible but not (FPL≤185)	1597	4.8	49.2

Yes	1543	79.6	40.0
Prenatal care sought	N=3435	N=313	N=3122
No	8	0.0	0.3
Yes	3427	100.0	99.7
Pre-pregnancy body mass index	N=3549	N=321	N=3228
Underweight	114	1.8	2.9
Normal	1784	441.0	49.5
Overweight	851	23.4	24.2
Obese	800	12.4	23.4
Smoking during pregnancy	N=3569	N=325	N=3244
Never	3011	59.5	83.8
Before only	339	20.4	9.6
Before and/or during	219	20.1	6.6
Prenatal depression	N=3557	N=323	N=3234
No	3240	79.5	92.3
Yes	317	20.5	7.7
Route of Delivery	N=3561	N=326	N=3235
Spontaneous vaginal	2393	67.8	69.4
Assisted vaginal	137	2.5	3.5
Cesarean section	1031	29.7	27.1

Infant characteristics at birth

Previous livebirths	N=3621	N=332	N=3289
None	1587	44.9	41.7
1	1173	25.2	34.5
2	510	16.7	14.2
≥3	351	13.2	9.5
Gestational week at birth	N=3624	N=332	N=3292
<37 weeks (preterm)	208	4.6	5.8
≥37 weeks	3416	95.4	94.2
Sex	N=3624	N=332	N=3292
Male	1881	54.1	53.8
Female	1743	45.9	46.2
Low birthweight	N=3624	N=332	N=3292
No	3464	95.8	96.0
Yes	160	4.2	4.0
Admission to NICU or infant transferred	N=3591	N=328	N=3263
No	3315	92.9	92.7
Yes	276	7.1	7.3
Ever breastfed infant	N=3624	N=332	N=3292
No	127	4.5	4.0
Yes	3497	95.5	96.0

Median total breastfeeding duration (weeks)	52.1 [26.1, 73.9]	52.1 [26.1, 69.5]	52.1 [26.1, 78.2]
Median exclusive breastfeeding duration (weeks)	21.7 [8.7, 30.4]	17.4 [4.3, 28.0]	21.7 [8.7, 30.4]

*2012-13 excluded from analysis

1 - weighted proportion

2 - includes Asian/Pacific Islander, American Indian or Alaska Native, Other, and Hispanic origin unknown or not stated.

Table 5.2. Prevalence of ever breastfed infant by maternal characteristics and exposure: Oregon PRAMS data, 2008-2015*

	Ever breastfed infant	
	Overall	
Total number of births	n	Percent ¹
Exposure		
Prenatal food insecurity		
No	3187	96.0
Yes	310	95.5
Maternal demographics		
Maternal age at birth (years)		
Under 20	138	95.8
20-24	578	96.2
25-29	1014	97.6
30-34	1067	95.5
35-39	570	93.5
40+	130	94.0
Maternal race/ethnicity		
non-Hispanic white	1032	95.9
non-Hispanic black	239	93.0
Hispanic	789	96.9
Other ²	1426	95.5
Marital status		
Not married	939	92.3
Married	2555	97.6
Maternal educational attainment		
No high school diploma or GED	488	92.3
High school diploma or GED	613	94.0
Some college	1024	96.8
Bachelor's degree or higher	1360	98.5
Federal poverty level		
0-99%	931	92.7
100-185%	658	97.6
≥186%	1757	97.5
Prenatal WIC participation		
Not eligible (FPL>185)	1567	97.7
Eligible but not (FPL≤185)	364	97.0
Yes	1455	93.7
Prenatal care sought		
No	8	100.0

Yes	3308	96.0
Pre-pregnancy body mass index		
Underweight	111	95.2
Normal	1733	97.0
Overweight	816	95.6
Obese	767	94.9
Smoking during pregnancy		
Never	2924	96.9
Before only	328	95.8
Before and/or during	191	86.0
Prenatal depression		
No	3128	96.2
Yes	306	94.0

*2012-13 excluded from analysis

1 - weighted prevalence

2 - includes Asian/Pacific Islander, American Indian or Alaska Native, Other, and Hispanic origin unknown or not stated.

Table 5.3. Unadjusted and adjusted OR of prenatal food insecurity and ever breastfed overall and by term births: Oregon PRAMS data, 2008-2015*

	Prenatal FI	
	Overall OR (95% CI)	Term births (37+) OR (95% CI)
Breastfeeding initiation		
Unadjusted	0.88 (0.39, 1.99)	0.87 (0.37, 2.02)
Confounder-adjusted models		
Model 1a	1.34 (0.57, 3.13)	1.23 (0.50, 3.00)
Model 1b	1.31 (0.56, 3.05)	1.18 (0.49, 2.84)
Model 1c	1.41 (0.58, 3.47)	1.24 (0.49, 3.10)
Models adjusted for potential mediators		
Model 2	1.46 (0.60, 3.60)	1.30 (0.51, 3.28)
Model 3	1.42 (0.56, 3.56)	1.24 (0.48, 3.20)
Model 4	1.56 (0.61, 4.00)	1.32 (0.50, 3.46)

*2012-13 excluded from analysis

1a – adjusted for maternal age, maternal education, maternal race/ethnicity, maternal BMI, marital status, survey year

1b – Model 1a + number of previous live births

1c – Model 1b + federal poverty level

2 – Model 1c + WIC use

3 – Model 4 + prenatal depression

4 – Model 5 + maternal smoking

Table 5.4. Unadjusted and adjusted TR of prenatal food insecurity and exclusive breastfeeding duration overall and by term births: Oregon PRAMS data, 2008-2015*

	Prenatal FI	
	Overall TR (95% CI)	Term births (37+) TR (95% CI)
Exclusive breastfeeding duration		
Unadjusted	0.76 (0.50, 1.17)	0.82 (0.53, 1.25)
Confounder-adjusted models		
Model 1a	0.90 (0.59, 1.38)	0.98 (0.64, 1.50)
Model 1b	0.90 (0.59, 1.39)	0.98 (0.64, 1.51)
Model 1c	0.89 (0.57, 1.39)	0.95 (0.61, 1.48)
Models adjusted for potential mediators		
Model 2	0.90 (0.58, 1.41)	0.96 (0.61, 1.49)
Model 3	0.90 (0.57, 1.41)	0.96 (0.61, 1.52)
Model 4	0.91 (0.58, 1.44)	0.99 (0.62, 1.57)

*2012-13 excluded from analysis

1a – adjusted for maternal age, maternal education, maternal race/ethnicity, maternal BMI, marital status, survey year

1b – Model 1a + number of previous live births

1c – Model 1b + federal poverty level

2 – Model 1c + WIC use

3 – Model 2 + prenatal depression

4 – Model 3 + maternal smoking

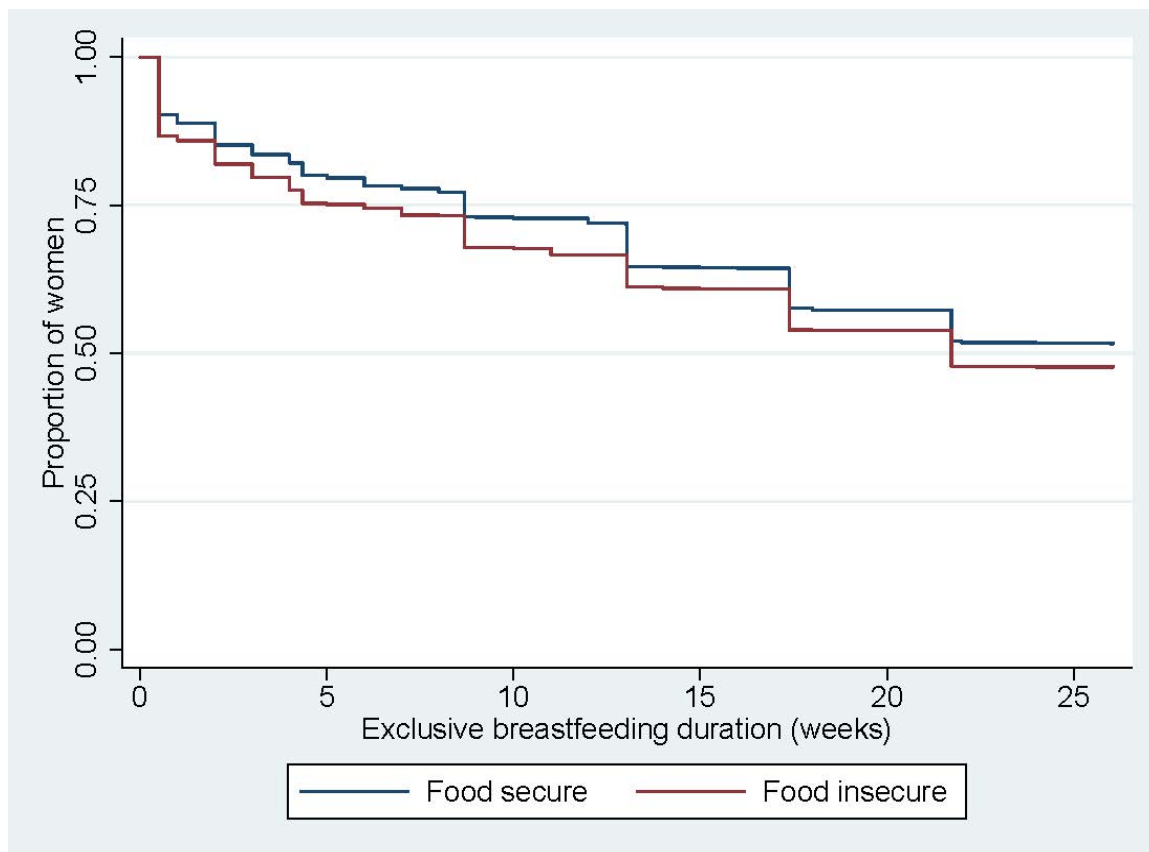


Figure 5.1. Kaplan-Meier curve of the proportion of women who remained exclusively breastfeeding their infant. The blue line indicates food secure women, while the dark red line indicates food insecure women.

Table 5.5. Unadjusted and adjusted TR of type of maternal food insecurity and exclusive breastfeeding duration overall and by term births: Oregon PRAMS data, 2008-2015*

	Prenatal FI	
	Overall TR (95% CI)	Term births (37+) TR (95% CI)
Exclusive breastfeeding duration		
Unadjusted		
Prenatal FI only	0.62 (0.35, 1.13)	0.68 (0.37, 1.26)
Persistent FI	0.88 (0.48, 1.60)	0.94 (0.53, 1.68)
Confounder-adjusted models		
Model 1a		
Prenatal FI only	0.72 (0.40, 1.31)	0.81 (0.44, 1.49)
Persistent FI	1.11 (0.62, 2.01)	1.21 (0.68, 2.12)
Model 1b		
Prenatal FI only	0.72 (0.40, 1.33)	0.81 (0.43, 1.50)
Persistent FI	1.10 (0.60, 1.99)	1.19 (0.67, 2.10)
Model 1c		
Prenatal FI only	0.68 (0.37, 1.27)	0.74 (0.39, 1.40)
Persistent FI	1.08 (0.59, 2.01)	1.18 (0.65, 2.12)
Models adjusted for potential mediators		
Model 2		
Prenatal FI only	0.69 (0.37, 1.28)	0.75 (0.40, 1.40)
Persistent FI	1.10 (0.59, 2.05)	1.19 (0.65, 2.15)
Model 3		
Prenatal FI only	0.69 (0.37, 1.30)	0.77 (0.40, 1.47)
Persistent FI	1.10 (0.58, 2.08)	1.20 (0.65, 2.21)
Model 4		
Prenatal FI only	0.69 (0.36, 1.32)	0.77 (0.40, 1.49)
Persistent FI	1.18 (0.62, 2.23)	1.28 (0.68, 2.83)

*2012-13 excluded from analysis

1a – adjusted for maternal age, maternal education, maternal race/ethnicity, maternal BMI, marital status, survey year

1b – Model 1a + number of previous live births

1c – Model 1b + federal poverty level

2 – Model 1c + WIC use

3 – Model 2 + prenatal depression

4 – Model 3 + maternal smoking

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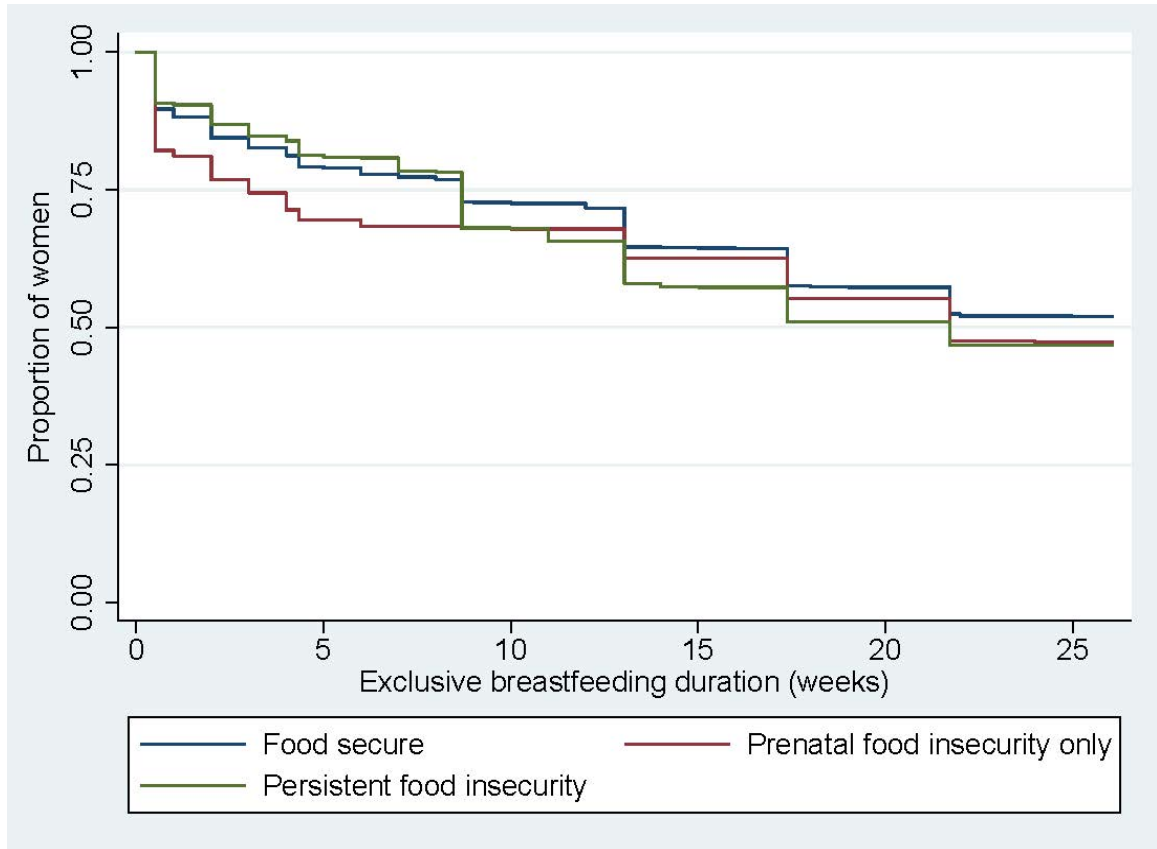
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SUPPLEMENTAL APPENDIX



Supplementary Figure 5.1. Kaplan-Meier curve of the proportion of women who remained exclusively breastfeeding their infant by type of food insecurity. The blue line denotes food secure women, the dark red line denotes food insecure women, and the green line denotes women experiencing food insecurity that persisted into the postpartum period.

Chapter 6: Aim 3—Maternal food security status and childhood obesity at 2 years: a sexually-dimorphic response?

6.1. Introduction

In the U.S., pediatric obesity, defined as being at or above the age-specific and sex-specific 95th percentile for BMI, has been on the rise.¹ Among youth aged 2-19, the prevalence of obesity was 18.5% in 2015-2016, up from 16.1% in 2008.² Among preschool-aged children (2-5 years), the prevalence was 13.9%.² Pediatric obesity is a public health concern as it can track throughout childhood and into adulthood, putting them at increased risk of developing chronic disease and comorbidities later on in life.³⁻⁵ While individual-level factors such as metabolism and genetics may predispose a child to have obesity,⁶⁻⁸ modifiable risk factors which are amenable to intervention should be prioritized to promote prevention. For example, the Life Course Health Development framework and the Developmental Origins of Health and Disease (DOHaD) hypothesis tells us that critical periods, or the first 1,000 days since conception, are an important time for growth and development.⁹⁻¹¹ Therefore, exposures, such as poor maternal nutrition, occurring in utero and early postnatal periods, may alter the growth trajectory of the child and should be considered.^{9,11-13}

Research has shown that poor maternal nutrition during pregnancy may influence fetal programming that is carried out by the placenta at the cellular level, which in turn may alter the metabolism of the fetus and lead to obesity and metabolic syndrome later in adulthood.^{14,15} These differences may be more pronounced among term births, given that the third trimester is a time of rapid adipose deposition.^{16,17} In the postpartum period, and especially during lactation, poor maternal nutrition may, from a biological perspective,

affect breastmilk quality and, through continued epigenetic programming, determine adiposity deposition in offspring because organs are still continuing to mature,^{18,19} or from a behavioral perspective, influence the decision-making in a child's food consumption.²⁰

Additionally, studies and animal models suggest that there may be a sexually dimorphic response to changes in the nutritional environment prenatally and postnatally due to the differences in how males and females grow in utero.²¹⁻²³ Some of these sex-specific differences in developing obesity may be due to genetics, as the genes on the X chromosomes have been shown to regulate body weight, and some may be due to sex-specific sensitivities to metabolic hormones that maintain glucose homeostasis, such as leptin and insulin.²⁴ Postnatally, animal models also suggest that composition of maternal milk is sex-specific, and poor maternal nutrition may affect metabolic hormones present in breastmilk that elicits sex-specific programming of glucose homeostasis, appetite, and eating patterns in offspring.²⁵⁻²⁸

Food insecurity may be one way that compromises the mother's nutritional status during the prenatal and early postnatal period, and has been increasingly recognized as a determinant of other poor health and behavioral outcomes, including low birthweight, gestational diabetes, increased BMI, and poor mental health.²⁹⁻³² Recent data indicate that 11.1% of U.S. households suffer from food insecurity, and it may be temporary or chronic.³³ Of note, pregnant women and single mothers are disproportionately affected.^{33,34} Although social supports such as the Women, Infants, and Child (WIC) program exist, many mothers still suffer from food insecurity, including those not eligible for such programs; among pregnant women in households <300% of the federal poverty level (FPL), 32% are marginally or food insecure.³⁴ Thus, because women are supporting

a developing fetus and require increased nutritional needs, any changes that make it harder to meet those needs such as food insecurity may influence fetal programming of poor health.

In light of the DOHaD hypothesis and research supporting differences by infant sex, few studies have explored the effects of maternal food insecurity during pregnancy and postpartum on early child outcomes such as obesity. This may, in part, be due to limited data sources with longitudinal data on both food insecurity and child health outcomes. Therefore, this investigation sought to explore the association between maternal food insecurity and childhood obesity at 2 years of age, and whether associations are modified by sex of the child.

6.2. Methods

Data source and analytic sample

This study was conducted using Oregon Pregnancy Risk Assessment Monitoring System (PRAMS) data of live births from 2008-2015 and the Oregon PRAMS-2 data follow-up survey of mothers who were re-interviewed two years later. The sampling frame for Oregon PRAMS was birth certificate records, using a stratified, random sample to identify resident women who had a live birth. Women of racial/ethnic minorities and non-Hispanic white women who delivered a low birthweight infant were oversampled. The analytic sample consisted of women with a singleton birth between 2008-2011 and 2014-2015, and who were followed up in PRAMS-2 from 2011-2013 and 2016-2017 with valid information on exposure (maternal food insecurity) and outcome (obesity at 2 years). Live births from 2012-2013 (and their follow-up in 2014-2015) were excluded from analyses due to data not being sufficiently collected as a result of changes in the

Centers for Disease Control and Prevention (CDC) PRAMS protocol. Responses were weighted for oversampling, nonresponse, and non-coverage in order to reflect Oregon's population of mothers who deliver a live birth. Further details about CDC PRAMS, Oregon PRAMS and PRAMS-2 methodology have been described elsewhere.^{35,36} This study received an exempt determination from the University of Maryland's Institutional Review Board.

Measures

The exposure variable was any maternal food insecurity, which was measured using the sole food security question in both Oregon PRAMS and PRAMS-2 ("During the past 12 months before your new baby was born, did you ever eat less than you felt you should because there wasn't enough money to buy food?" [prenatal food insecurity in PRAMS] and "In the past 12 months, did you ever eat less than you felt you should because there wasn't enough money to buy food?" [postpartum food insecurity in PRAMS-2]). The food insecurity question was based on certain questions (i.e., food cutting and availability of resources) of the USDA's 6-item abbreviated food security module that includes other questions on food availability, sufficiency, and resources.³³ Respondents who answered 'yes' to either of these two questions in PRAMS and PRAMS-2 were dichotomized as 'any food insecurity' or 'food secure' if they answered 'no' to both questions.

The outcome variable for this study was obesity status of the toddler at follow-up in PRAMS-2. Obesity status was determined using body mass index (BMI) as a proxy, and defined as at or greater than the age- and gender-specific 95th percentile as per accepted pediatric charts by the Centers for Disease Control (CDC) for children aged 2-

19.³⁷ BMI was calculated using questions on the child's height and weight. A SAS macro to calculate BMI percentiles for pediatric obesity is provided by the CDC online.³⁷ Values of BMI ≥ 50 kg/m² were excluded, as these are implausible values for children according to the percentile graphs, and may indicate errors in recording height.³⁸

Potential covariates considered for the association between maternal food insecurity and child obesity included maternal age, maternal education, maternal race/ethnicity, marital status, and pre-pregnancy BMI, which were derived from the birth certificate. Age was analyzed as a categorical variable with the following categories: <20, 20-24, 25-29, 30-34, 35-39, 40+. Maternal educational attainment was categorized as: 'less than high school', 'high school', 'some college', and 'college degree'. Maternal race/ethnicity was examined as: NH-White, NH-Black, Hispanic, and Other. Marital status at birth was dichotomized as 'married' or 'not married'. Pre-pregnancy maternal BMI was categorized as underweight (<18.5 kg/m²), normal (18.5-24.9 kg/m²), overweight (25-29.9 kg/m²), and obese (≥ 30 kg/m²).

Furthermore, we included variables that were related to the socioeconomic status of the mother, such as previous livebirths, and income according to the federal poverty level (FPL). Previous livebirths was categorized as 0, 1, 2, and 3+, and the FPL was calculated using questions on household income in the previous year and the number of people in the household that relied on the household income for each survey year.

Behavioral covariates, such as prenatal depression, and maternal smoking before and/or during pregnancy, and prenatal WIC participation were also considered in the association. Prenatal depression was determined by the question in PRAMS on how often the mother felt down, depressed, or hopeless during their pregnancy. Mothers who

responded ‘almost always’ or ‘always’ were considered to have prenatal depression, while mothers who responded ‘never’, ‘rarely’, or ‘sometimes’ were considered to not have prenatal depression. Maternal smoking was examined using responses to questions on smoking in the prior 3 months and last 3 months of pregnancy and . Mothers who responded as ‘I didn’t smoke then’ were classified as ‘non-smokers’. Mothers who responded to smoking any amount before pregnancy and not smoking during pregnancy were classified as ‘pre-pregnancy smoking only’. Mothers who smoked any amount before and during pregnancy were classified as ‘smoking before and during pregnancy’. Prenatal WIC participation was determined by the question in PRAMS, and was categorized as ‘yes’ or ‘no’. Finally, survey year was also examined to adjust for any differences by year.

Analysis plan

The distribution of maternal and infant characteristics within the categories of maternal food insecurity and by sex of the child were examined. The prevalence of obesity by maternal characteristics was also examined in each respective group. Multivariable logistic regression was used to model the relationship between maternal food insecurity and obesity status at two years of age, with a tiered approach to covariates. First, Model 1a included demographic characteristics, such as maternal age, maternal education, marital status, race/ethnicity, maternal pre-pregnancy BMI, and survey year. Second, variables related to poverty were included. Model 1b included number of previous live births in addition to covariates included in Model 1a. Finally Model 1c included income according to the federal poverty level (FPL) in addition to covariates adjusted for in Model 1b. Because experiences of food insecurity may also

influence behavioral factors that could impact child obesity and act as mediators, we also examined three additional factors (prenatal depression, maternal smoking, and prenatal WIC participation). Given the design of the survey, we could not determine whether these factors preceded food insecurity, and thus, act as potential confounders, or occurred after, in which case they would be considered mediators. Therefore, we modeled them in separate models using a tiered approach. Model 2 included all the confounders in Model 1c and prenatal depression. Model 3 included covariates included in Model 2 and maternal smoking status. Finally, Model 4 included covariates in Model 3 in addition to prenatal WIC participation. Additionally, because of the large proportion of males who were born preterm (before 37 weeks) and the fact that preterm birth may affect infant growth and feeding due to its multifactorial etiology,³⁹ models were also restricted to term births. Variance inflation factor (VIF) for multicollinearity was examined for models.

Odds ratios (OR) and 95% confidence intervals (CI) for the association between food insecurity and obesity (vs. not obese) were estimated. Given the possible sexual-dimorphic response to the nutritional environment of the infant, we stratified results by sex of the child a priori. Additionally, because food insecurity may be a temporary or chronic phenomenon, a sensitivity analysis was conducted classifying maternal food insecurity by phase of pregnancy in which it occurred: prenatal food insecurity only, postpartum food insecurity only, and persistent food insecurity, which included mothers that experienced food insecurity in both the prenatal and postpartum periods. All analyses were conducted using Stata 15 survey data analysis command, using weights to account for the complex sampling design of PRAMS-2.

6.3. Results

The final unweighted analytic sample included N=3,022 women from 2008-2015. The weighted prevalence of any maternal food insecurity overall was 16.5% (unweighted N=459). Table 6.1 presents select maternal and toddler characteristics by maternal food security status and by sex of the child, weighted to represent the population of Oregon mothers during this time period. Overall, the majority of women were between 24-34, non-Hispanic white, and delivered at term (37+ weeks). Food insecure women were more likely to have a high school education or less, were under 100% of the FPL, have a delivery paid for by Medicaid, participated in prenatal WIC, and were obese compared to food secure mothers. Additionally, food insecure women were more likely to have a child who was above the 95th percentile for BMI at 2 years.

Among Oregon mothers who had a male offspring, food insecure women were more likely to be younger, non-Hispanic white, not married, have a high school education or less, were under 185% of the FPL, participated in prenatal WIC, and were smokers compared to food secure women. Similar distributions were seen among food insecure mothers who had a female offspring compared to food secure women. When comparing differences by sex of offspring, food insecure women who had a male offspring were likely to be younger, had a higher non-Hispanic white population, were not married, had lower levels of education, had a lower poverty level, had a delivery by Medicaid, participate in prenatal WIC, and had a lower prevalence of smoking compared to food insecure women who had a female offspring. Food insecure women who had male offspring were also more likely to be born preterm (<37 weeks), admitted to the NICU, and had a higher prevalence of obesity at 2 years compared to food insecure women who

had female offspring. Patterns among food secure women were not widely different by sex of the offspring.

The prevalence of obesity by maternal characteristics and exposure overall and by sex of the child, with unweighted N and weighted percentage is presented in Table 6.2. There was a higher prevalence of obesity among food insecure mothers who had male children compared to women food insecure women who had female children. Non-Hispanic black and Hispanic women and women with no high school diploma were more likely to have an offspring that was obese. In addition, women who were smokers and had prenatal depression were more likely to have a daughter who was obese at 2 years.

Unadjusted and adjusted ORs modeling maternal food security status and obesity in the child at 2 years of age and stratified by sex of the child for all births and term births are presented in Table 6.3. Unadjusted results revealed that compared to mothers who were food secure, maternal food insecurity was associated with an increased odds of having an offspring with obesity at 2 years of age for all births. Stratified analyses suggested that the magnitude of effect was stronger in male offspring of food insecure mothers compared with female offspring of food insecure mothers. Adjustment for sociodemographic variables appeared to explain part of the overall association, as the magnitudes decreased and became non-significant. When stratified by sex, the magnitude of the odds ratio of obesity was stronger among food insecure mothers who had a male offspring, though non-significant. Among female offspring, the magnitude of the association reversed regardless of gestational age, though non-significant. Addition of potential behavioral mediators (i.e., prenatal depression, maternal smoking, and prenatal WIC participation) in the model appeared to explain the effect of maternal food security

on obesity in the offspring overall. When stratified by sex, the addition of the mediators moved the estimate away from the null among female offspring, while the magnitudes decreased very little among male offspring. Patterns were similar when restricting to term births; however, the overall magnitude of association was larger among males for all models. It should be noted that the VIF results did not reveal any multicollinearity between confounders.

Figure 6.1 depicts the marginal predicted mean obesity at 2 years by sex of the child and maternal food security status for all births (1A) and term births (1B), holding all other covariates constant using Model 1c. The predicted mean obesity among male offspring born to food insecure mothers appear to be higher than food secure mothers, whereas female offspring had similar predicted means regardless of maternal food security status. The difference in predicted means within sexes was not significant ($p=0.176$ and 0.555 for males and females, respectively) nor when restricted to term births ($p=0.085$ and 0.504 for males and females, respectively).

Sensitivity analysis where maternal food insecurity status was further classified into three categories (prenatal food insecurity only, postpartum food insecurity only, and persistent food insecurity) is presented in Supplementary Table 6.1. Magnitudes of risk of obesity still held among male offspring among mothers experiencing food insecurity during pregnancy or postpartum compared to food secure mothers. Similar magnitudes of the association were also seen among female offspring as in the main analysis. Persistent food insecurity appeared to have no effect on obesity on either sex of the child for all births. Among term births, postpartum food insecurity remained significantly associated

with odds of obesity; however, persistent food insecurity was associated with a decreased odds of obesity among female term births.

6.4. Discussion

In our sample, the prevalence of any food insecurity and obesity in this population was 16.5% and 18.6% respectively; however, national estimates of food insecurity ranged from 12.6 to 14.6% during this time period, and estimates of early childhood obesity (ages 2-5) ranged from 8.4 to 13.9%, highlighting the importance of addressing these issues.^{33,40} This study suggests that the association between maternal food insecurity status and child obesity at 2 years of age varies by sex of the child. Maternal food insecurity showed an increased magnitude of obesity among male offspring compared to food secure mothers, whereas maternal food insecurity had no effect on obesity among female offspring compared to food secure mothers. Restricting to term births showed slightly elevated magnitudes among women experiencing prenatal and postpartum insecurity that were driven by male offspring. While the magnitude of association suggested an increased odds of obesity, this estimate was imprecise due to the small sample size of those experiencing food insecurity.

Our finding of the differential effect by sex of the toddler appears to be consistent with previous research that indicate that early life programming, for which the placenta is mainly responsible, may have a long-term sex-specific response to under- or overnutrition during critical periods.²⁴ While sex differences are continuing to be delineated, research from the Dutch famine and experimental animal models suggest that males are more vulnerable to the mother's current diet, whereas female offspring are vulnerable to a mother's lifetime nutrition and metabolism.²² For example, in the case of

overnutrition, studies among mothers with gestational diabetes and hyperglycemia was associated with an increased BMI at 5-7 years or adiposity in male offspring but not female offspring,⁴¹⁻⁴³ while maternal BMI was a predictor of adiposity in female offspring.⁴⁴ Additional animal models have found that offspring of pregnant rats that were nutrient restricted during pregnancy were likely to be obese later in life and have different gene expression in adipose tissue, with females being unaffected.²⁴ It is suggested that the differing responses to stresses occurring during pregnancy may be due to how placental genes are regulated differently by sex.²² Because boys grow faster than girls in utero, changes to a mother's current diet causes the placental surface to expand in order to sustain the rapid fetal growth among male offspring, while females adapt the growth rate in response to a poor nutritional environment.²² Additional animal models suggest that females are vulnerable to programming of glucose homeostasis by the nutritional environment, while males are vulnerable to programming of adiposity and body weight.⁴⁵⁻⁴⁷ Ultimately, these different compensatory survival mechanisms may translate to adverse birth outcomes and poor health outcomes in later life.²²

It is worthy to note that in our sample, there was a higher proportion of males born preterm among food insecure mothers than females (12.8% vs 2.8% for males and females respectively). Restricting to term births strengthened the association, which appears to be largely driven by the response among male offspring in our sample. The higher magnitudes seen among term births may be because preterm birth is a multi-etiological process that affects infant growth differently,³⁹ or reflects the length of time that the mother may have experienced food insecurity. Thus, restricting to term births helps to account for some of these factors.

Our finding that the magnitudes held when looking at transient food insecurity (i.e., food insecurity in either the prenatal or the postpartum period only) also suggest that these windows of development are important. In the postnatal period, and particularly during lactation, poor maternal nutrition may increase specific fatty acids in the breastmilk that may not be absorbed well by the infant's developing digestive system, leading to increased hunger.²⁶ Additionally, studies primarily done in animals suggest that poor maternal nutrition may also play a role in programming organ systems involved in glucose homeostasis, appetite, and eating patterns in offspring as a result of specific metabolic hormones present in breastmilk, such as leptin and insulin.²⁶⁻²⁸ For example, male rat offspring that were exposed to a maternal high-fat diet in pregnancy and lactation exhibited decreased leptin sensitivity, which is implicated in obesity,⁴⁸ while females only exhibited this from prenatal exposure.⁴⁹ An additional animal study showed that only female offspring exposed to maternal overnutrition during either pregnancy or postpartum exhibited programming of glucose homeostasis in utero.⁴⁷ Although our data are unable to ascertain the mother's nutritional impact from food insecurity, these studies seem to suggest that perhaps critical windows of when homeostasis systems may be vulnerable to programming effects differ between the sexes.²⁴ It should be noted that animal models may not necessarily translate to human studies; however, they provide some insight and it is important that future research continue to explore these differences by sex.

While these biologic factors may be important in the postnatal period, there may also be contextual factors specific to food insecurity and its related domains (e.g., affordability or availability) that may affect a child's diet in the early years that increase

risk of obesity. For example, a previous study using Oregon PRAMS-2 suggests that postpartum maternal food insecurity was associated with an increased likelihood of drinking soda and decreased consumption of fruit and vegetables.²⁰

Interestingly, our findings suggest that persistent food insecurity had little to no effect on obesity in the offspring unlike mothers that experienced transient food insecurity, despite these windows of development overlapping. In line with the DOHaD hypothesis, such a finding could be that since the nutritional environment in utero is the same postnatally, the fetus has already adapted to that environment and may have a lower risk, unlike a mismatched nutritional environment, in which the risk for obesity and poor health increases.⁵⁰ Alternatively, previous research on food insecure households with children has shown that there may be a buffering effect, in which parents or other older members may compromise their nutrition in order to protect children from compromising their nutrition.^{51,52} If mothers are indeed suffering from chronic food insecurity within the household, it is unclear whether a similar buffering mechanism may be occurring with pregnant women, in which another household member may be protecting the pregnant woman from compromising their nutrition status.³⁴ However, this interpretation should be taken with caution as the sample size of this specific group of women is very small, and such a hypothesis is unable to be tested specifically using this data. Future research on the effects of chronic food insecurity are needed.

Strengths & Limitations

There are strengths of this analysis that should be noted. To our knowledge, this study is the first to have explored maternal food security status on infant outcomes. Second, data are limited that contain longitudinal data to assess maternal food insecurity

and later child development. In addition, given that Oregon PRAMS and PRAMS-2 are state-level population-based data, and given the longitudinal nature of the linked datasets, it allows us to be certain of the temporality between exposure and outcome. Additionally, these data are generalizable to mothers in Oregon due to the sampling and weighting of the data to represent Oregon women.

This analysis also has some limitations to note. First, only one question was used to measure food insecurity in the prenatal and postpartum period in Oregon PRAMS, and is therefore unable to determine the extent of a mother's food insecurity or whether she is suffering from under- or overnutrition. Although the single-item food insecurity question is adapted from USDA's six-item measure, it does not encompass the entire construct of food insecurity that the full module measures, which includes dimensions of affordability and availability of nutritious meals, as well as sufficiency of groceries and food.³³ Therefore, this measure lacks the precision to know the true extent of the food insecurity in the prenatal and postnatal period. Nonetheless, this question in PRAMS is still considered an indicator of food insecurity as per the USDA, and it does not discount the fact that women may still be coping by eating a poorer quality of food at low cost compared to if food insecurity were measured by the longer food security modules or other diet-related questions. To improve accuracy of the measure, future surveys and/or studies could consider incorporating the full food insecurity module or even a recently validated 2-item measure, derived from two questions from the full 18-item measure related to stress from sufficiency and affordability of food.^{53,54}

Second, maternal report of height and weight were used to measure the child's BMI, which may result in measurement error. Although we excluded cases that were implausible ($\geq 50 \text{ kg/m}^2$),³⁸ future studies should consider biometric data that were directly collected to better assess obesity, including body fat percentage, waist circumference, or clinical tests that are also associated with obesity. Therefore, this study can only be compared to other studies that similarly use parental reports of height and weight.

Third, the sample size for the exposure in this analysis was small, which may explain the wide confidence intervals seen in the findings. Fourth, although benefits such as WIC may help to provide a means of improving a mother's diet quality, other safety-net benefits, such as the Supplemental Nutrition Assistance Program (SNAP) may also be beneficial in improving food security status; however, we are unable to ascertain the effects of SNAP using these data. However, it should be noted that SNAP may not improve diet quality the same way that WIC does, due to the eligible foods that WIC allows.⁵⁵ Further study may be warranted on how these existing supports or new interventions may take into account developmental programming and its potential long-term sexually dimorphic response. Finally, it should be noted that Oregon has a different racial and ethnic make-up compared to the rest of the country. Given that racial and ethnic minorities are disproportionately affected by obesity and/or food insecurity and may also differ in body composition, additional data across the country are needed to fully ascertain the association of maternal food security status during critical periods of child development on child obesity outcomes. Thus, findings may not be generalizable beyond the state of Oregon.

Conclusion

In summary, maternal food insecurity is prevalent in the U.S. In our sample in Oregon, 16.5% were experiencing food insecurity. This study suggests that the overall association of maternal food insecurity on early childhood obesity appears to be explained by sociodemographic factors; however, findings suggest that temporary food insecurity during critical periods may be more important than persistent food insecurity. Additionally, there appears to be a long-term sex-specific response to food insecurity, with a stronger effect among male offspring. While the magnitude of the association was suggestive of a potential effect on childhood obesity, the limited sample size and imprecise measure of food insecurity precluded more definitive conclusions. Future studies should include a more representative sample and improved measurement of food insecurity to fully ascertain the effects of maternal food security status on childhood obesity, and whether a sexually dimorphic response truly exists. These investigations will help to better understand how important changes to the in utero environment are and inform interventions that may be necessary to improve the long-term health of males and females.

**Table 6.1. Weighted percent distribution of selected maternal and infant characteristics by food insecurity status:
Oregon PRAMS data, 2008-2015***

	Total N (unweighted)	Food insecure ¹			Food secure ¹		
		Overall	Male	Female	Overall	Male	Female
Maternal characteristics at time of birth							
Maternal age at birth (years)	N=3022						
< 20	116	7.7	11.3	3.7	3	2.7	3.3
20-24	498	36.3	32.0	41.2	15.7	15.7	15.7
25-29	865	28.4	29.1	27.6	32.8	34.7	30.5
30-34	924	16.7	15.2	18.4	29.6	28.5	30.9
35-39	504	9.3	10.0	8.5	15.0	14.3	15.9
≥40	115	1.5	2.4	0.6	3.9	4.1	3.6
Maternal race/ethnicity	N=3011						
non-Hispanic white	940	66.7	67.6	65.5	71.0	71.2	70.9
non-Hispanic black	202	2.2	1.8	2.7	1.6	1.5	1.8
Hispanic	629	23.5	23.5	23.5	16.6	17.2	15.9
Other ²	1240	7.6	7.1	8.2	10.7	10.1	11.4
Marital status	N=3,018						
Not married	795	53.0	56.0	49.7	25.1	24.5	25.8
Married	2223	27.0	44.0	50.3	74.9	75.5	74.2
Maternal educational attainment	N=3013						
No high school diploma	386	20.0	22.1	17.6	11.7	12.4	10.8
High school diploma or GED	527	37.4	35.2	39.9	21.0	19.5	22.7
Some college	891	33.1	34.7	31.4	26.7	28.6	24.5
Bachelor's degree or higher	1209	9.5	8.1	11.1	40.6	39.5	41.9
Median federal poverty level (%)	211 [91, 374]	89 [48, 154]	89 [46, 132]	89 [50, 165]	249 [122, 396]	261 [128, 396]	245 [115, 396]

	Total N (unweighted)	Food insecure ¹			Food secure ¹		
		Overall	Male	Female	Overall	Male	Female
Federal poverty level categories	N=2909						
0-99%	765	56.9	59.2	54.4	20.8	20.0	21.7
100-185%	557	25.6	27.6	23.3	18.1	16.8	19.6
≥186%	1587	17.5	13.2	22.3	61.1	63.2	58.7
Insurance pre-pregnancy	N=2908						
Medicaid	402	22.8	26.9	18.1	12.6	11	14.3
Other	1838	35.3	33.1	37.9	66.5	67.2	65.6
Self-pay	668	41.9	40.1	44.0	20.9	21.7	20
Payment source at delivery	N=3007						
Medicaid	1139	65.8	67.8	63.4	33.2	33	33.5
Private	1772	30.4	28.3	32.6	64.3	64.6	64
Self-pay	40	2.0	1.8	2.2	1.3	1.3	1.4
Other	56	1.9	2.0	1.7	1.1	1.1	1.2
Prenatal WIC participation	N=2934						
Not eligible (FPL>185)	305	14.0	9.6	18.8	10.5	9.4	11.7
Eligible but not (FPL≤185)	1422	12.1	9.4	15.0	55.4	57.1	53.4
Yes	1207	73.9	80.9	66.1	34.0	33.3	34.9
Prenatal care sought	N=2875						
No	5	0.0	0.0	0.0	0.1	0.1	0.1
Yes	2870	100.0	100	100	99.9	99.9	99.9
Pre-pregnancy body mass index	N=2965						
Underweight	103	3.1	2.6	3.7	2.9	2.9	2.9
Normal	1506	40.8	38.6	43.3	51.6	51.4	51.8
Overweight	686	23.1	23.2	23.0	23.2	24	22.4
Obese	670	32.9	35.6	30.0	22.3	21.7	22.9
Smoking before/during pregnancy	N=2975						
Never	2514	61.4	57.9	65.2	85.3	84.7	86.1
Before only	286	21.3	27.1	14.7	9.3	10.4	8.0

	Total N (unweighted)	Food insecure ¹			Food secure ¹		
		Overall	Male	Female	Overall	Male	Female
Before and/or during Prenatal depression	175 N=2967	17.3	14.9	20.1	5.4	4.9	5.8
No	2718	81.6	82.4	80.7	93.5	93.8	93.1
Yes	249	18.4	17.5	19.3	6.5	6.2	6.9
Gestational diabetes	N=2978						
No	2633	86.8	87.5	86.1	90.6	91.1	90.1
Yes	345	13.2	12.5	13.9	9.4	8.9	9.9
Route of Delivery	N=2968						
Spontaneous vaginal	1996	65.3	65.7	65.0	69.8	67	73.1
Assisted vaginal	124	3.4	3.0	3.8	4.0	4.6	3.2
Cesarean section	848	31.2	31.3	31.2	26.2	28.4	23.6
Maternal food security status	N=3022						
Food secure	2563	-	-	-	-	-	-
Prenatal food insecurity only	149	32.4	30.5	34.5	-	-	-
Postpartum food insecurity	193	42.1	41.2	43.1	-	-	-
Persistent food insecurity	117	25.5	28.2	22.4	-	-	-
Infant characteristics at birth							
Previous livebirths	N=3019						
None	1405	43.9	44.7	43	44.4	44.9	43.9
1	957	28.0	28.8	27.0	34.2	32.3	36.4
2	411	17.0	16.1	18.1	12.9	13	12.9
≥3	246	11.1	10.4	11.9	8.4	9.8	6.8
Gestational week at birth	N=3022						
<37 weeks (preterm)	178	8.6	13.6	2.8	5.2	5.6	4.7
≥37 (term)	2844	91.4	86.4	97.2	94.8	94.4	95.2
Sex	N=3022						
Male	1583	53.2	-	-	53.7	-	-
Female	1439	46.8	-	-	46.3	-	-

	Total N (unweighted)	Food insecure ¹			Food secure ¹		
		Overall	Male	Female	Overall	Male	Female
Low birthweight	N=3022						
No	2890	94.7	93.0	96.5	96.2	96.2	96.2
Yes	132	5.3	7.0	3.5	3.8	3.8	3.8
Admission to NICU or infant transferred	N=2994						
No	2759	89.7	83.8	96.4	92.9	92.2	93.7
Yes	235	10.3	16.2	3.6	7.1	7.8	6.3
Ever breastfed infant	N=2985						
No	96	3.6	2.0	5.4	3.6	4	2.9
Yes	2889	96.4	98.0	94.6	96.4	95.8	97.1
Toddler characteristics at 2 years of age							
Mean age of toddler (months)	25.0±0.9	25.0±0.9	25.2±1.0	25.0±0.8	25.0±0.9	25.0±0.9	25.0±0.9
Obesity status	N=3022						
<95th percentile	2592	81.4	78.9	84.2	86.9	87.1	86.6
≥95th percentile	430	18.6	21.1	15.8	13.1	12.9	13.4

*2012-13 excluded from analysis

1 – weighted prevalence

2 – includes Asian/Pacific Islander, American Indian or Alaska Native, Other, and Hispanic origin unknown or not stated.

Table 6.2. Prevalence of obesity at 2 years of age by maternal characteristics and exposure: Oregon PRAMS data, 2008-2015*

Exposure	Infant obesity @ 2 years of age							
	Overall				Male			
	n	Percent ¹	p-value	n	Percent ¹	p-value	n	Percent ¹ p-value
Maternal demographics								
Any food insecurity								
No	341	13.1	0.034	189	12.9	0.023	152	13.4 0.511
Yes	89	18.6		49	21.1		40	15.8
Maternal age at birth (years)								
Under 20	17	14.2		9	14.3		8	13.2
20-24	94	17.0		50	17.9		44	16.2
25-29	121	13.9	0.581	69	14.3	0.502	52	13.4 0.401
30-34	125	13.6		65	12.7		60	14.6
35-39	57	11.3		36	14.7		21	7.7
40+	16	13.0		9	6.4		7	22.5
Maternal race/ethnicity								
non-Hispanic white	116	12.9		68	13.3		48	12.4
non-Hispanic black	41	21.6	0.002	16	15.9	0.314	25	26.8 <0.001
Hispanic	111	18.6		56	16.8		55	20.9
Other ²	161	12.7		97	16.1		64	9.1
Marital status								
Not married	166	19.1	<0.001	87	18.8	0.018	79	19.5 0.007
Married	262	11.8		150	12.2		112	11.3
Maternal educational attainment								
No high school diploma	75	21.5	0.003	37	19.5	0.337	38	24.2 <0.001

Infant obesity @ 2 years of age								
	Overall			Male			Female	
	n	Percent ¹	p-value	n	Percent ¹	p-value	n	Percent ¹ p-value
High school diploma or GED	95	14.2		48	12.0		47	16.5
Some college	140	14.9		80	14.6		60	15.4
Bachelor's degree or higher	118	10.6		72	13.4		46	7.5
Federal poverty level								
0-99%	153	18.3		76	16.7		77	20.1
100-185%	86	13.8	0.010	53	16.7	0.144	33	10.7
≥186%	175	11.6		99	11.7		76	11.6
Prenatal WIC participation								
Not eligible (FPL>185)	153	11.5		91	12.2		62	10.7
Eligible but not (FPL≤185)	45	13.3	0.028	24	11.1	0.173	21	15.1
Yes	222	16.7		116	16.4		106	17.2
Prenatal care sought								
No	0	0.0	0.487	0	0.0	0.566	0	0.0
Yes	407	14.0		229	14.3		178	13.8
Pre-pregnancy body mass index								
Underweight	6	13.5		1	9.0		5	18.4
Normal	180	11.1	0.033	103	11.3	0.155	77	10.9
Overweight	104	16.8		58	17.4		46	16.2
Obese	132	17.5		72	18.1		60	16.7
Smoking before/during pregnancy								
Never	325	12.5		188	13.3		137	11.6
Before only	61	17.8	0.005	31	17.1	0.526	30	19.1
Before and/or during	39	24.7		17	18.0		22	30.9
Prenatal depression								
No	377	13.4	0.030	208	13.8	0.466	169	12.9
Yes	46	20.9		24	17.0		22	24.9

*2012-13 excluded from analysis

1 – weighted prevalence

2 – includes Asian/Pacific Islander, American Indian or Alaska Native, Other, and Hispanic origin unknown or not stated.

Table 6.3. Unadjusted and adjusted OR of any food insecurity and obesity at 2 years of age overall and by infant sex: Oregon PRAMS data, 2008-2015*

	All births			Term births (37+ weeks)		
	Overall OR (95% CI)	Male OR (95% CI)	Female OR (95% CI)	Overall OR (95% CI)	Male OR (95% CI)	Female OR (95% CI)
Obesity @ 2 years						
Unadjusted	1.51 (1.03, 2.22)	1.80 (1.08, 3.00)	1.22 (0.68, 2.17)	1.55 (1.04, 2.31)	2.06 (1.21, 3.53)	1.09 (0.61, 1.97)
Confounder-adjusted models						
Model 1a	1.21 (0.81, 1.81)	1.58 (0.92, 2.70)	0.90 (0.50, 1.64)	1.29 (0.85, 1.94)	1.82 (1.04, 3.21)	0.84 (0.45, 1.54)
Model 1b	1.22 (0.82, 1.83)	1.58 (0.92, 2.71)	0.91 (0.49, 1.71)	1.31 (0.87, 1.98)	1.84 (1.05, 3.23)	0.85 (0.45, 1.61)
Model 1c	1.15 (0.75, 1.75)	1.54 (0.86, 2.74)	0.83 (0.44, 1.57)	1.26 (0.82, 1.95)	1.80 (0.99, 3.28)	0.81 (0.42, 1.55)
Models adjusted for potential mediators						
Model 2	1.10 (0.71, 1.70)	1.53 (0.85, 2.77)	0.75 (0.39, 1.44)	1.20 (0.76, 1.87)	1.80 (0.97, 3.33)	0.71 (0.36, 1.41)
Model 3	1.00 (0.65, 1.56)	1.43 (0.80, 2.56)	0.66 (0.34, 1.29)	1.10 (0.70, 1.73)	1.64 (0.90, 3.03)	0.66 (0.33, 1.32)
Model 4	1.03 (0.66, 1.59)	1.43 (0.79, 2.57)	0.67 (0.34, 1.32)	1.14 (0.72, 1.81)	1.71 (0.91, 3.19)	0.69 (0.34, 1.39)

*2012-13 excluded from analysis

1a – adjusted for maternal age, maternal education, maternal race/ethnicity, maternal BMI, marital status, survey year

1b – Model 1a + number of previous live births

1c – Model 1b + federal poverty level

2 – Model 1c + prenatal depression

3 – Model 2 + maternal smoking

4 – Model 3 + WIC use

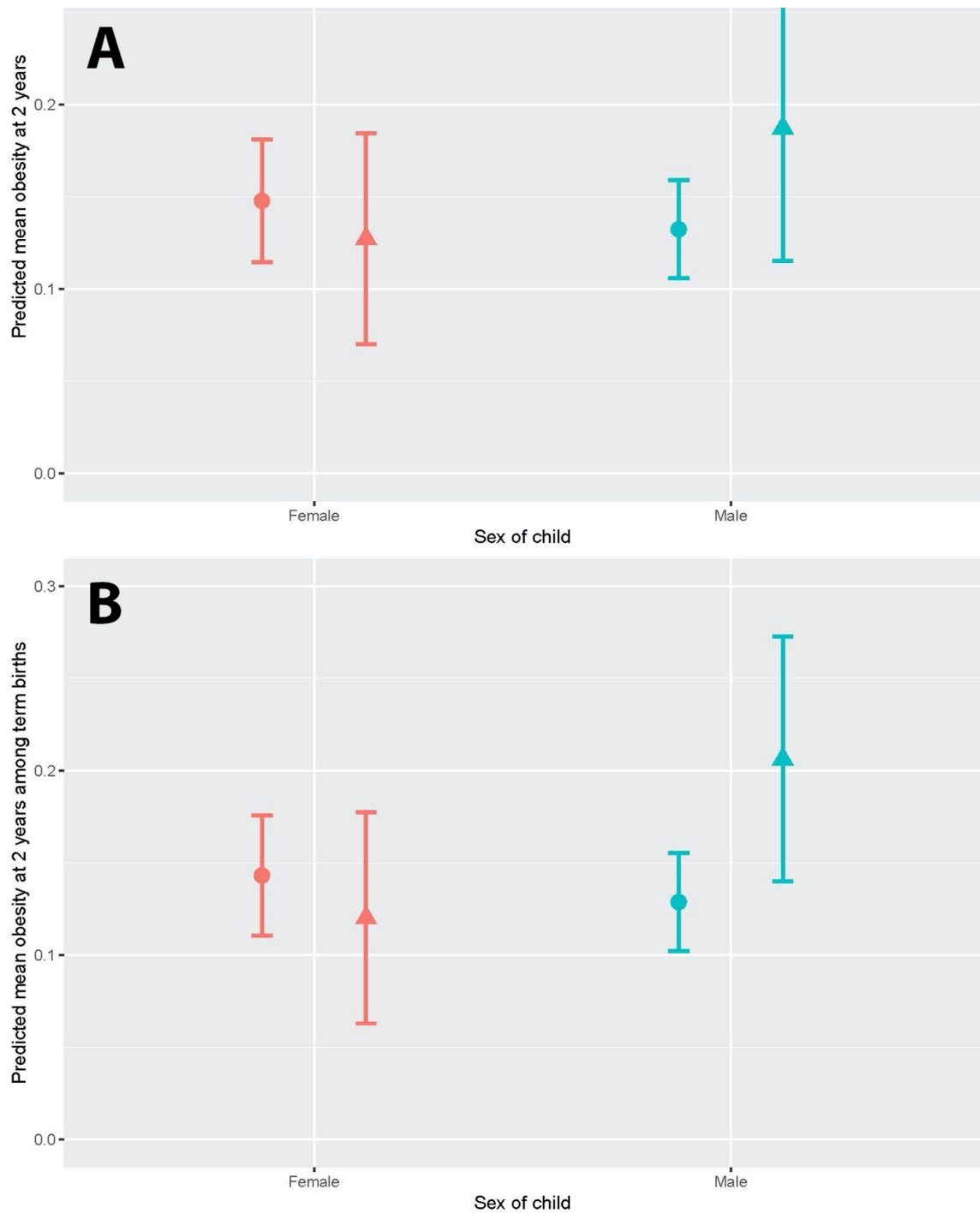


Figure 6.1. Predicted mean obesity at 2 years among A) all births and B) term births. Circles indicate food secure mothers, and triangles indicate food insecure mothers.

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SUPPLEMENTAL APPENDIX

Supplementary Table 6.1. Unadjusted and adjusted OR of prenatal food insecurity and obesity at 2 years of age overall and by infant sex:

	All births			Term births		
	Overall OR (95% CI)	Male OR (95% CI)	Female OR (95% CI)	Overall OR (95% CI)	Male OR (95% CI)	Female OR (95% CI)
Unadjusted						
Prenatal FI	1.36 (0.73, 2.55)	1.72 (0.71, 4.18)	1.04 (0.45, 2.38)	1.41 (0.74, 2.67)	1.83 (0.73, 4.54)	1.05 (0.45, 2.43)
Postpartum FI	1.88 (1.10, 3.19)	2.34 (1.17, 4.70)	1.43 (0.62, 3.30)	2.14 (1.24, 3.72)	3.07 (1.46, 6.46)	1.48 (0.64, 3.44)
Persistent FI	1.14 (0.51, 2.54)	1.18 (0.42, 3.35)	1.09 (0.31, 3.79)	0.91 (0.38, 2.15)	1.28 (0.43, 3.82)	0.45 (0.16, 1.25)
Confounder-adjusted models						
Model 1a						
Prenatal FI	1.15 (0.60, 2.18)	1.49 (0.63, 3.51)	0.79 (0.31, 2.02)	1.24 (0.64, 2.38)	1.60 (0.66, 3.86)	0.84 (0.33, 2.14)
Postpartum FI	1.53 (0.89, 2.62)	2.10 (1.01, 4.38)	1.13 (0.48, 2.68)	1.77 (1.01, 3.09)	2.64 (1.18, 5.91)	1.23 (0.52, 2.91)
Persistent FI	0.82 (0.36, 1.84)	0.98 (0.34, 2.75)	0.69 (0.20, 2.38)	0.69 (0.28, 1.67)	1.16 (0.38, 3.51)	0.28 (0.09, 0.84)
Model 1b						
Prenatal FI	1.16 (0.61, 2.21)	1.47 (0.62, 3.46)	0.82 (0.31, 2.13)	1.25 (0.65, 2.41)	1.56 (0.65, 3.77)	0.86 (0.33, 2.25)
Postpartum FI	1.53 (0.89, 2.61)	2.09 (1.00, 4.35)	1.12 (0.48, 2.61)	1.78 (1.02, 3.10)	2.64 (1.19, 5.88)	1.21 (0.52, 2.82)
Persistent FI	0.84 (0.37, 1.90)	1.02 (0.36, 2.87)	0.70 (0.20, 2.44)	0.72 (0.29, 1.74)	1.24 (0.41, 3.74)	0.28 (0.09, 0.92)
Model 1c						
Prenatal FI	1.10 (0.57, 2.15)	1.47 (0.62, 3.50)	0.74 (0.27, 2.01)	1.22 (0.62, 2.40)	1.55 (0.63, 3.80)	0.81 (0.30, 2.20)
Postpartum FI	1.43 (0.82, 2.49)	2.02 (0.91, 4.47)	1.02 (0.44, 2.40)	1.70 (0.96, 3.02)	2.58 (1.09, 6.10)	1.14 (0.48, 2.68)
Persistent FI	0.79 (0.35, 1.77)	0.99 (0.35, 2.80)	0.62 (0.18, 2.18)	0.69 (0.28, 1.69)	1.21 (0.39, 3.68)	0.27 (0.08, 0.90)
Models adjusted for potential mediators						
Model 2						
Prenatal FI	1.02 (0.50, 2.07)	1.40 (0.55, 3.55)	0.67 (0.26, 1.73)	1.11 (0.54, 2.29)	1.46 (0.55, 3.87)	0.70 (0.27, 1.84)
Postpartum FI	1.38 (0.79, 2.44)	2.03 (0.91, 4.51)	0.93 (0.38, 2.28)	1.64 (0.91, 2.94)	2.59 (1.09, 6.15)	1.01 (0.41, 2.52)
Persistent FI	0.77 (0.34, 1.73)	1.01 (0.36, 2.86)	0.56 (0.15, 2.02)	0.67 (0.27, 1.62)	1.24 (0.41, 3.75)	0.24 (0.07, 0.82)
Model 3						
Prenatal FI	1.00 (0.50, 2.00)	1.37 (0.54, 3.46)	0.61 (0.24, 1.57)	1.11 (0.55, 2.54)	1.44 (0.55, 3.76)	0.67 (0.26, 1.72)

	All births			Term births		
	Overall OR (95% CI)	Male OR (95% CI)	Female OR (95% CI)	Overall OR (95% CI)	Male OR (95% CI)	Female OR (95% CI)
Postpartum FI	1.34 (0.76, 2.39)	2.00 (0.91, 4.36)	0.87 (0.33, 2.32)	1.62 (0.89, 2.94)	2.61 (1.11, 6.11)	0.98 (0.38, 2.33)
Persistent FI	0.53 (0.23, 1.18)	0.65 (0.22, 1.93)	0.44 (0.13, 1.53)	0.40 (0.18, 0.93)	0.73 (0.23, 2.25)	0.21 (0.06, 0.77)
Model 4						
Prenatal FI	1.01 (0.50, 2.03)	1.37 (0.54, 3.47)	0.60 (0.24, 1.54)	1.13 (0.56, 2.30)	1.46 (0.56, 3.84)	0.66 (0.26, 1.68)
Postpartum FI	1.37 (0.77, 2.44)	2.00 (0.91, 4.39)	0.86 (0.31, 2.35)	1.66 (0.91, 3.00)	2.67 (1.13, 6.30)	0.96 (0.36, 2.54)
Persistent FI	0.55 (0.24, 1.23)	0.65 (0.22, 1.94)	0.49 (0.14, 1.67)	0.42 (0.18, 0.97)	0.74 (0.24, 2.29)	0.23 (0.06, 0.84)

*2012-13 excluded from analysis

1a – adjusted for maternal age, maternal education, maternal race/ethnicity, maternal BMI, marital status, survey year

1b – Model 1a + number of previous live births

1c – Model 1b + federal poverty level

2 – Model 1c + prenatal depression

3 – Model 2 + maternal smoking

4 – Model 3 + WIC use

Chapter 7: Concluding Remarks

7.1. *Summary of Major Findings*

In summary, findings suggest that maternal food security is prevalent and may perhaps be an important factor that is associated with health outcomes across pregnancy and the early childhood years through both biologic and behavioral pathways, and that some outcomes may differ by sex. Although findings were not statistically significant, it should be noted that interpretations of the findings should not depend on statistical significance, but rather commenting on trends and magnitudes may also be informative.¹⁷⁵ As such, this investigation highlights the patterns seen from all three analyses. In **Aim 1**, we found that mothers experiencing prenatal food insecurity had an increased odds of delivering a low birthweight infant, though much of the association was explained by sociodemographic characteristics. However, from a biologic pathway, findings appeared to support a sex-specific association, with female infants born to food insecure mothers having a higher odds of low birthweight or term low birthweight, which differed from the original hypothesis.

In **Aim 2**, prenatal food insecurity appeared to be associated with decreased breastfeeding initiation, but adjustment for sociodemographic covariates revealed that food insecurity did not deter mothers from initiating breastfeeding. Additionally, food insecurity minimally affected exclusive breastfeeding duration. Magnitudes also suggested that mothers experiencing more prolonged food insecurity may have a slightly longer exclusive breastfeeding duration. The relationship between maternal food insecurity and breastfeeding is likely through a behavioral pathway (Figure 1.1), as it

appears to be complicated by covariates that influence breastfeeding behaviors, such as maternal education and marital status.

Finally, findings in **Aim 3** suggested that sociodemographic characteristics explain most of the association between maternal food insecurity status and childhood obesity at 2 years; however, from a biologic pathway, the magnitudes seemed to suggest a stronger effect among male offspring born to food insecure women in Oregon. Additional sensitivity analyses found that maternal postpartum food insecurity was a stronger predictor of obesity at 2 years. Similar to Aim 1, restriction to term births strengthened the effects among male offspring.

7.2. *Strengths & Limitations*

Limitations have been described for each separate study in Chapters 4-6; however, limitations that are common to the overall investigations are important to note. For example, the largest limitation in this investigation was the imprecise measurement of maternal food insecurity. Because only a single-item question (adapted from a question on USDA's 6-item measure) was used in PRAMS and PRAMS-2, it does not capture other dimensions of food insecurity such as affordability and sufficiency of healthy foods (Figure 2.1), which are equally important, and may also affect inferences made. Moreover, because the assessment of food insecurity is an ever measure using a reference period of the previous twelve months (as is also the case with other precise measures of food insecurity), it may not speak to duration of food insecurity. As such, the true severity of food insecurity may be underreported. Furthermore, because the food insecure population is more likely to be mobile and therefore be difficult to contact,⁶² the enormity of the food insecure population may be missed within this investigation.

Despite this, PRAMS is one of the few datasets that includes a question on the mother's individual experience of food insecurity during pregnancy and postpartum in select states, and similar studies have used this same question to explore maternal food insecurity.^{57,60–62} Moreover, because this question is used in the official abbreviated 6-item measure by the USDA, it is still considered to be a food insecurity indicator, and answering positively to one question indicates that one may be marginally food secure (Table 2.1). Prior research has shown that even marginally food secure people have outcomes that are similar to the food insecure population.⁵⁹

For Aims 2 & 3, generalizability is lower due to the use of state-level data from Oregon. However, their data were unique because of their longitudinal component to PRAMS. Nonetheless, inferences from these Aims can only be made about Oregon women. Additionally, because Oregon has a different population make-up (predominantly non-Hispanic white) than the rest of the U.S. population, population-level inferences cannot be made about pregnant women nationally. As such, these investigations may not contribute to understanding the racial and ethnic disparities that exist with food insecurity and birth outcomes. It should also be noted that PRAMS-2 included women who chose to be followed-up. While PRAMS-2 was re-weighted to account for those women lost to follow-up, and thus is generalizable to the original surveyed population,¹⁷⁶ a comparison of the two samples shows that women who did not respond to PRAMS-2 were likely to be younger, Hispanic, not married, lower education, below a FPL of 185%, WIC participants, and smokers during pregnancy (Table 7.1). While it is possible it may underestimate the true effect of food insecurity and affect generalizability, these factors were adjusted for in models, and conditioned on these

factors, the outcome would no longer be associated with the lost to follow-up.¹⁷⁷

Nonetheless, this investigation was able to provide some insight on how maternal food insecurity may affect early life course health and how additional research with accurate measures are needed.

This investigation also has some strengths. First, this is one of the first investigations exploring the mother's individual experience of food insecurity on birth and early childhood outcomes particularly in a high-income setting, using either cross-sectional or longitudinal approaches. For example, Aim 1 uses multi-state PRAMS data to explore the individual experience of prenatal food insecurity on low birthweight, and therefore has a large sample size and improved generalizability to the women of these states. The investigations presented in Aims 2 & 3 are one of the few studies that have looked at individual maternal food insecurity on breastfeeding initiation, exclusive breastfeeding duration, and early childhood obesity in the U.S. context. Additionally, Aims 2 & 3 leveraged the longitudinal aspect of Oregon PRAMS and their follow-up PPRAMS-2, which establishes temporality of the exposure and outcome.

7.3. Public Health Implications & Future Directions

Food insecurity is a public health concern, and it seems apparent from this investigation that food insecurity is prevalent primarily among low-income mothers. These findings underscore the importance of exploring all aspects of food insecurity and add to the limited understanding of how food insecurity may potentially affect birth and early childhood outcomes of public health concern, with specific focus on maternal experiences of food insecurity.^{43,178} Few studies have explored how the timing of food insecurity in relation to pregnancy affects birth outcomes, such as birthweight, and early

childhood outcomes, such as obesity. As there is a lack of quantitative studies with a longitudinal approach, this investigation sought to fill that gap by taking into consideration both prenatal and postpartum food insecurity experienced by the mother.

Furthermore, this investigation adds to the growing evidence that there appears to be sex differences in response to exposures during critical periods of development.^{38,39} For example, in Aim 1, results were suggestive of a stronger association of being born with low birthweight among females, while magnitudes suggested males were likely to develop obesity at 2 years in Aim 3. Previous research suggests that females may be better suited to in utero environmental stressors, at the expense of their birthweight, while males require more energy to sustain their growth.³⁹ However, it appears that males may be predisposed to adiposity by age 2. Although the exact mechanisms on how sexually-dimorphic responses occur are continuing to be elucidated in animal models, these suggestive findings indicate that future well-powered human research related to in utero exposures, such as food insecurity, should continue to stratify findings by sex to better ascertain how sex differences to the in utero environment affect health outcomes. These studies may help generate future research to develop targeted interventions if needed. While sex-specific interventions may not necessarily be feasible in pregnancy, interventions that benefit both sexes, such as exercise during pregnancy, may be helpful in preventing poor growth and obesity phenotypes, and has been shown to improve glucose and insulin tolerance of both sexes.^{179,180}

This investigation also acknowledges data gaps that still exist. Because of the imprecise measure of food insecurity in PRAMS, this investigation highlights the need for additional, and importantly longitudinal, nationally-representative datasets that

incorporate the full USDA's six-item questionnaire to accurately capture the concept of food insecurity, or even 2-item question that has been recently shown to have good validity.^{55,56} Additionally capturing the experience of food insecurity as it relates to the prenatal and postpartum period is needed.

Coordination of efforts through the USDA Economic Research Service, which is largely responsible for monitoring and measuring food insecurity, is recommended to identify new longitudinal datasets or facilitate linkages with existing datasets. As the USDA continues to collaborate with community partners to refine the food insecurity measure, the USDA should also consider establishing programmatic initiatives to facilitate data linkages, such as linking WIC and SNAP data to birth records.

Coordination with the CDC to improve the food insecurity measure in PRAMS and/or support linking with administrative data, may also be useful particularly in states that do not currently examine food insecurity in their PRAMS survey. Such efforts could help to explore potential risk factors and intervention strategies that could be used at a national-level. Additionally, while this investigation was not able to comment on racial and ethnic disparities, it highlights the need to also have data that explores how food insecurity may contribute to disparities in birth and child outcomes. Mechanisms that may mediate such associations may also require further study either through animal models or longitudinal studies with larger sample sizes, including placental programming, maternal weight gain, smoking, or infant food insecurity.

Finally, exploring how existing safety-net programs, such as SNAP, and more specifically WIC, given the target population, can be optimized to improve food insecurity and diet quality of mothers will be important for future research work. The

WIC population presents an important population to study the effects of food insecurity. Unfortunately, due to sample size issues when restricting to the WIC-eligible population (i.e., $\leq 185\%$ of FPL) particularly in Aims 2 & 3, this investigation was unable to properly explore the effects of WIC. A preliminary analysis exploring this among WIC-eligible NH-White and NH-Black mothers in Aim 1 appeared to show some suggestion of attenuation of low birthweight among WIC participants who were food insecure, though non-significant (Table 7.2).

Although this investigation was interested in the sex-specific response, this investigation also highlights the need for improved measurement of food insecurity to ascertain whether WIC may be beneficial and improve disparities. As such, leveraging the WIC agency to capture precise data on food insecurity is recommended to explore whether food insecure women of all races/ethnicities are being supported, and to find alternative intervention strategies. For example, in addition to ensuring healthy options are available in the WIC program, making use of the referral services for those mothers undergoing stress due to food insecurity is recommended. In terms of breastfeeding, exploring how the breastfeeding education component can be improved or tailored for this population may be warranted, and examining policies such as breastfeeding in the workplace or family leave may be essential for this population. Furthermore, given WIC's finite eligibility, helping this population transition to other benefits, such as SNAP, may be important in optimizing nutrition post-pregnancy and perhaps as important, between pregnancies, to ensure optimal infant health. Of particular relevance, the current COVID-19 pandemic has undoubtedly left many families to lose supports and increase food insecurity, and yet the poor reach of recent pandemic-related supports

exposes the unpreparedness of handling food insecurity and hunger. To protect vulnerable populations including mothers and children, there is a clear need for additional nutrition safety nets at the federal level.

7.4. Conclusion

In conclusion, in the context of the life course framework, this preliminary investigation highlights the importance of continuing to explore exposures that occur during critical periods, such as maternal food insecurity, on later health outcomes. For outcomes relating to growth, exploring sex differences is also important to further understand how male and female infants respond to in utero exposures and growth over their early life course. This investigation sought to illuminate the links between food insecurity and birth and childhood outcomes, and these results should help to spur additional research to advance the current understanding of maternal food insecurity.

APPENDIX A: TABLES

Table 2.1. Definitions of the levels of food security as used by the USDA.

Ranges	Definition
<i>Food Security</i>	
High food security	No reported indications of food-access problems or limitations
Marginal food security	One or two reported indications—typically of anxiety over food sufficiency or shortage of food in the house. Little or no indication of changes in diets or food intake.
<i>Food insecurity</i>	
Low food security	Reports of reduced quality, variety, or desirability of diet. Little no indication of reduced food intake (previously ‘food insecurity without hunger’)
Very low food security	Reports of multiple indications of disrupted eating patterns and reduced food intake (previously ‘food insecurity with hunger’)

Table 7.1. Weighted percent distribution of selected maternal and infant characteristics of women that participated in PRAMS-2 vs those that did not participate in PRAMS-2 overall: Oregon PRAMS data, 2008-2015*

	Overall	Followed to PRAMS-2	Lost to follow-up
Maternal characteristics at time of birth			
Maternal age at birth (years)	N=9,008	N=3,817	N=5,191
< 20	6.2	4.0	9.2
20-24	20.4	19.1	24.8
25-29	30.1	32.2	29.7
30-34	26.9	27.0	23.3
35-39	13.6	14.2	11.0
≥40	2.8	3.4	1.9
Maternal race/ethnicity	N=9,698	N=3,805	N=5,893
non-Hispanic white	68.1	68.3	62.6
non-Hispanic black	2.0	2.0	2.7
Hispanic	19.6	19.5	23.5
Other ²	10.3	10.2	11.3
Marital status	N=9,734	N=3,813	N=5,921
Not married	35.1	31.0	43.6
Married	64.9	69.0	56.4
Maternal educational attainment	N=9,703	N=3,804	N=5,899
No high school diploma	17.0	14.8	22.2
High school diploma	23.7	24.1	28.0
Some college	31.2	27.2	32.6
Bachelor's degree or higher	28.1	33.8	17.2
Federal poverty level categories	N=8,971	N=3,636	N=5,335
0-99%	33.8	28.6	43.4
100-185%	20.4	20.1	22.2
≥186%	45.8	51.2	34.4
Payment source at delivery	N=8,107	N=2,932	N=5,175
Medicaid	45.4	40.9	54.7
Private	51.4	56.3	41.7
Self-pay	1.6	1.4	1.7
Other	1.6	1.4	1.8
Prenatal WIC participation	N=44,067	N=3,292	N=5,618
Not eligible (FPL>185)	39.5	45.1	27.9
Eligible but no use (FPL≤185)	12.8	11.1	14.5
Yes	47.7	43.8	57.6
Prenatal care sought	N=9,173	N=3,622	N=5,551

No	0.3	0.3	0.4
Yes	99.7	99.7	99.6
Pre-pregnancy body mass index	N=8,823	N=3,734	N=5,089
Underweight	3.0	2.8	3.2
Normal	48.0	48.9	45.5
Overweight	24.6	24.2	25.0
Obese	24.4	24.1	26.3
Smoking before/during pregnancy	N=9,514	N=3,746	N=5,768
Never	77.8	81.5	72.7
Before only	11.7	10.8	13.1
Before and/or during	10.7	7.6	14.2
Prenatal depression	N=9,424	N=3,728	N=5,696
No	90.2	91.1	89.0
Yes	9.8	8.9	11.0
Prenatal food insecurity	N=9,532	N=3,762	N=5,077
No	90.5	90.1	89.3
Yes	9.5	9.9	10.6
Route of Delivery	N=9,573	N=3,751	N=5,823
Spontaneous vaginal	68.5	68.3	69.0
Assisted vaginal	3.8	3.4	3.8
Cesarean section	27.8	28.3	27.2
Infant characteristics at birth			
Previous livebirths	N=9,005	N=3,814	N=5,191
None	41.0	41.6	38.7
1	32.8	33.5	32.7
2	14.9	14.5	15.9
≥3	11.2	10.4	12.7
Gestational week at birth	N=9,007	N=3,817	N=5,190
<37 weeks (preterm)	6.6	7.1	6.4
≥37 weeks	93.4	92.9	93.6
Sex	N=9,742	N=3,817	N=5,925
Male	51.0	54.0	48.8
Female	49.0	46.0	51.2
Low birthweight (<2500g)	N=9,501	N=3,817	N=5,191
No	94.9	94.9	94.8
Yes	5.1	5.1	5.2
Admission to NICU	N=9,501	N=3,763	N=5,738
No	91.8	92.0	91.7
Yes	8.2	8.0	8.3

Ever breastfed	N=9,426	N=3,751	N=5,675
No	5.0	4.0	6.1
Yes	95.0	96.0	93.9

*2012-2013 were excluded from analysis

1 – weighted proportion

2 – includes Asian/Pacific Islander, American Indian or Alaska Native, Other, and Hispanic origin unknown or not stated

Table 7.2. Exploration of prenatal food insecurity on low birthweight with WIC use as an interaction among non-Hispanic white and non-Hispanic black women: PRAMS data 2009-2017.

	Unadjusted model		
	Overall	NH-White	NH-Black
All births			
Prenatal FI	1.17 (0.90, 1.50)	1.31 (0.98, 1.74)	0.70 (0.39, 1.25)
Prenatal FI x WIC Use	0.91 (0.66, 1.25)	0.77 (0.53, 1.12)	1.88 (0.94, 3.77)
Term births			
Prenatal FI	0.96 (0.64, 1.42)	1.18 (0.76, 1.85)	0.41 (0.16, 1.01)
Prenatal FI x WIC Use	1.24 (0.76, 2.02)	1.04 (0.59, 1.84)	2.91 (1.03, 8.72)
Model 5*			
All births			
Prenatal FI	0.95 (0.71, 1.27)	1.04 (0.76, 1.44)	0.72 (0.39, 1.33)
Prenatal FI x WIC Use	0.98 (0.68, 1.40)	0.80 (0.53, 1.20)	1.76 (0.85, 3.64)
Term births			
Prenatal FI	0.81 (0.52, 1.25)	1.07 (0.66, 1.73)	0.39 (0.14, 1.05)
Prenatal FI x WIC Use	1.25 (0.73, 2.13)	0.95 (0.52, 1.74)	2.93 (0.95, 8.99)

*adjusted for maternal age, maternal education, maternal race/ethnicity, maternal BMI, marital status, survey year, number of previous livebirths, federal poverty level, WIC use, maternal smoking

APPENDIX B: FIGURES

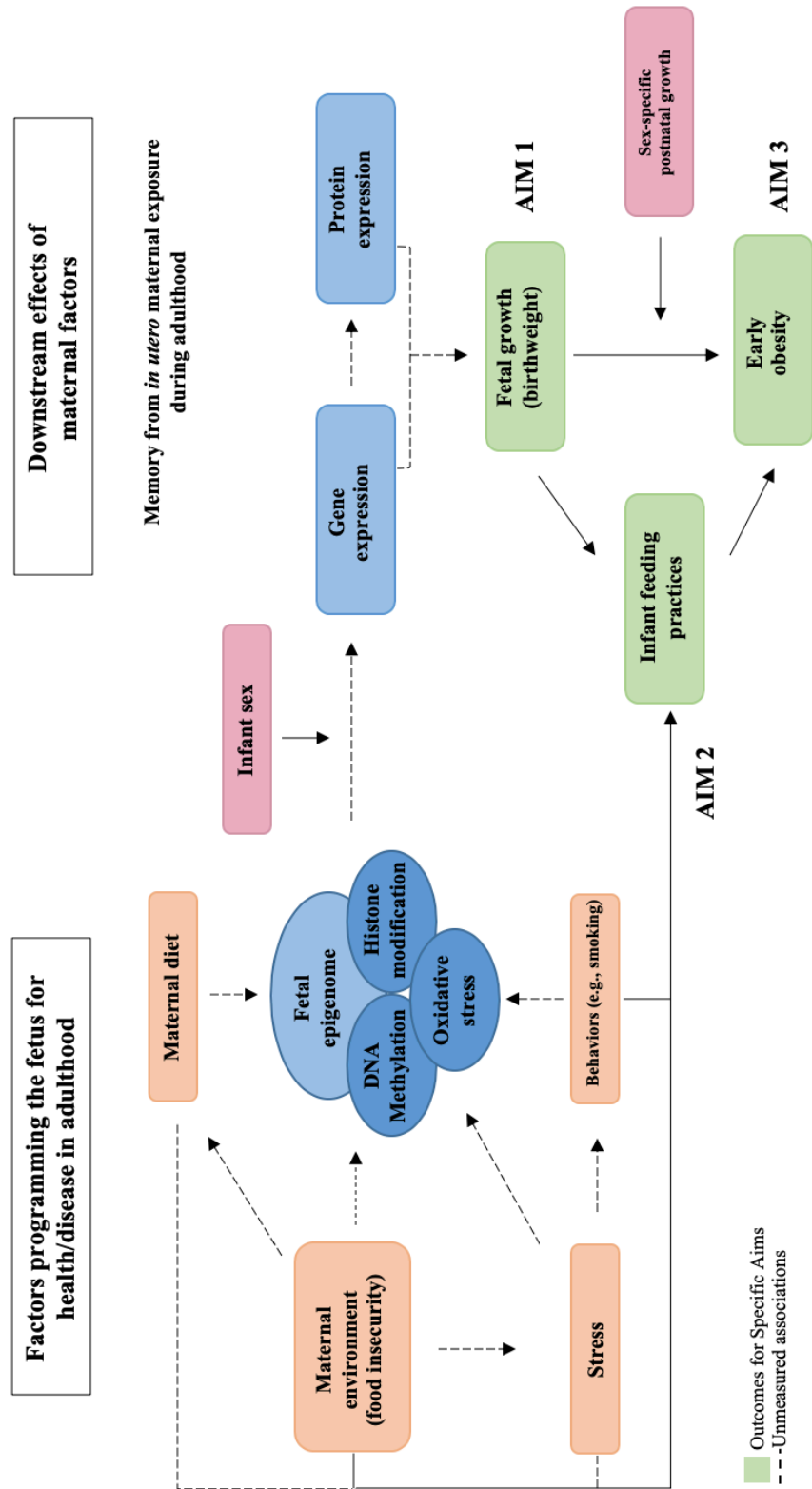


Figure 1.1. Conceptual model of food insecurity and birth, infant, and early childhood outcomes (adapted from Ganu RS, Harris RA, Collins K, Aagaard KM. Maternal diet: a modulator for epigenomic regulation during development in nonhuman primates and humans. *Int J Obes Suppl.* 2012;2(S2):S14-S18, by permission of Springer Nature Publishing⁽³⁰⁾).

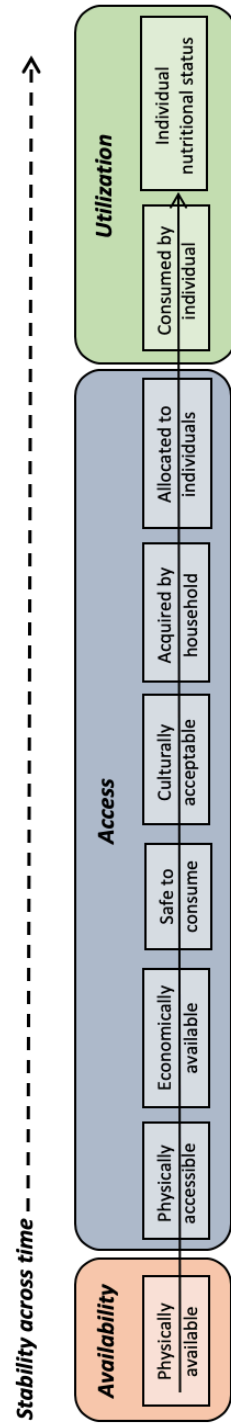


Figure 2.1. Heuristic of dimensions of food security (adapted from Jones AD et al., What are we assessing when we measure food security? A compendium and review of current metrics. *Adv Nutr* 2013;4(5):481-505, by permission of Oxford University Press⁴⁵).

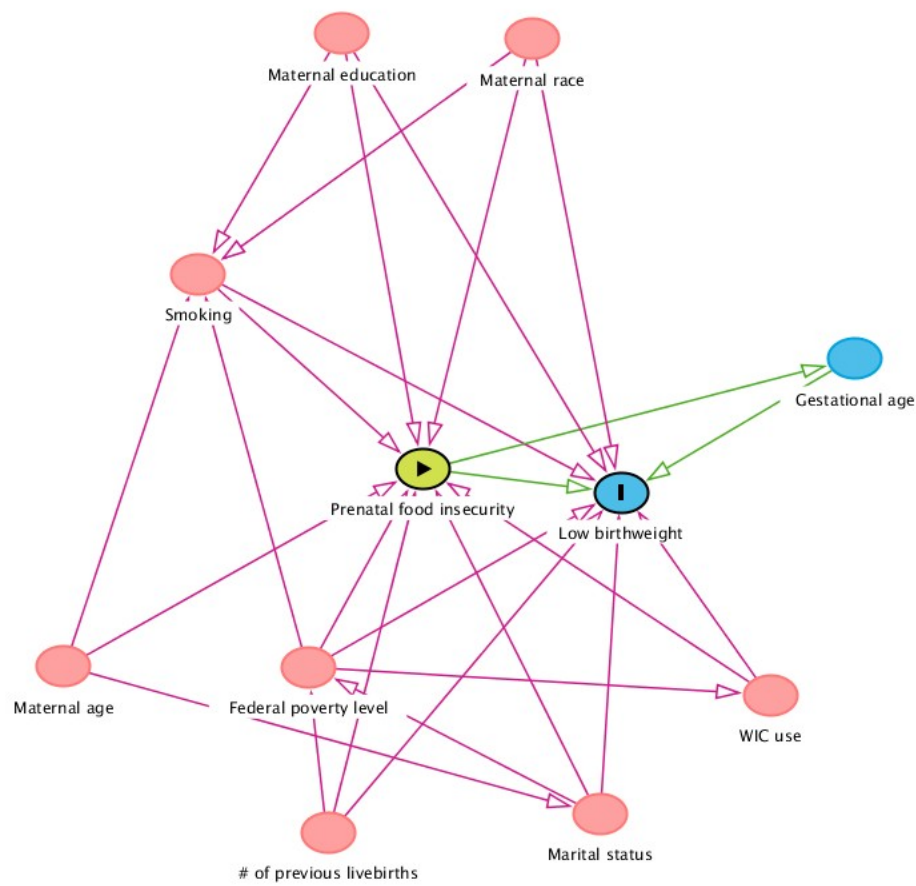


Figure 3.1. Directed acyclic graph (DAG) of confounders conceptualized for Aim 1.

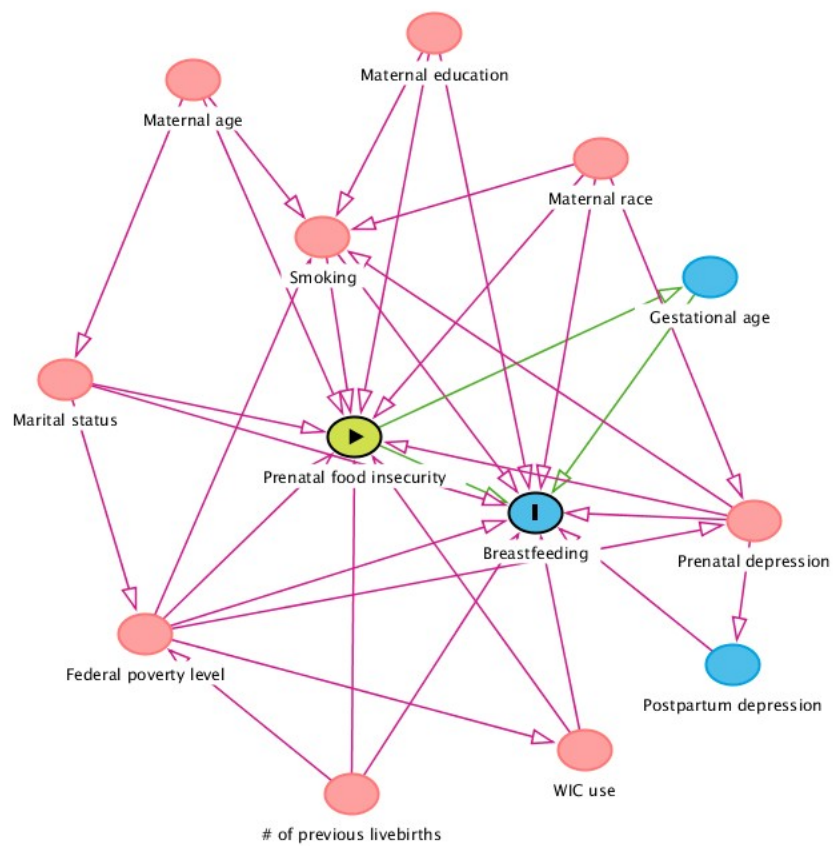


Figure 3.2. Directed acyclic graph (DAG) of confounders conceptualized for Aim 2.

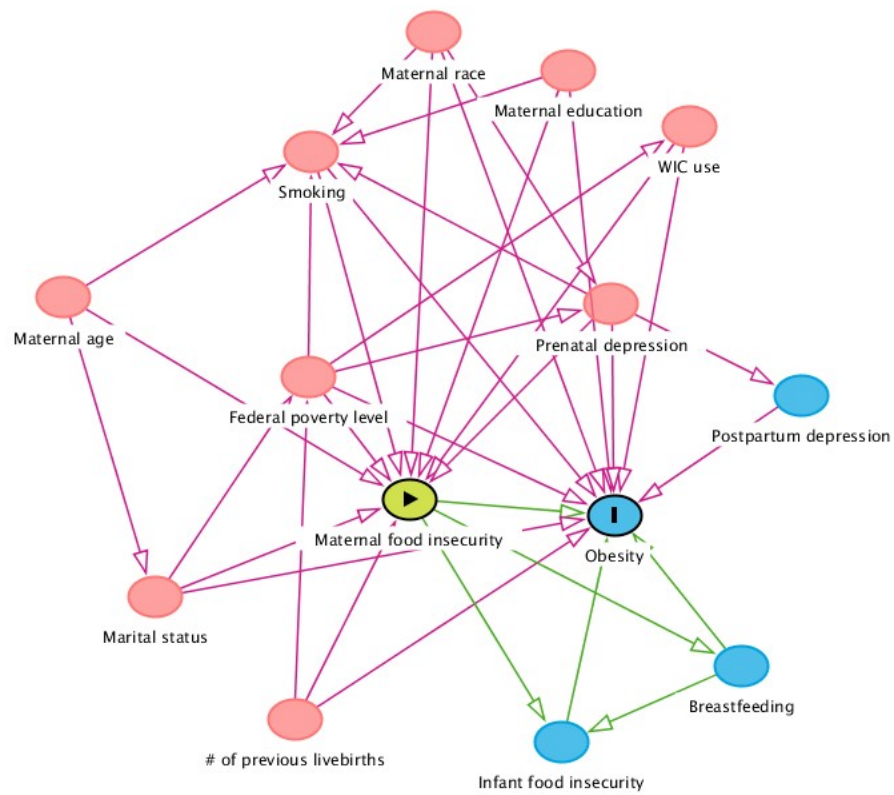
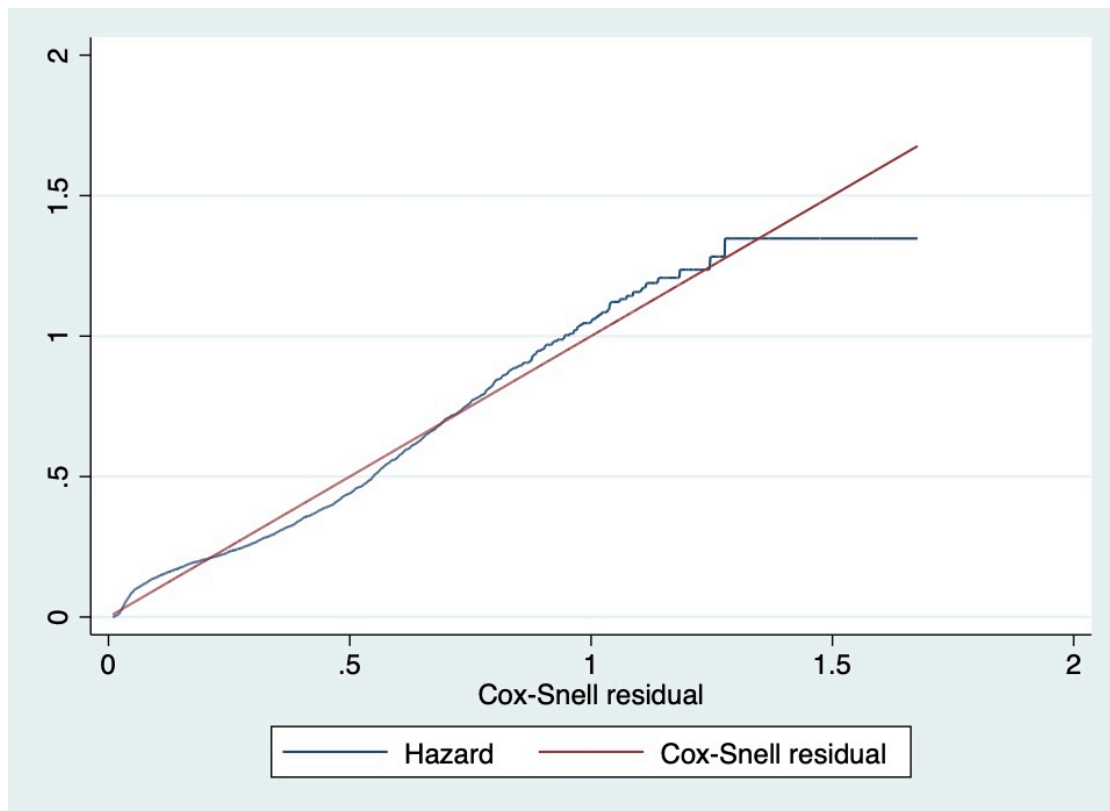
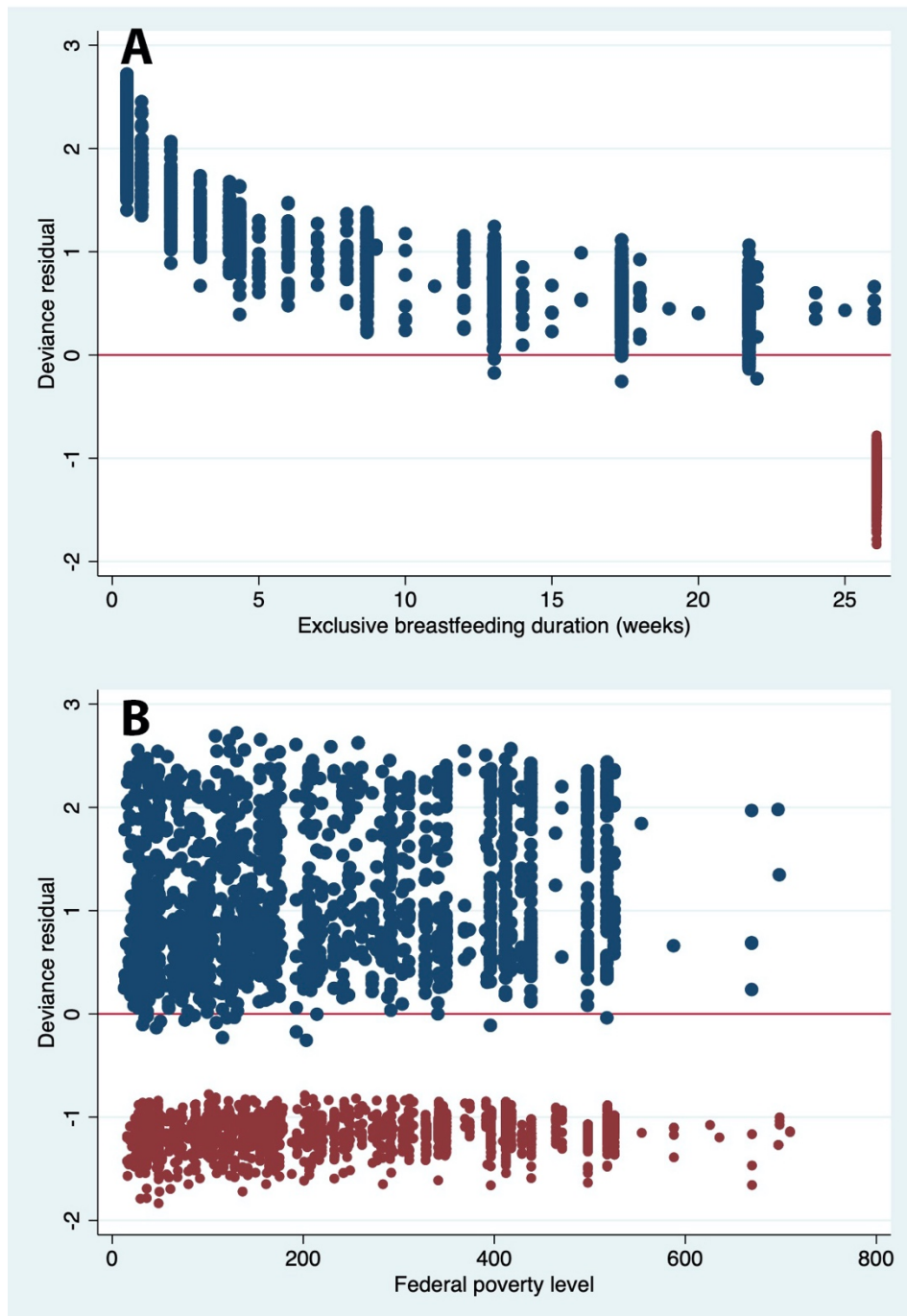


Figure 3.3. Directed acyclic graph (DAG) of confounders conceptualized for Aim 3.



Supplementary Figure 5.2. Plot of the Cox-Snell residual of the parametric accelerated failure time model for exclusive breastfeeding duration.



Supplementary Figure 5.3. Deviance residual of the parametric accelerated failure time model plotted against A) exclusive breastfeeding duration (outcome) and B) federal poverty level (covariate). Red dots indicate observations that were censored at 6 months' duration of breastfeeding.

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