ASSOCIATION BETWEEN EARLY MATERNAL DEPRESSION AND CHILD GROWTH: A GROUP-BASED TRAJECTORY MODELING ANALYSIS

BY

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DISSERTATION

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ABSTRACT

Childhood overweight and obesity have become a primary social and public health concern. Over the past 30 years, rates of childhood overweight and obesity in the United States have increased dramatically from 6% to 32%. Childhood obesity is also a health equity issue: overweight and obesity disproportionately affect more low-income and minority children than White and middle class children. A potential risk factor of interest is maternal depression. To date, there are mixed findings available on the association between maternal depression and childhood obesity development. To address these gaps in the literature, this study used innovative statistical techniques to explore: 1) The association between maternal depression at age 1 and/or age 3 and childhood obesity longitudinally; 2) The association between child-level factors (sleep, television viewing, outdoor play, and dietary intake) and at-risk growth trajectories from birth to age 9; 3) The association between clan-level factors (maternal involvement and breastfeeding duration) and at-risk growth trajectories from birth to age 9; 4) Differences in growth trajectory solutions by race/ethnicity; and 5) Common and unique risk factors that predict at-risk growth among children from different racial/ethnic groups.

This study used data from the Fragile Families Child Well Being Study (FFCWS) to investigate the research questions. FFCWS is a national dataset that has information on 4,898 women from predominantly non-marital, low-income, minority groups in the United States. This study used information collected at the birth of the child (wave 1) through age 9 (wave 5). The analytic sample consisted of 3,500 mother-children dyads. Group-based trajectory modeling, general linear models, and multivariable logistic regression were used to test the different research questions in the full sample and the stratified sample by race/ethnicity.

The results indicated that there was no association between maternal depression and childhood obesity development in this sample of low-income and mostly minority participants. Only maternal pre-pregnancy BMI and number of biological children were significant predictors of at-risk growth trajectories in the full sample. It was also found that there group-trajectory solutions were different based on race/ethnicity. Specifically, a three-group trajectory solution was found for the full sample and for the sample of White children. Among the samples of Black and Latino children, a two-group trajectory solution was found. The most worrisome results were found among Latino children who showed a rapid BMI z-score increase from birth until age 5, which placed them at an increased risk of overweight/obesity during early childhood. Also, child-level and clan-level factors were found to be associated differently with at-risk group membership by race/ethnicity. Suggestions for designing childhood obesity prevention interventions based on research are discussed. Implications for theory, policy, and future research are identified.

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Para mi Familia y en especial para Heladio Leaño Leaño

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CHAPTER 1: INTRODUCTION

Childhood overweight and obesity have become a primary social and public health concern (Lobstein et al., 2015; Wang & Lobstein, 2006). Over the past 30 years, rates of childhood overweight and obesity in the United States (US) have increased dramatically from 6% in 1980 to about 32% in 2014 (Ogden, Carroll, Kit, & Flegal, 2014; Ogden, Yanovski, Carroll, & Flegal, 2007; Wang & Lobstein, 2006). Childhood overweight is defined by the Centers for Disease and Control Prevention (CDC) as children between the ages of 2 and 19 years of age whose body mass index (BMI) is at or over the 85th percentile and childhood obesity is defined as BMI at or over the 95th percentile (Kuczmarski et al., 2002; Ogden et al., 2014). Childhood overweight and obesity are problems that affect children globally; however, their prevalence in the US is among the highest in developed countries. The prevalence of childhood overweight and obesity in the US is 31.8% among children between the ages of 2 and 19 (Ogden et al., 2014). By age group, adolescents between the ages of 12 to 19 years of age have the highest prevalence (34.5%) and those with the lowest are preschool-aged children between the ages of 2 to 5 (22.8%) (Ogden et al., 2014). Over time, regardless of the multiple efforts conducted to reduce childhood obesity, the majority of these rates have not shown signs of decreasing, at least until recently (Ogden et al., 2014).

Despite the steady increase in childhood obesity, recent studies have shown mixed findings on trends among preschoolers (Ogden et al., 2014; Skinner, Perrin & Skelton, 2016). A recent study showed that obesity trends among 2-5 year old children have slightly decreased between 2003-2004 and 2011-2012 (Ogden et al., 2014). Similar results were found by a study using data from 11.6 million low-income 2-5 year old children from 43 US states/territories that were part of the Pediatric Nutrition Surveillance System (PedNSS) from 2008 through 2011

(Centers for Disease Control and Prevention [CDC], 2013). The researchers found that 19 of the states/territories witnessed a significant decrease in obesity prevalence from 2008 to 2011. However, it was also found that 21 states/territories did not see a change in the prevalence of childhood obesity and three experienced a significant increase (CDC, 2013). To add to the controversy over trends, a recent study used data from NHANES, the same data used by Ogden and colleagues (2014), and showed that overweight and obesity trends have increased significantly between 1999 and 2014, even among 2-5 year old children (Skinner, Perrin, and Skelton; 2016). A more recent study by Skinner and colleagues (2018), demonstrated that from 2015 through 2016 there was an increase in obesity prevalence among preschool-aged children.

What can account for these mixed findings? Although Ogden (2014) and Skinner (2016) used the same NHANES dataset, they found different patterns mainly because the baseline year used by each study was different. While Ogden and colleagues (2014) used 2003 as their baseline; Skinner and colleagues (2016, 2018) used 1999 as theirs. This difference in baseline measurement is important since the prevalence of childhood obesity peaked in 2003; so any comparison to that year seems like a decrease. However, when the baseline is before 2003, then a downward trend in the prevalence of childhood obesity is no longer evident (Dietz, 2016). Although this recent literature on childhood obesity seems to provide inconclusive findings, the literature is in agreement that childhood obesity remains a problem that needs to be addressed through the creation and implementation of prevention interventions and policies.

Childhood overweight and obesity are not only a health problem, but also a health equity issue: overweight and obesity disproportionately affect more low-income and minority children than White and middle class children (Caprio et al., 2008; Ogden et al., 2014). A nationally representative study with 9120 participants found that childhood obesity was more prevalent

among Latino/a children than among any other ethnic group (Ogden et al., 2014). In fact, the prevalence of childhood overweight and obesity among Latino/a and Black children was higher (38.9% and 35.2%, respectively) compared to the prevalence among their White (28.5%) and Asian (19.5%) counterparts (Ogden et al., 2014). These rates are comparable to a recent study among 15,418 children where it was found that the prevalence of overweight and obesity at age 4 was 29.9% (Guerrero et al., 2015). This study also found that compared to White children, Latino and Black children had higher BMI scores at age 4, and had higher rates of change in BMI score trajectories at ages 5 and 6 (Guerrero et al., 2015).

Consequences of Childhood Overweight/Obesity

Childhood overweight and obesity have gathered substantial attention given the long-lasting detrimental health and mental health consequences in children (Kelsey, Zaepfel, Bjornstad, & Nadeau, 2014; Park, Falconer, Viner, & Kinra, 2012). For instance, it has been shown that childhood obesity can lead to the development of metabolic syndrome, dyslipidemia, type 2 diabetes, nonalcoholic fatty liver disease, hypertension, chronic inflammation, asthma, and sleep apnea, breast cancer, and polycystic ovarian syndrome, among others (Ebbeling, Pawlak, & Ludwig, 2002; Kelsey et al., 2014; Park et al., 2012). In terms of mental health consequences, childhood obesity has been found to be associated with lower self-esteem, eating disorders, and higher depression and anxiety levels (Zametkin, Zoon, Klein, & Munson, 2004). Although all these consequences of childhood obesity are worrisome, one of the main concerns is that children who are overweight/obese will be more likely to remain overweight/obese during adolescence and even adulthood (Cunningham, Kramer, & Narayan, 2014; Morrison, Friedman, & Gray-McGuire, 2007; Singh, Mulder, Twisk, Van Mechelen, & Chinapaw, 2008). A study (n = 771) followed 6-19 year old children for about 25 years and found that 63% of participants

who were overweight as children were obese as adults (Morrison et al., 2007). Due to the detrimental and long-lasting consequences of childhood obesity, compounded with the fact that childhood obesity can continue into adulthood, it is important to prevent its development as early as possible.

Although prevention interventions need to be implemented to decrease overweight/obesity trends, it is first necessary to determine their etiology (Ogden et al., 2007). Regarding the causes of childhood overweight and obesity, it can be argued that a myriad of factors influence their development, which can start as early as pregnancy. In fact, there are multiple risk factors during pregnancy and infancy that are associated with childhood obesity such as maternal BMI, gestational weight gain, gestational diabetes, refraining from breastfeeding, and depression, among others (Kim, Sharma, & Callaghan, 2012; Lau, Liu, Archer, McDonald, & Liu, 2014; Olson, Strawderman, & Dennison, 2009; Taveras, Gillman, Kleinman, Rich-Edwards, & Rifas-Shiman, 2010; Whitaker, Jarvis, Beeken, Boniface, & Wardle, 2010). Although the development of childhood overweight and obesity start as early as the gestational period (Hillier et al., 2007), the majority of prevention interventions that have been developed focus on implementing interventions among school age children, which may be too late. Therefore, identifying early precursors of obesity and developing prevention interventions during pregnancy and infancy can aid in the quest of *preventing* childhood obesity.

A potential risk factor of interest is maternal depression since it affects 10- 30% of US women during the perinatal period (defined as the beginning of pregnancy through one year postpartum), and about 14% of women at any other point in life (Bennett, Einarson, Taddio, Koren, & Einarson, 2004; Gavin et al., 2005; Pooler, Perry, & Ghandour, 2013). Although depression can affect everyone, it has been found to be more prevalent among low-income

women (20 to 30%) when compared to middle-class women (10 to 15%) (Bennett et al., 2004; Gaynes et al., 2005; Hobfoll & et al., 1995; Pooler et al., 2013). In fact, a recent study investigated whether income, education, and occupational prestige were associated with postpartum depression (Segre, O'Hara, Arndt, & Stuart, 2007). They found that the most significant predictor of postpartum depression was low income. A more recent study indicated that about 20% of low-income women who were part of the Women, Infants, and Children (WIC) program experienced postpartum depression (Pooler et al., 2013). Regarding the association between childhood obesity and maternal depression, there have been a few studies that have examined this association and overall, the current state of the literature is mixed and inconclusive (Lampard, Franckle, & Davison, 2014; Milgrom, Skouteris, Worotniuk, Henwood, & Bruce, 2012). Therefore, it is important to examine the association between maternal depression and childhood obesity in diverse populations using longitudinal and high quality data.

Purpose

The purpose of this study was to investigate the relationship between maternal depression and childhood obesity development among low-income families from different minority groups. Using current literature in the field of childhood obesity and drawing upon socio-ecological, feminist, and intergenerational transmission theories and concepts, this study investigated the influence of maternal depression on the development of childhood obesity through different mechanisms. This study also aimed to identify growth patterns from birth to age nine. Finally, this study examined the role that sleep, television viewing, outdoor play, dietary intake, breastfeeding, and maternal involvement had on the development of risky growth trajectories.

Data and Objectives

This study used data from the Fragile Families and Child Wellbeing Study, one of the

most comprehensive, longitudinal studies of non-marital births in the United States (Reichman, Teitler, Garfinkel, & McLanahan, 2001). This study used data from time points at birth, 1, 3, 5, and 9 years. This study had four main objectives: 1) to examine longitudinal associations between early maternal depression and childhood overweight and obesity at age nine; 2) to examine longitudinal associations between early maternal depression and childhood growth trajectories from birth to age nine; 3) to determine whether factors such as sleep, outdoor play, television viewing, dietary intake, breastfeeding, and maternal involvement were associated with risky growth trajectories; 4) to determine whether growth trajectories differed by race/ethnicity. Overall, this study investigated the role that risk and protective factors for childhood obesity were associated with the development of risky growth trajectories using a national dataset of non-marital, low-income mothers.

Contributions to the Literature

It is expected that this study will make the following contributions to the field:

1. Address the mixed findings in the literature regarding the effects of early maternal depression on child overweight and obesity risk by employing a national dataset of non-marital mothers in the US.

2. Investigate the effects of maternal depression on child overweight and obesity risk by using early maternal depression from two time points (1 and 3 years after birth).

3. Determine whether there are differences in growth patterns based on racial/ethnic backgrounds.

4. Further current theoretical knowledge by investigating factors at different levels that play a role in the development of childhood overweight and obesity.

CHAPTER 2: LITERATURE REVIEW

This chapter presents the theoretical frameworks that were used to guide this study as well as the current literature available on maternal depression and childhood obesity. Moreover, this chapter offers a review of the information available on the independent associations between the potential risk and protective factors during the first 5 years related to childhood overweight and obesity. This chapter first provides a summary of socio-ecological, intergenerational transmission, and feminist theories, which guided this study. Then, I provide a review of the association between childhood overweight and obesity and maternal depression. After this, a review of potential risk and protective factors (sleep, television viewing, outdoor play, dietary intake, maternal involvement, and breastfeeding) of childhood overweight are reviewed. Subsequently, sociodemographic factors deemed relevant to this study are presented. This chapter concludes with the research questions and the hypotheses that were evaluated in the study.

Theory

Socio-ecological theory, developed by Urie Bronfenbrenner's (1979), is the predominant integrative framework used in the field of childhood obesity. This model bridges the gap between theories that only take into account the micro environment and those that only address the macro environment. This framework acknowledges the influence that different contexts have on the individual from the personal level all the way to the influence that policies and culture have on people (Bronfenbrenner, 1979). However, given that this theory was developed to explain human development, it is necessary to use an adaptation of the socio-ecological model that has been developed exclusively to map the influence on the development of childhood obesity, such as the 6 C’s model (Harrison et al., 2011). Moreover, it is necessary to supplement

the 6 C's model to better understand maternal depression and childhood overweight and obesity. Therefore, intergenerational transmission, and feminist theory were also used to guide this study.

Social-ecological theory- 6 C's Model

The 6 C's model, a model based on Bronfenbrenner's socio-ecological theory, was developed in order to show the range of influences on the development of childhood overweight and obesity (Harrison et al., 2011). This model suggests that there are multiple factors at multiple levels that are associated with the development of childhood overweight and obesity (Harrison et al., 2011). Moreover, this model suggests that there are different dimensions that range from the personal (micro-level) all the way to the cultural level (macro-level) that influence the development of childhood overweight and obesity (Harrison et al., 2011). According to this model, there are a total of 6 levels: cell, child, clan, community, country, and culture. Factors contributing to the development of childhood overweight and obesity each fall under one or more of these levels (Harrison et al., 2011). When focusing particularly on maternal characteristics, each sphere has multiple factors that play a significant role in the development of obesity, and thus, using this framework to explain their influence seems appropriate. For instance, maternal obesity, education, mental health, and socioeconomic status are all factors that have been shown to influence the development of childhood overweight and obesity and they can be placed under the family level (Harrison et al., 2011).

Moreover, the relationship between the mother and the child is a particular system or dyad that can be placed across the 'clan' and 'child' levels and demands particular attention. Mothers are very important not only for the physical, but also for the emotional development of children and the attachment formed between mother and infant starts very early in life (Ainsworth, 1979). However, there are multiple barriers that might prevent a mother from being

responsive to her child's needs, including maternal depression (Feng, Shaw, Skuban, & Lane, 2007) and the stress created by low socioeconomic status (Cook & Frank, 2008; Diener, Casady, & Wright, 2003). The lack of responsiveness to children's cues and needs can have detrimental consequences on the mother-child relationship and they can also affect the wellbeing of the child. Furthermore, other factors such as race/ethnicity, gender, and dietary intake fall under the child-level (Harrison et al., 2011). Overall, different factors at each level contribute to the development of childhood overweight and obesity. By using the 6 C's socio-ecological model, the interrelatedness of these factors can be better understood and examined.

Intergenerational transmission: An application of Social Learning Theory

Intergenerational transmission has been commonly used to explain certain behaviors such as intimate partner violence (IPV) (Black, Sussman, & Unger, 2010), but more recently it has been used to explain childhood obesity (Jääskeläinen et al., 2011; Whitaker et al., 2010). Although not a theory per se, the concept of intergenerational transmission is based on social learning theory, originally developed by Albert Bandura (Bandura, 1969, 1977). Social learning theory posits that children learn through observation and modeling of people's actions (Bandura, 1971). Moreover, social learning theory suggests that children's behaviors will be reinforced through reward or punishment; if the child is rewarded, it is more likely that s/he will repeat that action (Bandura, 1969, 1971). Although social learning theory is a strong theoretical framework to help explain the observation and learning of obesogenic behaviors, it does not take into account the genetic factors that play an important role in childhood obesity. For that reason, intergenerational transmission is a broader concept that encompasses genetic and epigenetic factors that can help explain childhood obesity development.

According to intergenerational transmission, obesity is passed down from one generation

to the next through genetics, epigenetics, and observed obesogenic behaviors (Jääskeläinen et al., 2011; Rey-López, Vicente-Rodríguez, Biosca, & Moreno, 2008; Smith et al., 2009). In fact, children of overweight/obese parents are more likely to be overweight/obese at birth suggesting genetic transmission of obesity (Jääskeläinen et al., 2011; Smith et al., 2009). One study (n = 7078 children, n = 4432 families) suggested that only 2.3% of children with normal-weight parents developed obesity; compared to 21.7% of children who had obese parents and 35.3% of children with severely obese parents (Whitaker et al., 2010). This information shows that intergenerational transmission of obesity can be quite high and it suggests that genetic and epigenetic influences may be at play.

Intergenerational transmission can also be used to explain the transmission of obesogenic behaviors across family members (Rey-López et al., 2008). For instance, behaviors that promote health and wellbeing are set early in life through observation, learning, and modeling. However, children are also prone to learn and model behaviors from parents that might not promote a healthy lifestyle. For instance, obesogenic behaviors such as television viewing, physical activity, and type of food intake can be modeled and learned by children. Some of these obesogenic behaviors, such as television watching, are associated with childhood obesity (Rey-López et al., 2008; Taveras et al., 2010). Moreover, a recent study showed that parental television viewing was the main predictor of children television viewing (Bleakley, Jordan, & Hennessy, 2013). Thus, demonstrating that television viewing by children can be influenced and modeled after parents, so if parents spend more than 2 hours watching television, the children will as well (Bleakley et al., 2013). This is problematic given that behaviors set early on in life are likely to be continued into adulthood and may be more resistant to change. Another behavior that has been shown to be associated with childhood obesity is physical activity, particularly

among younger children (Hills, Andersen, & Byrne, 2011). In fact, studies evaluating physical activity among children have found that conditional parental support, such as carrying out physical activities with the children, is associated with more physical activity and less sedentary behaviors (Beets, Cardinal, & Alderman, 2010). Finally, another behavior that is associated with childhood obesity is fast food and soda consumption (Taveras et al., 2010). In the case of food preferences by children, some studies have found that there is a genetic influence, making certain children more prone to eat similar foods to those eaten by the parents and siblings (Breen, Plomin, & Wardle, 2006). However, the environment also plays a significant role since modest but significant evidence shows that children learn parent's food preferences (Young, Fors, & Hayes, 2004).

Other obesogenic factors can also be cultural and transmitted from parents to children. One of the best examples is acculturation, which is the acquisition of some cultural traits from the host country. Research has shown that recently arrived Asian and Latino immigrants are more likely to have diets that include more consumption of fruits and vegetables, less consumption of soda and they are also more likely to cook meals at home compared to White participants (Allen et al., 2007). However, as parents and children become more acculturated, consumption of fruit and vegetables among Latinos decreases and soda intake increases, making their diet more similar to that of native born White participants. This demonstrates that acculturation can have some outcomes that are detrimental for health, and as parents acculturate to new diets, their children do as well (Allen et al., 2007).

Finally, as part of the intergenerational transmission concept, it is important to discuss the role of poverty, which has also been shown to be "passed down" through generations (Bird, 2007). According to different studies, the transmission of poverty is mostly due to the lack of

access to material resources such as food and housing, and also due to the lack of community resources such as access to quality education and safe neighborhoods where the children can play freely (Bird, 2007). This transmission of poverty is of particular interest to the field of childhood obesity given that previous research has shown that one of the main social determinants of health is socioeconomic status (SES) (Marmot, 2005). Studies have shown that low SES is inversely associated with childhood adiposity and obesity (Shrewsbury & Wardle, 2008). Overall, intergenerational transmission can highlight how obesity is transmitted from mother to child through genetics, behaviors, socioeconomic, and environmental factors. However, it is important to bear in mind that while intergenerational transmission of child overweight and obesity is observable, its processes and mechanisms are not yet well understood and it should not be viewed as deterministic. In fact, research has demonstrated that it may be possible to interrupt many of these intergenerational cycles by developing interventions for parents and children (Sacher et al., 2010).

Feminist Theory

Feminist theory derives from critical theory and it aims to critique systemic oppression directed primarily towards those who identify as women (Creswell, 2012). Feminist theory came about as part of a movement in response to a patriarchal society, where the voices, struggles, and needs that are heard are primarily those of men, thus leaving behind and ignoring those of women (Lugones & Spelman, 1983). Although feminist theory has multiple branches such as Chicana feminism, Black feminism, and Asian-American feminism, among others, there are some principles that are similar across the different movements (White & Klein, 2008). Principles include the search for equality and justice for women (Garcia, 1989). However, each branch of feminism that was born during the 1960's and 1970's had specific onsets and focused

on challenging oppressive traditions that were particular to each culture. At the same time, during the 1980's feminist theory increasingly focused on the intersection of race, socioeconomic status, and sex as the main source of oppression (Collins, 1989; Garcia, 1989). The concept of intersectionality was first brought up during this time and it refers to the intersecting identities of women of color; race, class, and gender and the ways in which women's personal lives are oppressed by the larger society (Collins, 1986, 1989; Crenshaw, 1989). Moreover, second-wave feminism –which I refer to as a way to integrate the different branches of feminism, rather than as a way to reduce the importance of each of them– refers to the multiple disadvantages and the oppression that women face on their everyday lives (Collins, 1989; Garcia, 1989).

Health and social inequalities based on gender are feminist concerns. Females are still at a greater disadvantage on many health metrics when compared to men (Sen, Ostlin, & George, 2007). Although women's life expectancy is, on average, five years higher than that of males, women suffer from more illnesses than men throughout their lifetime, which reduces their quality of life (Read & Gorman, 2010). Women in the US and around the world have lower access to economic resources, tend to have low-paying jobs, and are less likely to hold positions of leadership (Read & Gorman, 2010). At the same time, women are more likely to obtain lower remuneration for the same work performed by males; indicating that sex discrimination is one of the main causes of these disparities (Corbett & Hill, 2012). These differences in income and resources are reflected in access to health and health-related resources, which can place women at a higher risk for untreated illnesses (Sen et al., 2007). Another way in which women are clearly disadvantaged and disproportionally underrepresented, is in the research arena, where most clinical research trials have historically been conducted with males (Simon et al., 2005).

This issue was raised in a study in 2000 study that suggested that for studies funded by the National Institutes of Health (NIH), only 38% enrolled women (Harris & Douglas, 2000). Since then, many funding agencies have implemented guidelines and policies to increase enrollment of women in clinical trials, but it remains low (Geller, Koch, Pellettieri, & Carnes, 2011; Liu & Mager, 2016). Increased enrollment of women is necessary in order to adequately provide disease-sex-specific recommendations (Melloni et al., 2010). Yet another problem is that symptoms and issues that are predominantly faced by women may be left out of research (Read & Gorman, 2010).

An illustrative example of health inequality among men and women is depression. Although it has been shown that women report higher levels of depressive symptoms than men and that depression can exacerbate other health conditions, depression is usually underdiagnosed, undertreated, and it is usually expected to go away on its own (Regitz-Zagrosek, 2012). This lack of attention to women's health and mental health, along with lack of research conducted on women, translates into a lack of attention in healthcare and policy development. Although depression could be easily diagnosed by implementing universal depression screenings, more than half of women with depression go undiagnosed (Beck, 2006). As a consequence, mothers with health and mental health issues may not receive the care they need during or after pregnancy, which in turn affects the lives and development of their offspring (Dabelea, 2007; Ertel, Koenen, Rich-Edwards, & Gillman, 2010; Milgrom et al., 2012; Poston, 2011; Taveras et al., 2010).

Another aspect that needs to be taken into account is the societal pressure that women face, along with sources of oppression. Men and women are socialized very differently from early childhood; for instance, even before birth, children are already gendered in strict ways;

boys are given blue and green clothes, along with cars and trucks to play with, while girls are given pink and purple clothes as well as dolls and tea sets (Blakemore & Centers, 2005). Gender roles and expectations do not stop there; in fact, they continue throughout life. For instance, during adulthood, women are expected to marry, have children, and enjoy every second of marriage and motherhood, since it is supposed to be a defining characteristic of womanhood (Annandale & Clark, 1996; Garcia, 1989). In fact, reproduction is highly scrutinized among women; if they decide not to have children, then they are deemed selfish (Letherby, 2002). However, if they do have children, it is expected that they have them during adulthood, in a heterosexual couple, after marriage, and once the couple is financially stable. Mother shaming and/or blaming usually does not end there, in many instances, mothers are blamed for all sorts of issues that are related to their children and their families (Letherby, 2002). For example, if mothers are depressed, they are blamed for not enjoying what is deemed to be the best time of womanhood, which puts women at further risk of not disclosing their feelings and not treating emotional complications such as postpartum depression (Abrams, Dornig, & Curran, 2009; Beck, 2002). Depression is only one example of many, since mothers tend to be blamed for many physical and/or emotional issues that the child has.

Finally, women's oppression not only takes place at the individual and family level, but it is also reinforced at the policy level. In fact, there are many policies that promote mother blaming and double standards. For instance, recommendations for breastfeeding suggest that women should exclusively breastfeed for the first 6 months of the child's life given all the multiple benefits that it can have, including decreased risk for childhood obesity (Arenz, Rückerl, Koletzko, & von Kries, 2004; Horta & Victora, 2013). Regardless of these recommendations, only 18.8% of women exclusively breastfeed for 6 months in the US (CDC,

2014). Previous studies have shown that one of the main reasons for women to stop breastfeeding early is the need to return to work (Kimbro, 2006; Ogbuanu, Glover, Probst, Liu, & Hussey, 2011). Currently, there are not any federal policies that promote paid maternity leave, which would allow women to stay home for longer periods of time and could increase breastfeeding rates (Calnen, 2007; Guendelman et al., 2009). Moreover, there are not any current policies that can support mothers who decide to go back to work but who desire to continue breastfeeding, such as providing onsite child care or making it possible for mothers to pump at work (Fein, Mandal, & Roe, 2008; Johnston & Esposito, 2007). Although it is quite clear that breastfeeding is best for the majority of children, maternity leave and workplace policies are not implemented to promote it. This lack of policy implementation particularly affects women who are low-income and who cannot afford to stay home more than the required amount of time (Calnen, 2007) and whose workplaces are not amenable to the needs of breastfeeding workers.

Overall, feminist theory offers an important framework to investigate women's health and mental health, particularly among low-income women. Feminist theory offers a framework to understand how the lack of attention that is paid to women ultimately affects children, families, and society as a whole. As mentioned earlier, depression among women is usually undiagnosed and it is necessary to call for more stringent approaches to detect depressive symptoms as soon as possible. This study identified the role that maternal depression had on childhood obesity development to demonstrate public health policy makers and researchers that it is necessary to implement universal depression screening, especially during early childhood. Also, from a feminist perspective, women are usually blamed for everything that happens to their children, whether it is good or bad. Blaming mothers for the development of childhood obesity development is not the exception. However, assuming that children are more likely to be

overweight or obese because their mothers are depressed has no scientific basis. Yet, studies have rarely explained the reasons for which this association might be present in the first place. To better understand the key contributors of childhood obesity development, this study investigated the effects that different obesogenic behaviors (e.g. lack of sleep and fast food consumption) had on childhood obesity development. Testing the effects that these obesogenic behaviors have on childhood obesity development will allow researchers in the future to identify the mechanisms through which maternal depression can exacerbate childhood obesity development. Finally, feminist theory offers a framework to indicate how the lack of attention that is paid to women ultimately affects children, families, and society as a whole.

The socio-ecological framework also provides an important framework to understand and test the factors that influence childhood obesity development at different levels. This study specifically expected that factors at the child and clan levels would impact childhood overweight and obesity development. Finally, applying the intergenerational transmission concept, this study expected to find that genetics (e.g. maternal weight) and obesogenic behaviors (e.g. lack of sleep and fast food consumption) increased the risk of children to follow risky growth trajectories. Having explained the different ways in which socio-ecological, intergenerational transmission, and feminist theories guided this study, the next section contains a review of the literature on child overweight and obesity and their association to maternal depression and potential risk and protective factors for childhood overweight and obesity.

Review of the literature

Childhood overweight and obesity

According to the Center for Disease Control and Prevention (CDC), childhood overweight and obesity are assessed using Body mass index (BMI), which is calculated using the

weight and height of the individual (kg/m^2) (CDC, 2015). Childhood overweight is defined as having a BMI at or above the 85^{th} percentile and childhood obesity is defined as having a BMI at or above the 95^{th} percentile (CDC, 2015). The percentiles are calculated based on CDC sex-specific BMI-for-age growth charts, which were developed using a nationally representative sample from the US for children ages 2-19 (Ogden et al., 2014). For children under the age of 2, sex-specific weight-for-length growth charts from the World Health Organization (WHO) are typically used to measure childhood obesity (de Onis, Garza, Onyango, & Martorell, 2006).

One of the main concerns about childhood obesity is that it can have long-lasting detrimental consequences in children (Ebbeling et al., 2002). More specifically, it has been shown that childhood obesity can lead to the development of type 2 diabetes, metabolic syndrome, hypertension, asthma, and sleep apnea, among others (Ebbeling et al., 2002; Kelsey et al., 2014). Childhood obesity can also have mental health repercussions including lower self-esteem, eating disorders, and higher depression and anxiety levels (Zametkin, Zoon, Klein, & Munson, 2004). One of the main consequences of childhood obesity is that children who are overweight/obese will be more likely to remain overweight/obese during adolescence and even adulthood (Cunningham et al., 2014; Singh et al., 2008). A recent longitudinal study found that children who were at the 85th percentile at the age of 5 had a 25% probability of becoming obese at the age of 14, and those who were at the 95th percentile at the age of 5 had a 47% probability of being obese at the age of 14, compared to a probability of 6% among those who had a normal weight at the age of 5 (Cunningham et al., 2014). This study also found significant disparities among racial/ethnic groups. Obesity was much more prevalent among Latino children at all ages and it was more prevalent among Black children after third grade compared to their White counterparts (Cunningham et al., 2014). Given all these consequences, it is important to prevent

the development of childhood obesity as early as possible.

Maternal depression

Depression has been defined as a common psychological disorder characterized by depressed mood (dysphoria) or lower interest or pleasure in most activities (anhedonia) the majority of the time (American Psychiatric Association, 2013). Maternal depression has mostly been studied in relation to the postpartum period, which includes the first year of the baby's life, when an estimated of 10-30% of mothers display elevated depressive symptoms (Bennett et al., 2004; Hobfoll & et al., 1995; Pooler et al., 2013). The highest rates of depression tend to be found among low-income and minority women, whose rate of depression falls between 20-30% (Hobfoll & et al., 1995; Pooler et al., 2013; Segre, O'Hara, Brock, & Taylor, 2012; Segre et al., 2007). A study based on 4,332 postpartum women in the US investigated whether income, education, and occupational prestige was associated with postpartum depression (Segre et al., 2007). The study found the strongest significant predictor of postpartum depression was income (Segre et al., 2007). A more recent study of 75,234 women indicated that about 20% of low-income women who were part of the Women, Infants, and Children (WIC) program experienced postpartum depression (Pooler et al., 2013). In contrast to post-partum or perinatal depression, depression among mothers that does not occur during pregnancy or within one year after giving birth is referred to as maternal depression and its prevalence is at least 10% (Ertel, Rich-Edwards, & Koenen, 2011). Rates of major depression are difficult to determine and compare among studies because the majority of studies assess *depressive symptoms* rather than *major depression* (Duarte, Shen, Wu, & Must, 2012; Gross, Velazco, Briggs, & Racine, 2013; Surkan et al., 2014; Wang et al., 2013). Moreover, the measurement used to assess either depressive symptoms or major depression varies widely, making it difficult to compare.

Regardless of whether the mother has postpartum depression or depression at any other point in time, it is important to focus on the detrimental consequences that it can have on children. It has been shown that women who suffer from maternal depression have difficulties in the mother-child attachment relationship and these effects last up to three years after birth (Milgrom, Ericksen, McCarthy, & Gemmill, 2006). Specifically, mothers with postpartum depression have been found to be less responsive to their children's needs and less verbally stimulating (Milgrom et al., 2006). Other consequences of postpartum depression on the infants include more awakenings throughout the night and more minor health issues (Gress-Smith, Luecken, Lemery-Chalfant, & Howe, 2012). Also, studies have shown that children of mothers who had postpartum depression exhibited more aggression, more anxious-depressed behavior, and also more attention-hyperactivity disorder compared to children of mothers without postpartum depression (Beck, 1998).

Given the importance that childhood obesity has received in recent years, some researchers have started to look at the association between maternal depression and childhood obesity, which as of now, shows mixed findings (Lampard et al., 2014; Milgrom et al., 2012). Given the importance of timing when it comes to depression, it is important to make a distinction between research that has been conducted around pregnancy and the postpartum period (perinatal period) and research that has been conducted on maternal depression at any other time. The next section will first discuss the current knowledge on postpartum depression and childhood obesity, followed by a discussion on maternal depression and childhood obesity.

Perinatal depression and childhood obesity

Studies looking at the association between perinatal depression and childhood obesity have yielded mixed findings around the world. For instance, a systematic review looking at the

association of perinatal depression and childhood obesity, suggested that there is limited evidence of an association and most of it is low quality, which does not lend itself to conclusive findings (Milgrom et al., 2012). Below is a discussion of some studies that have been conducted in the US investigating the association between perinatal depression and childhood obesity.

One of the few studies that has been conducted on perinatal depression using longitudinal data is Project Viva, a diverse prospective study of 1,826 pregnant women and their offspring in Massachusetts (Taveras et al., 2010). Depression was measured at mid-pregnancy using the Edinburgh Postnatal Depression Scale [EPDS] with a cutoff score of 13. It was shown that Latino children of mothers with elevated depressive symptoms during the antenatal period were 2.96 (95% CI: 1.55- 5.62) times more likely to develop childhood obesity at age 3 years compared to children whose mothers did not present elevated symptoms. However, after adjusting for parental BMI and SES, the effect of maternal depression was no longer significant. A second study that focused primarily on a minority group used data from 181 Latino mother-child dyads (Wojcicki et al., 2011). Depression was assessed during the antenatal period and then 4-6 weeks postpartum using a Center for Epidemiologic Studies Depression Scale [CES-D] score ≥ 16, an EPDS score ≥ 13, or a clinical formal diagnosis of depression. Children's anthropometric measurements were assessed at 6, 12, and 24 months of age. This study found no association between elevated depressive symptoms and childhood obesity (Wojcicki et al., 2011). Instead, maternal depression was associated with children being underweight. These results need to be taken with caution since this study was particularly limited by small sample size and it followed the children only to age 2.

Another study used the same Project Viva dataset as the study by Taveras and colleagues (2010) but they used a sample of 838 mother-child dyads (Ertel et al., 2010). These data were

comprised of mostly white, married, middle-income women. Depression was assessed using the EPDS at mid-pregnancy and also at 6 months postpartum. Anthropometric measures were gathered among children at ages 1, 2 and 3 years. It was shown that antenatal depression was not associated with BMI percentile at age 3 years but it was associated with higher central adiposity measured using subscapular skinfold thickness (Ertel et al., 2010).

A more recent study that used nationally representative data from the Early Childhood Longitudinal Study- Birth Cohort (ECLS-B; n = 6,550) explored the relationship between maternal depressive symptoms with longitudinal BMI growth trajectories (Surkan et al., 2014). In this study maternal depressive symptoms were assessed using the CESD when the child was 9 months of age. Child weight-for-length (WFL) was assessed when the child was 9 months and then BMI was calculated when the child was 4, 5, and 6 years of age. Random effects models were used to construct longitudinal BMI trajectories. The results showed that children who had mothers with mild symptoms of depression when they were nine months, had higher BMIs over time, compared to the children whose mothers had no depressive symptoms. However, this association was no longer significant in the fully adjusted model.

Summary and limitations of literature review on perinatal depression and childhood obesity

Overall, the literature on the association between perinatal depression and childhood overweight and obesity is inconclusive and is also limited. There are not many longitudinal studies that analyze the effect that maternal mental health can have on the children's weight status. Moreover, the few studies that have been published in this area have many limitations. First of all, the sample size of some studies was quite small. For instance, the study conducted by Wojcicki et al. had a very small sample size (n = 181) given that the population of interest was Latino children whose mothers had depression during pregnancy or postpartum and who were

overweight or obese. This study had a low prevalence of childhood overweight and obesity, which led them to have cell sizes of 10 or less. Another limitation is that there is a wide variety of depression measurements that are used, which makes comparison across studies difficult. For example, some studies use the CESD, while others used the EPDS to obtain information on maternal depressive symptomology. Using different measures can provide different results on the associations found because the cutoff points for depression and level of chronicity are different. Therefore, it would be useful to use one standardized measure with high specificity and sensitivity. Another limitation regarding depression and its measurement is that studies usually only report on the presence of depressive symptoms rather than major depression, which might make a huge difference since mothers with major depression might differ from those with depressive symptoms. Thus, reporting the presence of depressive symptoms as well as major depression, whenever possible, is highly recommended.

Maternal depression, childhood obesity, and risk factors for childhood obesity

Current research on maternal depression does not differ much from that on perinatal depression and childhood obesity. In fact, a systematic review of nine studies published between 1980 and January 2013 suggested that given the early stages of this literature, there are mixed findings depending on the severity of depression (Lampard et al., 2014). Moreover, this systematic review suggested that a better understanding of theory-driven moderators and mediators between maternal depression and childhood obesity is needed. Below is a description of key studies that have been conducted in the US on maternal depression and childhood obesity.

The majority of the studies investigating the association between maternal depression and childhood obesity in the US have demonstrated that a positive association exists (Duarte et al., 2012; Wang et al., 2013). A longitudinal study using national data from the Study of Early Child

Care and Youth Development (SECCYD) on 1,090 mother-child dyads found a positive association. Specifically, the study showed that children of mothers with recurrent depression at 3 measurement points were 1.88 times more likely to be overweight or obese at first grade and third grade, and two times more likely at sixth grade after controlling for child and maternal confounding variables (Wang et al., 2013). Another study using cross-sectional data from 401 low-income mothers and 5-year old children found that children of mothers with moderate to severe symptoms of depression were 2.62 times more likely to be overweight or obese compared to children of mothers without depression (Gross et al., 2013). In addition, this study looked at obesogenic behaviors and their association with maternal depression. Researchers found that mothers with mild symptoms were more likely to have children who consumed sweetened beverages, were less likely to have breakfast, and ate out more often compared to mothers without depressive symptoms. Moreover, it was found that mothers with mild and moderate symptoms had children who slept fewer hours, in average, compared to mothers without depressive symptoms (Gross et al., 2013).

Other studies have gone beyond testing the association between maternal depression and childhood obesity by testing some mediators for this association (Duarte et al., 2012; McConley et al., 2011). A cross-sectional study (n = 4,601) investigated the mediators between maternal depression and childhood obesity and the role of parenting quality (McConley et al., 2011). All measures were gathered when the child was in fifth grade. The results showed that higher levels of maternal depressive symptoms were associated with higher child BMI percentile. Also, it was shown that this association was mediated by lower parenting quality, which in turn was associated with obesogenic behaviors such as more sedentary behaviors and more leisure activities. Finally, it was shown that sedentary behaviors and leisure activities were associated

with higher child BMI percentile (McConley et al., 2011).

Another study looked at the longitudinal association of maternal depression and childhood obesity using Early Childhood Longitudinal Study- Kindergarten (ECLS-K), a nationally representative dataset of more than 21,000 participants (Duarte et al., 2012). This study showed that among girls, severe symptoms of maternal depression during kindergarten were associated with lower BMI z-scores at third grade but with higher BMI z-scores at fifth grade (Duarte et al., 2012). Also, it was shown that severe symptoms at third grade were associated with higher BMI z-scores in fifth grade, among girls. For boys, severe depressive symptoms were associated with higher BMI z-scores at fifth grade. This study also looked at mediators that were related to physical activity and sedentary behaviors (Duarte et al., 2012). It was found that among girls, severe maternal depressive symptoms and childhood obesity were mediated by lower physical activity compared to peers and increased television watching during weekends. Among boys, it was shown that healthy eating mediated the relationship between severe maternal depressive symptoms and childhood obesity (Duarte et al., 2012).

Summary and limitations of literature review on maternal depression and childhood obesity

Overall, much of the literature reviewed on maternal depression and childhood obesity in the US suggests that there is a positive association. Moreover, most of the mechanisms studied are related to food intake and exercise levels, potentially ignoring other mechanisms that might further explain the relationship between maternal depression and childhood obesity. However, this literature has similar limitations to those observed on the association between perinatal depression and childhood obesity. For instance, the majority of these studies are unable to account for intrauterine factors, which can have repercussions on the development of childhood obesity. Another limitation of the studies reviewed is that many of them use on cross-sectional

data, which does not say much about the long-term effects of maternal depression or about the directionality of the relationship. Thus, it is necessary to investigate the long-term effects that maternal depression has on children's obesity development, which in turn can also help with assessing the directionality of this relationship. Thinking from a socio-ecological perspective, the literature reviewed does not clarify the role of factors that go beyond the child level. Thus, it is necessary to investigate the child level as well as other levels of the socio-ecological model to identify how they impact children's development of childhood obesity. Finally, from an inter-generational transmission point of view, it is pertinent that research includes information on maternal pre-pregnancy weight and intrauterine factors that can have a strong association with childhood obesity development.

Besides maternal depression, there are many other risk and protective factors at the child and clan levels that have been identified to be independently associated with childhood obesity development and maternal depression. In the next section, I provide a review of these key factors.

Child-level factors

The factors at the child-level deemed relevant to childhood obesity include behaviors such as sleep, television viewing, physical activity, and dietary intake. Sleep was chosen as a child-level factor given all the effects it has on child development and also because it is a relatively new factor that is being investigated as affecting childhood obesity development (Chen, Beydoun, & Wang, 2008; Speirs, Liechty, & Wu, 2014). The other three factors; television viewing, physical activity, and dietary intake are behaviors that have been consistently used in the literature to test excess of energy intake and decrease in energy output (Martinez, 2000), a concept that has gained noteworthy traction in the field of childhood obesity. Moreover,

all these behaviors have been consistently found to be associated independently with childhood obesity and maternal depression in cross-sectional studies. Below is a review of the information on how these factors have been found to influence childhood obesity development and maternal depression.

Sleep

Sleep is highly important for the cognitive development and wellbeing of children (Sadeh, Gruber, & Raviv, 2003). Sleeping guidelines depend on the age of the child; younger children should have more hours of sleep (Paruthi et al., 2016). For instance, infants between the ages of 4 and 12 months should sleep 12 to 16 hours per day, children between 1 and 2 years should obtain 11-14 hours, children 3 to 5 should have between 10-13 hours, children 6 to 12 should try to get 9-12 hours of sleep, and teenagers between the ages of 13 and 18 should aim for 8-10 hours of sleep per day (Paruthi et al., 2016). Given the importance of sleep and its effects on different areas of child development, amount of sleep among children and its association with childhood obesity have recently begun to be investigated (Chen et al., 2008; Speirs et al., 2014).

A study exploring the association between sleep and childhood obesity used nationally representative data from the Child Development Supplement (CDS) (Snell, Adam, & Duncan, 2007). This study found that children who had at least 11 hours of sleep had lower BMIs, compared to children who slept 9 or 10 hours. Moreover, this study found that going to bed at a later time, was associated with higher BMI five years later (Snell et al., 2007). These findings are similar to those found by studies synthesizing the information available on sleep and childhood obesity (Chen et al., 2008; Magee & Hale, 2012). A meta-analysis suggested that children under the age of 10 whose parents reported less hours of sleep were more likely to be overweight or obese (Chen et al., 2008). Moreover, it was found that children who had short sleep duration had

a 58% increased risk to be overweight or obese, compared to children who had the recommended amount. A systematic review synthesized longitudinal studies and encountered that among children, there was an association between shorter sleep duration and weight gain (Magee & Hale, 2012). In summary, the majority of studies conducted on sleep duration and childhood obesity indicate that shorter sleep duration is associated with higher BMI in children, particularly among younger children who have less autonomy and for whom parents play a more active role.

Sleep patterns among children have also shown to be an area of interest for researchers who study maternal depression. Some studies have shown that there might be an association between maternal depression, particularly during the postpartum period, and child sleep (Dennis & Ross, 2005; Field, 2010). For instance, Dennis and Ross (2005) investigated the relationship between child sleep patterns and maternal depression. They found that mothers with elevated depressive symptoms were more likely to report that their child woke up more often throughout the night and that their child did not sleep well, compared to women with low levels of depression (Dennis & Ross, 2005). However, one of the main limitations of this study is that it used self-report of child and mother sleeping patterns; thus, it is possible that they were not completely accurate. In order to address this limitation, a more recent study used actigraphs on the mothers and the babies in order to measure light and motion activity, providing a more objective measure of sleep patterns (Armitage et al., 2009). It was found that infants from mothers who had depression, a history of depression, or elevated depressive symptoms had shorter sleeping episodes, took longer to fall asleep, and had more awakenings throughout the night compared to infants of mothers without depression (Armitage et al., 2009). Moreover, this study indicated that these sleep patterns among infants were persistent from two to six weeks postpartum (Armitage et al., 2009). Taken together, these studies suggest that there is an

association between higher maternal depression and lower child sleep quality and the repercussions can be long-lasting. It is possible that maternal depression is associated with diminished child sleep, which in turn might be associated with childhood obesity. Besides maternal depression, television viewing is a factor that can affect sleep. A longitudinal study of 1,864 children showed that for every hour increase in television viewing, children had 7 minutes less of sleep per day (Cespedes et al., 2014). Thus, television is an important factor to investigate in relation to childhood obesity and maternal depression.

Television viewing

When investigating the topic of childhood obesity, one of the most commonly studied risk factors include sedentary behaviors such as watching television (Zhang, Wu, Zhou, Lu, & Mao, 2015), during which calorie intake can increase (Francis & Birch, 2006). Although the new recommended amount of television for children is one hours or less per day ("American Academy of Pediatrics: Children, adolescents, and television," 2001; "Media and Young Minds," 2016), children are currently watching about 7.3 hours per day (Rideout, Foehr, & Roberts, 2010). Studies conducted on television viewing and childhood obesity have suggested that children who spend more hours watching television, have higher odds of being overweight/obese (Davison, Marshall, & Birch, 2006; Proctor et al., 2003; Rey-López et al., 2008; Zhang et al., 2015). A study used data from 106 preschool-aged children and found that by age 11, children who watched 3 or more hours of television per day had higher BMI than those who watched television less than 2 hours (Proctor et al., 2003). Another study by Davison, Marshall, and Birch (2006) indicated that cross-sectional findings were not significant for the association between television viewing and childhood obesity. However, the longitudinal analysis demonstrated that girls who watched more than 2 hours of television per day, at three different time points (7, 9,

and 11 years of age), were 13.2 times more likely to become overweight by age 11, demonstrating that television viewing can play an important role in the development of childhood obesity. A systematic review of sedentary behaviors and childhood obesity (Rey-López et al., 2008) found that especially among younger children, spending more than 2 hours watching television was associated with childhood obesity. Similar findings were shown by a more recent meta-analysis (Zhang et al., 2015), which suggested that children who watched television the most had 47% higher odds of childhood obesity, compared to those who watched the least amount of television. Furthermore, the pooled analysis showed that for each hour of television viewing per day, the risk of childhood obesity increased by 13% (Zhang et al., 2015).

Television viewing by children has also been found to be associated with maternal depression, starting very early on (Anand, Downs, Bauer, & Carroll, 2014; Bank et al., 2012; Burdette, Whitaker, Kahn, & Harvey-Berino, 2003; Conners, Tripathi, Clubb, & Bradley, 2007). For example, a study conducted among 6-9 month old children showed that children of mothers who had elevated symptoms of depression, watched more hours of television per day, compared to children of mothers without depressive symptoms (Bank et al., 2012). Another study investigated whether children from 0-2 years of age differed on television viewing based on maternal depressive symptoms, race/ethnicity, and public insurance (Anand et al., 2014). This study found that children of mothers with elevated depressive symptoms were 1.47 times more likely to watch television, compared to mothers with low levels of depressive symptoms. Also, it was found that children with public insurance watched more television compared to children who had private insurance. Moreover, it was found that Black and White children were likely to watch more television compared to children from Latino backgrounds (Anand et al., 2014). The association between maternal depression and television viewing has recently been found among

infants, but previous studies also show that this association also exists among older children. One study found that children of mothers with elevated symptoms of depression watched more hours of television, compared to children whose mothers had low symptoms (Conners et al., 2007). Similar results were found by Burdette et al. (2003), who conducted a cross-sectional study of 3 and 4 year old children on television viewing. The researchers found that if the mothers had elevated depressive symptoms, their children were significantly more likely to watch 3 or more hours of television per day. Overall, there is a noteworthy amount of information suggesting that maternal depression is independently associated with television viewing as early as 6 months of age.

Other common factors that are usually investigated in relation to childhood obesity development are outdoor play and physical activity, since they provide a complementing measurement to investigate how active or sedentary children are. The next section summarizes the literature available on outdoor play, childhood obesity, and maternal depression.

Outdoor play

Outdoor play among young children has decreased by more than 25% since the 1980's (Hofferth, 2009; Hofferth & Sandberg, 2001). However, outdoor play is very important for the physical and psychological well-being of children and it is important to promote it (Burdette & Whitaker, 2005b). From a health perspective, outdoor play is highly important since it is during these times that children are physically active without having to engage in structured activities, therefore making outdoor play a good measure of physical activity (Burdette & Whitaker, 2005b). Moreover, although measuring physical activity using accelerometers is highly recommended, it is particularly difficult to do among preschool-aged children and in large studies; therefore, it is necessary to use alternative mechanisms that can offer an estimate of it

(Burdette, Whitaker, & Daniels, 2004). Burdette and colleagues conducted a study comparing three different measurements of physical activity: 1) accelerometers, 2) parental report using a checklist, and 3) parental recall of minutes playing outdoor daily. This study suggested that the accelerometer measurement of physical activity was significantly correlated with parental report of time spent playing outdoors; therefore, making outdoor play a strong surrogate measure of physical activity (Burdette et al., 2004).

Current studies conducted on the association between outdoor play and childhood obesity are scarce and offer mixed findings. A cross-sectional study (n = 3141) conducted investigated associations between neighborhood safety, outdoor play, television viewing and childhood obesity among 3 year old children (Burdette & Whitaker, (2005a). This study found that television viewing and outdoor play were not associated with childhood obesity at age 3. However, another study using the same dataset but using data from age 5 found that hours of outdoor play and child BMI were negatively associated (Kimbro, Brooks-Gunn, & McLanahan, 2011). In fact, for each hour of outdoor play, there was a half percentile point decrease on child BMI (Kimbro, Brooks-Gunn, & McLanahan, 2011). In both of these studies outdoor play was measured through self-report on the average number of hours the child played outdoors (Burdette & Whitaker, 2005a; Kimbro, Brooks-Gunn, & McLanahan, 2011). Although the information on outdoor play is scarce, there is a large amount of research suggesting that there is a strong association between lower physical activity and childhood obesity (Must & Tybor, 2005; Sherburne Hawkins & Law, 2006). Two reviews on the association between physical activity and childhood obesity (Must & Tybor, 2005; Sherburne Hawkins & Law, 2006) suggest that the majority of the studies conducted indicate there is an inverse association between physical activity and childhood obesity, particularly among older children.

Given that many times the mother is the main child caregiver, she is probably the main person who takes the child out to play. However, when mothers are depressed, they might be less likely to do so (Kimbro & Schachter, 2011). Although there is not much information available on the association between maternal depression and child outdoor play, a study by Kimbro and Schachter (2011) demonstrated that maternal depression moderated the association between neighborhood poverty and maternal fear of children playing outside when the child was 5 years old. Another study examined the relationship between maternal depression and child physical activity among children in Mexico (Fernald, Jones-Smith, Ozer, Neufeld, & DiGirolamo, 2008). This study found that maternal depression when the child was 15 months old was associated with low levels of physical activity when the child was between 4 and 6 years old, even after controlling for confounding variables. Overall, the relationship between outdoor play and childhood obesity offers mixed findings but when a wider range of measures of physical activity are used, there is greater evidence to suggest there is a negative association.

Besides sedentary behaviors and outdoor activity, many studies investigating the etiology of childhood obesity development focus on the role that dietary intake plays (Martinez, 2000). Most of the foods investigated by previous research focus on foods that are high in calories and fats such as fast food and soda, and also on foods that are a nutritious source of energy like fruits and vegetables. The next section provides a summary of the key findings on the relationships between these food sources and their association with childhood obesity.

Dietary intake

Dietary intake among children is also a concern among many researchers given that diet has changed drastically over the past decades (Bowman, Gortmaker, Ebbeling, Pereira, & Ludwig, 2004). Intake of sugar-sweetened beverages (SSBs), including soda, increase of fast

food consumption, and decrease of fruit and vegetable intake, are among the most studied reasons of childhood obesity. Research on soda and other SSBs suggests that there is a positive association between SSBs and childhood obesity (Malik, Schulze, & Hu, 2006). A systematic review of 30 studies compiled research available on the relationship between SSBs and childhood obesity from 1966 through 2005. The researchers found that six out of 13 cross-sectional studies looking at this association found a positive significant association between consumption of SSBs and childhood obesity. This study also found that four out of six prospective studies found a positive significant association. The researchers concluded that taken together, the different studies support the link between SSBs and childhood obesity.

More recent studies on the consumption of soda and childhood obesity, particularly among toddlers and preschool-aged children has emerged (DeBoer, Scharf, & Demmer, 2013; Warner, Harley, Bradman, Vargas, & Eskenazi, 2006). A study found that 56% of children consumed soda at the age of 2 years (Warner et al., 2006). Also, it was shown that children who consumed one or more sodas per day were 3.39 times more likely to be obese, compared to those that drank less than one soda per day. A more recent study explored the longitudinal association between SSBs and weight gain among 2-5 year old children. This study did not find an association between SSBs and childhood obesity at age 2, but it did find an association at ages 4 and 5. Furthermore, this study found that children who had one or more SSB per day at age 2 had a greater BMI z-score increase at age 4, compared to those that had less than one drink per day. Overall, although there are some studies that have not found an association between SSBs and childhood obesity (Blum, Jacobsen, & Donnelly, 2005; Newby et al., 2004), the majority of the studies demonstrate that there is an association between regular/daily intake of SSBs and childhood obesity.

Fast food is also a contributor of the increase in caloric intake by providing high energy foods and large portion sizes (Boutelle et al., 2006; Bowman et al., 2004; Harrison & Liechty, 2012). One study using data from 6,212 children ages 4-19 showed a positive association between fast food consumption and energy intake (Bowman et al., 2004). In contrast, another study using data from 902 middle and high school adolescents did not find any associations between fast food and children's weight status or BMI (Boutelle et al., 2006). Experimental studies have also been conducted to determine whether decreasing fast food intake could reduce energy intake (Ebbeling et al., 2007). A study (n = 18) investigating whether smaller portions and lower eating rate of fast food could reduce energy intake did not find significant differences on energy intake across participants from the three different feeding conditions (Ebbeling et al., 2007). Overall, research on fast food consumption and energy intake demonstrates that there is not a clear association and also that there is very little research conducted among preschool-aged children, leaving a gap to fill in.

In contrast to soda and fast food intake, the consumption of fruits and vegetables has been thought to be a protective factor against childhood obesity since they tend to be low in fat but high in water and fiber, making them a healthy food source (Makris & Foster, 2011). Although increase of fruit and vegetable consumption has multiple health benefits and satiation with nutritious foods could reduce intake of low nutrient but high caloric foods, it is not very clear whether these benefits extend to the prevention of childhood obesity; current literature on the topic suggests mixed findings (Ledoux, Hingle, & Baranowski, 2011; Newby, 2009). A systematic review found that among studies conducted on 2-5 year old children, consumption of fruits and vegetables was not associated with changes in BMI, BMI z-scores, or weight (Newby, 2009). The studies found in this review demonstrated inconsistent findings that varied by age and

sex, making it difficult to draw overall conclusions for the protective role of fruits and vegetables on childhood obesity. A more recent systematic review of longitudinal and experimental studies found mixed associations on fruit and vegetable intake (Ledoux et al., 2011).

Thus, the associations between fruit and vegetable consumption and childhood obesity are not well established. It is important to consider that the age groups of the studies that found a negative or mixed association recruited elementary and middle school children. On the other hand, the studies that did not find an association were done with preschool-aged children. It is possible that consequences of dietary intake take time to become evident and for that reason they only become noticeable when children are in elementary school or even later. Moreover, what we can learn from the current literature is that age might be an important factor when examining the role of fruit and vegetable consumption. Finally, it is necessary to investigate the role that fruits and vegetables play in the prevention of childhood obesity.

To summarize, the literature on dietary intake and its association with childhood obesity, there are some groups of foods that are better understood and have more scientific information available regarding their disadvantages or benefits than others. Currently, we know that soda and fast food can be detrimental for children and there is consistent evidence to suggest that they are associated with childhood obesity. However, although it would seem to be intuitive that consumption of fruits and vegetables should be inversely associated with childhood obesity, the few studies conducted on this to date do not portray a clear picture. Thus, more longitudinal and experimental studies looking at preschool-aged children's diet need to be undertaken.

Given that children, particularly preschool-aged children, rely heavily on their parents/caregivers to fulfill their feeding needs, it is important to consider how parents make decisions to feed their children and how depression can affect their decision making. Although

there is very little research investigating the ways in which maternal depression is associated with children's dietary behaviors, a recent study, shed light on this topic (Vericker, 2015). This study analyzed cross-sectional data from the Early Childhood Longitudinal Survey- Birth Cohort (ECLS-B) on 5,100 mother-child dyads. Maternal depressive symptoms were measured using the CES-D when the children were five. To measure dietary intake, the parents reported how often their children had eaten a certain food or group of food over the last 7 days. The results showed that children of mothers with elevated depressive symptoms ate vegetables less often and consumed soda and sweet snacks more often than children of mothers without depressive symptoms. Moreover, it was found that children of mothers with moderate or severe depressive symptoms also consumed less fruits than mothers without symptoms. Finally, this study found that there was no difference between maternal depressive symptoms and children's consumption of fast food (Vericker, 2015). This study is among the first ones to investigate the relationship between maternal depression and dietary intake among children; therefore, it is necessary to continue building on this knowledge to better understand the ways in which maternal depression can be contributing to childhood obesity.

Clan-level factors

The clan level (also known as family level) takes into account characteristics of the parent, caregiver, or family where the child resides. The constructs examined at the clan-level deemed relevant to childhood obesity development are maternal involvement and breastfeeding. From a feminist perspective it is necessary to investigate whether women in a low-income sample initiate breastfeeding at similar rates than women from other socioeconomic status and whether breastfeeding for six months or more can protect their children from developing obesity. These constructs for the clan level were also chosen in order to investigate the physical

(breastfeeding) and emotional (maternal involvement) connection that is created between the mother and the child. Maternal involvement has been used as an indicator of the emotional and relational connection between the mother and the child. Breastfeeding serves not only to provide the best nutrition to the child, but also to promote holding and skin-to-skin contact, and to create a strong bond and connection between the child and the mother. The associations between maternal involvement and breastfeeding will be reviewed below.

Maternal involvement

Maternal involvement refers to the level of affection that a mother expresses and demonstrates to her children and the quality of relationships that develop. Previous studies have demonstrated that the quality of the relationship between mother and child can be associated with childhood obesity (Birch, Marlin, Kramer, & Peyer, 1981; Skouteris et al., 2012; Ventura & Birch, 2008). One of the first studies to examine these associations was conducted by Birch and colleagues (1981). This study suggested that, compared to mothers of normal weight children, mothers of obese children were more likely to interact negatively with their children and to have lower levels of verbal interaction, regardless of the mothers' BMI. More recent studies have suggested a similar association. For example, a study suggested that the quality of maternal-child relationship is quite important for the development of adolescent obesity (Anderson, Gooze, Lemeshow, & Whitaker, 2011). This study investigated the repercussions of early maternal-child relationships and the development of obesity at age 15. The researchers found that the prevalence of obesity was significantly higher for those children who had poor maternal-child relationships during the first 3 years of life (Anderson et al., 2011). Furthermore, it was found that adolescents who had worse maternal-child relationships, had 42% higher odds of being obese at the age of 15, compared to those who had good relationships (Anderson et al., 2011). Also, a systematic

review of five studies found associations between parent-child interactions and childhood obesity (Skouteris et al., 2012). Specifically, this systematic review found that family conflict, poor communication, and low levels of parental intimacy were associated with higher child weight status (Skouteris et al., 2012). The field of study on mother-child relationships and its effects on childhood obesity is still limited and it lacks information on preschool children. Therefore, it is important to assess maternal involvement among preschool-aged children.

Given that infants tend to spend the majority of the time with their mothers, they usually develop very strong bonds (Milkie, Nomaguchi, & Denny, 2015). However, when mothers are depressed, some difficulties in establishing and maintaining a strong relationship with the child might appear (Beebe et al., 2010). In fact, it was well-established three decades ago that mothers with depression might be more withdrawn and might not be emotionally available, making them less sensitive to their children's emotions and behaviors and thus hampering the mother-child relationship (Cohn & Tronick, 1983). A study investigated the longitudinal association between maternal depression and mother-child bonding (Moehler, Brunner, Wiebel, Reck, & Resch, 2006). It was found that mothers with depression, particularly postpartum depression, had more negative bonding patterns with their children. Moreover, it was found that mother-child negative bonding started as early as when the child was two weeks old and it was still present when the child was 14 months old. Although examining mother-child bonding does not equal maternal involvement, it is related since maternal involvement is the degree to which mothers express and demonstrate affection towards their children. Overall, the current information available on maternal depression and the relationships that are developed between mother and child suggest that maternal depression can disrupt the creation of a strong and safe bond. This indicates that it is important to better understand the role that maternal involvement might play in the

development of childhood obesity.

Maternal involvement could be considered as a measure of attachment, which is also promoted through breastfeeding because of the increased contact between the mother and the child (Jackson, 2016). However, maternal depression can influence breastfeeding initiation and duration (Hahn-Holbrook, Haselton, Schetter, & Glynn, 2013), which in turn may influence childhood obesity development (Grigoriadis et al., 2013). The next section describes the literature available on breastfeeding, maternal depression, and childhood obesity.

Breastfeeding

One way in which maternal depression can influence the development of childhood obesity is by decreasing healthy behaviors such as breastfeeding, which has been shown to be associated with a decreased risk of childhood obesity (Arenz et al., 2004; Gibbs & Forste, 2014; Yan, Liu, Zhu, Huang, & Wang, 2014). A systematic review aiming at summarizing the literature on postpartum depression and infant-feeding outcomes found that lower levels of depressive symptoms were associated with higher rates of breastfeeding (Dennis & McQueen, 2009). Moreover, it was found that mothers with elevated symptoms of depression were more likely to stop breastfeeding earlier than mothers without depressive symptomology. Another systematic review and meta-analysis reviewed information from 30 studies on the consequences of antenatal depression on perinatal outcomes and the pooled results found that maternal depression was associated with lower breastfeeding initiation (Grigoriadis et al., 2013).

The current literature available on maternal depression and breastfeeding suggests that there is an association; however, the directionality of it is still not clear. Recent research actually suggests that there is a bidirectional relationship, indicating that mothers who are depressed are less likely to breastfeed, which in turn might exacerbate depressive symptomology since the

mother may feel less competent. For example, a study demonstrated that women who had elevated symptoms of depression during the antenatal period ceased breastfeeding about 2.3 months earlier than women who did not have depressive symptoms (Hahn-Holbrook et al., 2013). Moreover, it was found that women who had elevated depressive symptomology during the antenatal period had lower rates of breastfeeding at 3 months but not at any other point beyond that. This study also demonstrated that women who breastfed with more frequency at 3 months had lower levels of depressive symptoms in subsequent time points (Hahn-Holbrook et al., 2013). Overall, there is support for the association between maternal depressive symptoms and shorter duration of breastfeeding, and this relationship may be bidirectional. Breastfeeding may be a factor in both maternal depression and child overweight and obesity, and it should be taken into account when studying these phenomena.

One of the main limitations of the current literature on breastfeeding and maternal depression or child obesity is the lack of inclusion of minority women, particularly Black women. Past research has demonstrated that Black women have lower rates of breastfeeding compared to the rates of other groups (CDC, 2010a). Moreover, depression rates among minorities have been found to be higher than for non-minority women (Taveras et al., 2010; Williams et al., 2007). Thus, it is necessary to investigate the association of depressive symptomology and breastfeeding using samples that particularly include and have a significant proportion of Black women and mothers from a wide range of socioeconomic status.

As mentioned earlier, breastfeeding has also been found to be associated with childhood obesity (Arenz et al., 2004; Gibbs & Forste, 2014; Yan et al., 2014). The most comprehensive and compelling evidence of the association between breastfeeding and childhood overweight and obesity has been provided in a recent meta-analysis of 25 studies encompassing findings from 12

different countries and an aggregate sample of 226,508 participants (Yan et al., 2014). The pooled odds ratios showed that breastfeeding was associated with lower levels of childhood obesity. In regards to breastfeeding duration, it was shown that there was a decrease in childhood obesity risk as breastfeeding duration increased. Particularly, it was shown that children who were breastfed for 7 or more months were significantly less likely to be obese (Yan et al., 2014). The literature on the association between breastfeeding and childhood overweight and obesity is compelling and demonstrates that children who are not breastfed are at a higher risk of childhood obesity. Therefore, breastfeeding is a highly important factor to take into account when investigating maternal depression and childhood overweight and obesity.

The current literature available on maternal depression, childhood obesity, and breastfeeding suggests independent associations between maternal depression and breastfeeding and between breastfeeding and childhood obesity. However, there is a lack of information available on the effect that breastfeeding has on child growth trajectories. The question on breastfeeding and childhood obesity development still remains: Does breastfeeding for less than six months reduces the risk of developing at-risk trajectories? This study aims to address this question by exploring whether breastfeeding duration is associated with the development of risk trajectories. Similarly, many questions remain regarding maternal involvement since the literature on this construct is inconclusive. It is necessary to determine whether more maternal involvement early on in life can prevent children from developing obesity.

Constructs at the child and clan levels have been identified as potential risk factors for childhood obesity development. Yet another factor that has been studied consistently is the role that race/ethnicity play in childhood obesity development. One of the most contended issues is whether race is truly the driving factor or whether socioeconomic status plays a stronger role in

childhood obesity development. Because these two constructs are highly intertwined with each other, it is difficult to identify the effect that race/ethnicity alone have on childhood obesity. The next section provides an overview of the literature on childhood obesity development and its association with race/ethnicity.

Race/ethnicity

The relationship between race and childhood obesity is currently inconclusive. Although many studies have shown that childhood obesity disproportionately affects Black and Latino/a children (Ogden, Carroll, Kit, & Flegal, 2012; Ogden et al., 2014; Taveras et al., 2010), other studies have shown that it is socioeconomic status and not race/ethnicity that is driving these disparities (Rogers et al., 2015). For example, a study used nationally representative data and showed that Latino/a and Black children had higher rates of childhood obesity compared to White and Asian children (Ogden et al., 2014). In fact, the prevalence among Latino/a children (38.9%) was almost twice as much as that of their Asian counterparts (Ogden et al., 2014). A smaller study using data on children from Massachusetts (n = 1,826) demonstrated that the prevalence of childhood obesity among Black and Latino children at age 3 was almost twice that of White children. Even after controlling for socioeconomic status, these racial disparities were attenuated but still present (Taveras et al., 2010). On the other hand, a recent study using data from 111,799 children and adolescents from Massachusetts showed that income but not race was significantly associated with obesity (Rogers et al., 2015). These studies demonstrate that there are intricate relationships between race, income, and childhood obesity, which might be difficult to tease apart.

One potential explanation- but often ignored in population studies- for why race/ethnicity can be a risk factor for childhood obesity is the higher prevalence of risk factors among minority

children (Taveras et al., 2010; Taveras, Gillman, Kleinman, Rich-Edwards, & Rifas-Shiman, 2013). These include modifiable factors such as lack of breastfeeding, breastfeeding for shorter duration, and early introduction of solids, which differ by racial/ethnic group, and if addressed early enough could reduce health disparities (Taveras et al. 2010, 2013). There are also risk factors for obesity that could be prevented early on such as inadequate sleep, having a television in the bedroom where the child sleeps, and high intake of fast food and sugary drinks, all of which are more common among Latino and Black families (Taveras et al. 2010, 2013). Overall, although some studies have shown that racial inequalities are a risk for childhood obesity; other studies suggest that income inequality is the driving factor. Therefore, it is necessary to identify data sets and design studies that attempt to untangle the contribution of race and income and also to identify modifiable risk factors by racial/ethnic group that can be prevented when addressed early on. In order to achieve this it is important to identify the role of background factors on maternal depression and childhood obesity.

Background factors

Child age

Childhood obesity also differs significantly by age (Ogden et al., 2014; Wang, 2011). A study using nationally representative data (Ogden et al., 2014) showed that 22.8% of children between the ages of 2-5 were overweight or obese, compared to 34.2% and 34.5% of children between the ages of 6-11 and 12-19, respectively. When taking into account race, the prevalence of obesity among Black adolescents ages 12-19 was 39.8%, almost double the prevalence among Black 2-5 year old children (21.9%). In contrast, among Latino children, the prevalence of obesity is already quite high among 2-5 year old children (29.8%), but it is still lower than among Latino adolescents between the ages of 12-19 (38.1%) (Ogden et al., 2014). Other studies

have demonstrated similar results (Wang, 2011). A nationally representative longitudinal study demonstrated that even with the increase in childhood obesity trends, the rate of obesity among children ages 2-5 has been consistently lower by about 10 percentage points compared to older children and adolescents (Wang, 2011). This information suggests that it takes time for obesity risk to manifest and it is necessary to take age into account when investigating childhood obesity.

Child sex

Height and weight standards differ by sex, for that reason, there are different growth charts for males and females (Ogden et al., 2014). However, it is unclear whether differences in childhood overweight and obesity rates by sex are due to biological sex differences or due to differences in behaviors and environment (Sweeting, 2008; Wisniewski & Chernausek, 2009). One of the biological differences is that males and females store fat differently; particularly during puberty, leading to differences in adiposity (Wisniewski & Chernausek, 2009). Another difference may be due to feeding attitudes by family members, which tend to be more restrictive for females than for males, and in turn these practices can lead to overeating, increasing the risk for overweight and obesity (Birch, Fisher, & Davison, 2003; Faith et al., 2004). Other behaviors that differ by sex and are associated with childhood obesity include physical activity and sedentary behaviors (Sweeting, 2008). Some studies have shown that males engage in physical activity more frequently compared to females, but they also spend more hours on sedentary behaviors such as watching television and playing video games (Beighle, Morgan, Le Masurier, & Pangrazi, 2006; Matthews et al., 2008; Ridgers, Salmon, Parrish, Stanley, & Okely, 2012). Some of these behaviors are ingrained in societal expectations of gendered attitudes; which allows males to do sports that include more aggression and competition, and it encourages women to do sports that require cooperation (Roth & Basow, 2004). Overall, sex can influence

the development of childhood obesity through different mechanisms; thus, it is an important background factor to consider when studying overweight and obesity outcomes.

Maternal education

Maternal education is another risk factor that contributes to the development of childhood obesity. Typically, children from low-income and low-education households tend to have rates of obesity that are twice the rate of obesity among children from more educated or wealthier households (Singh, Siahpush, & Kogan, 2010). A study conducted using nationally representative data from the National Survey of Children's Health (n = 46,707), found that children of parents with less than a high school diploma had 2.2 higher odds of being obese, compared to children with parents with a college education (Singh, Kogan, Van Dyck, & Siahpush, 2008). A more recent study demonstrated that the prevalence of childhood obesity among children of parents without a high school diploma was 3.1 times higher than for children with parents who had graduated from college (Singh et al., 2010). Household education, particularly maternal education, is another characteristic that can have a noteworthy impact on childhood obesity disparities, and thus needs to be taken into account when studying obesity.

Household income

Health inequality, particularly related to childhood obesity, has increased over the years (Pan, Blanck, Sherry, Dalenius, & Grummer-Strawn, 2012; Singh et al., 2010; Wang & Zhang, 2006). Different studies have shown that children from low socioeconomic backgrounds are at a higher risk for childhood obesity. For example, a study using nationally representative data showed that children of parents who were under the poverty line were 2.8 times more likely to be obese (Singh, Kogan, et al., 2008). An extension of this work demonstrated that between 2003 and 2007, childhood obesity increased by about 10% for all children (Singh et al., 2010).

However, when the analysis was broken down, the prevalence of obesity among children from low socioeconomic status increased by 27%. Moreover, it was shown that the repercussion of low income status was greater in 2007 than in 2003; children who were under the poverty line in 2007 had 243% higher odds of obesity versus 182% increased odds in 2003, compared to children from middle income families (Singh et al., 2010). This demonstrated that children from low socioeconomic status are at a greater disadvantage than children from middle and higher income status. Moreover, these studies show that health disparities are only widening and children from low socioeconomic families are at a higher risk for childhood overweight and obesity.

Maternal pre-pregnancy BMI

Prenatal visits offer a timely opportunity for healthcare providers to discuss with mothers-to-be the importance of diet, nutrition, and appropriate weight gain during pregnancy in order to avoid complications for the mother and the offspring (Whitaker et al., 2010; Whitaker, 2004). One of the main consequences of pre-pregnant maternal obesity for the offspring is the development of childhood obesity (Olson et al., 2009; Whitaker et al., 2010; Whitaker, 2004). A study that aimed to determine the role of maternal obesity on childhood found that the prevalence of obesity among children of mothers who were overweight or obese during the first trimester of pregnancy was between 2.4 and 4 times higher than it was for children of mothers who were within the normal weight range (Whitaker, 2004). Moreover, it was found that the prevalence of childhood obesity increased significantly as maternal BMI and child age increased. For instance, for mothers with a BMI ≥ 40 during the first trimester, their children at age 4 were 4.31 times more likely to be obese than children of mothers who were within normal weight range (Whitaker, 2004).

Another study found that children of mothers who were overweight and obese at the beginning of the pregnancy were more likely to be overweight or obese at age 3, compared to children of mothers who were of normal weight (Olson et al., 2009). Another study using data on 3,022 children from the National Longitudinal Survey of Youth's Child-Mother showed similar findings (Salsberry & Reagan, 2005). This study found that children of mothers who had a pre-pregnancy BMI ≥ 30 had children who were 1.37 times more likely to be obese at age 3 compared to children whose mothers' pre-pregnancy weight was within the normal range (Salsberry & Reagan, 2005). As children's age increased, the odds of childhood obesity did as well. In fact, the odds of childhood overweight and obesity by age 7 increased to 2.11 and 2.91, respectively (Salsberry & Reagan, 2005). Overall, the literature shows that higher maternal pre-pregnancy weight over the first trimester plays a significant role in the development of overweight and obesity during infancy and childhood, therefore, it is necessary to control for it whenever possible.

Proposed Study to Address Current Research Gaps

The current literature on the association between maternal depression and childhood obesity in the US is scarce and it offers mixed findings mostly because this field is still emerging. Moreover, there are few studies investigating differences in childhood obesity trends by race/ethnicity and the risk and protective factors for different groups. Therefore, in light of this review of putative risk and protective factors for childhood obesity, this study will address the following gaps.

First, previous studies have predominantly investigated maternal depression at one point in time and its association with childhood obesity (Duarte et al., 2012; Gross et al., 2013; Surkan et al., 2014); however, it is important to take into consideration the recurrence of depression to

better understand the effect that it can have on children's health. For instance, are children exposed to mothers with recurrent depressive symptoms during the first 3 years of life more likely to develop childhood overweight and obesity than those exposed only at one time point? Studying depression at two time points makes it possible to examine the cumulative effect of maternal depression on the development of childhood overweight and obesity.

Second, in order to truly understand the potential association between maternal depression and childhood obesity, it is necessary to investigate maternal depression's longitudinal effect on childhood obesity. This can be examined by identifying child growth trajectories. For example, techniques such as group-based trajectory modeling can be used to identify high and low-risk growth patterns among children. Then, risk factors at the child and clan levels for belonging to unhealthy growth trajectories can be identified.

Third, the literature on racial/ethnic disparities in childhood obesity remains inconclusive. Thus, it is necessary to continue investigating how child growth patterns might differ by race/ethnicity and also to understand what are the risk and protective factors at the child and clan levels that can impact these growth patterns.

Finally, from a theoretical point of view, many studies argue that childhood obesity is multi-determined and so should be understood from a socio-ecological perspective by investigating contributors to childhood obesity at different levels. However, as mentioned earlier, the few studies that have focused on investigating mediating factors between maternal depression and childhood obesity have focused almost exclusively on the child level, leaving aside potentially influential factors at all other levels. For instance, other levels such as the clan level should also be investigated as potential risk factors for childhood overweight and obesity. Therefore, in order to fill in this gap, studies are needed that investigate potential risk factors at

the child and clan levels. This would expand the current knowledge on how different factors at different levels can influence the development of childhood obesity and it lays the ground for future interventions that can help prevent the development of childhood obesity early on.

Research Questions and Hypotheses

The proposed study addresses the gaps outlined above by employing national longitudinal data, group-based trajectory modeling, and multi-level socioecological factors by race/ethnicity to identify child growth patterns over time. This study examines seven research questions and seven hypotheses. The research questions and hypotheses are listed in Table 2.1.

It is expected that the proposed study elucidates the association between recurrent maternal depression and childhood growth over time. Finally, it is expected that findings from the proposed study identify early risk factors for high risk trajectories that can become targets of intervention to prevent the development of childhood obesity.

Table 2.1: Research Questions and Hypotheses

Research Question	Hypotheses
1. Is maternal depression over the first three years of the child's life associated with childhood BMI z-score and with overweight (OW) and obesity (OB) status?	1a. Maternal depression over the first three years of the child's life is associated with higher BMI z-score when the child is 9 years, compared to children of mothers with no maternal depression. 1b. Maternal depression over the first three years of the child's life is associated with overweight (OW) and obesity (OB) status when the child is 9 years, compared to children of mothers with no maternal depression.
2. Do patterns of child growth follow a single or multiple trajectories?	2. Child growth patterns will follow multiple trajectories.
3. Is maternal depression over the first three years of the child's life associated with growth trajectories from birth to age 9?	3. Maternal depression over the first three years of the child's life will be associated with at-risk growth trajectory development.
4. Are child-level factors (sleep, television viewing, outdoor play, and dietary intake) associated with child growth trajectories from birth to age 9?	4. Fewer hours of sleep and outdoor play and less fruit and vegetable consumption; and more television viewing, fast food, and soda intake will be associated with at-risk growth trajectory development.
5. Are clan-level factors (maternal involvement and breastfeeding duration) associated with child growth trajectories from birth to age 9?	5. Lower levels of maternal involvement and breastfeeding for less than 6 months will be associated with at-risk growth trajectory development.
6. Are there different growth trajectories by race/ethnicity?	6. Trajectories of growth will differ by race/ethnicity.
7. What are the common and unique risk factors that predict at-risk growth among children from different racial/ethnic groups?	7. Significant predictors of the most at-risk trajectory will differ by race/ethnicity.

Note. OW: overweight; OB: obesity.

CHAPTER 3: METHODS

This chapter provides information about the development of this study. Specifically, the section below provides information on the data, sample, measures, and statistical analysis that was used to conduct this study. This chapter also provides a summary of the hypotheses and statistical methodology used to test them, and it provides the conceptual models that were tested.

Data

In order to answer the aforementioned research questions, this study used secondary data from Waves 1-5 from the Fragile Families Child Wellbeing Study (FFCWS). FFCWS is a national prospective study of non-marital births in the United States. Data were first collected when the children were born (wave 1), at one (wave 2), three (wave 3), five (wave 4), nine (wave 5), and fifteen years (wave 6); however, this study used data from birth through nine years of age (see table 3.2 for information about variables and wave of assessment). The first wave of interviews was collected at the hospital from February 1998 to September 2000 on 4898 mother-children dyads. Wave 2 follow-up interviews with the mothers and fathers were collected from 1999 to 2002, wave 3 interviews from 2001 to 2003, wave 4 interviews were conducted from 2003 to 2006, wave 5 interviews from 2007 to 2010, and wave 6 interviews started in 2015 and are still being conducted. Waves 5 and 6 included interviews from children and teachers in addition to the parents' interviews. In order to obtain more information about this dataset please see: http://www.fragilefamilies.princeton.edu/documentation/general

FFCWS used a stratified three-stage random sample design of cities that had a population of at least 200,000 people (Reichman et al., 2001). All cities in the United States that qualified based on population size were stratified based on labor market conditions, welfare generosity, and child support system rigor. Cities with extreme values on all three conditions and cities with

at least one middle value were placed in different groups for potential inclusion. Cities with combinations of extreme and middle values were randomly chosen, leaving an initial total of 16 cities. The cities that had extreme conditions were labeled “large sample cities” since 325 births were sampled there, and those that did not have extreme conditions were labeled “small sample cities”, since only 100 births were sampled from these cities (Reichman et al., 2001). Other five cities were purposefully selected; four of them were of high interest to funders and one of them was a leading city in welfare reforms. All five of these cities were “large sample cities”, leaving a total of 21 cities in the final sample. Two cities that had initially been randomly chosen had to be switched due to denied access to the selected hospitals. Two new cities with similar characteristics were chosen. After reviewing the new conditions of the cities, three cities were changed from “large sample” to “small sample” and vice versa (Reichman et al., 2001).

Once the cities were chosen, the hospitals were selected. In five cities, there were 5 birthing hospitals at most, so all the hospitals were included to conduct interviews. For the rest of the cities, a list of hospitals with the number of non-marital births was ranked from highest to lowest (Reichman et al., 2001). Then the hospitals were sampled in the order from highest to lowest until 75% of non-marital births in a particular city were covered. This strategy was used in all but two cities, Chicago and New York, where random sampling of hospitals with more than 1000 non-marital births per year was used (Reichman et al., 2001).

Once the hospitals had been selected, the participants were chosen. At each hospital, the researchers obtained information regarding the percentage of non-marital births in the hospital’s city during 1996 or 1997, and then married and unmarried births were randomly chosen until reaching said quotas. For the most part, the sample of non-marital births selected was representative of each of the 21 cities (Reichman et al., 2001). Participants were not included in

the sample if: 1) the parents were planning to put the child up for adoption, 2) the father of the baby was deceased at the time of birth, 3) there was not sufficient mastery of English or Spanish to complete the interview, 4) the mother or baby was too ill, or 5) the baby was deceased before the interview was carried out.

The majority of the Fragile Families data files are publicly available; however, census tract data, neighborhood related information, and medical records are only available through contractual agreement (Reichman et al., 2001). This study was based on both public and contractual data. A contractual data protocol for use of sensitive information was approved by the University of Illinois Institutional Review Board (Appendix A). The protocol and IRB approval were then submitted and approved by the FFCWS research team. The FFCWS is funded by the Eunice Kennedy Shriver National Institute of Child Health and Human Development (NICHD) of the National Institutes of Health and by other private foundations. FFCWS is one of the few datasets that has recent longitudinal data on parents and children from birth. Moreover, this dataset has information on mental health and health characteristics, making it one of the most comprehensive longitudinal studies of maternal and child health in the United States.

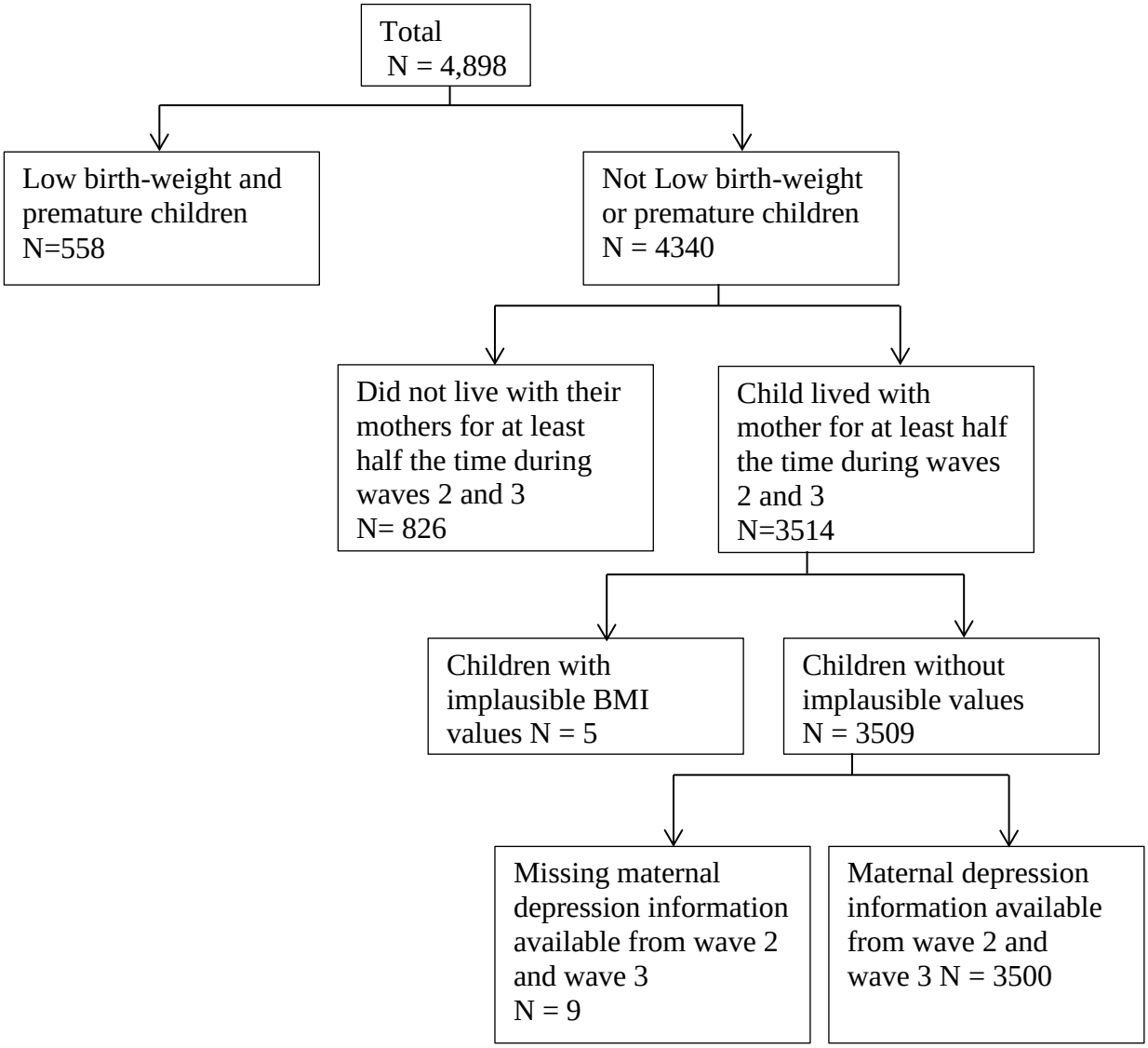
As mentioned earlier, there are a total of 6 waves spanning from wave 1, birth, to wave 6, age 15. This study will only use data from wave 1 through wave 5. The variables used in this study came from the core and in-home interviews.

Analytic Sample

The total number of participants in the study at baseline was 4,898. The sample included predominantly non-marital, low-income mothers and one focal child. The analytic sample excluded children whose weight at birth was low (less than 2,500 grams) and who were

premature. These children were taken out of the sample because they have different growth patterns than their normal weight counterparts. The second criteria of the children included in the analytic sample was that they had to reside with their mothers at least “half of the time” during waves 2 and 3, which were the times when maternal depression information was used. This criterion was necessary given that I am particularly interested in the effects that maternal bonding, or lack thereof, can have on the development of childhood obesity. If a child lives with a mother less than half of the time, then there could be a confounding factor because I would be unable to tease out the influence that the mother-child bond has on the child over time. Also, the analytic sample included children with plausible BMI values at waves 1, 3, 4 and 5. If the BMI z-score was below -5 or above 5 at baseline (wave 1), the value was deemed implausible based on recommendations set by the World Health Organization ([WHO], 2006). For waves 3 and 4, if children had missing or implausible information for height or weight, their BMI z-score would not be calculated. For wave 5, even if children had implausible values for weight or height, their BMI z-scores were calculated but flagged; only two values below -5 were flagged for wave 5. As part of the analytic sample all implausible BMI z-score values were taken out. Figure 3.1 shows the inclusion and exclusion criteria of the analytic sample.

Figure 3.1. Analytic sample (N = 3500)



The final analytic sample included information on 3500 mothers and children. Table 3.1 shows the characteristics of the full sample at baseline. Medical records were only available on 75% of the participants, since the rest could not be accessed because the hospitals or participants did not give permission. Regarding the characteristics of the mothers, the majority of participants were Black women, followed by Latina women and the majority had less than high school education. The average age of the mothers was 25 years and the pre-pregnancy BMI was 26.

Table 3.1: Descriptive Characteristics at Baseline of the Analytic Sample, Fragile Families Data (N = 3500)

Variables	n	%	*M*	SD	Min- Max	Missing n (%)
Child sex (from W1)	3500					0
Female	1669	47.69				
Male	1831	52.31				
Child BMI z-score	2469		-0.25	1.1	-4.78 - 6.68	1031 (29.46)
Maternal race	3491					9
White	797	22.83				
Black	1626	46.58				
Latino	938	26.87				
Other	130	3.72				
Maternal education	3496					4
High school or less	2192	62.7				
Some college or more	1304	37.3				
Federal poverty level	3500					0
0-99%	1200	34.29				
100-199%	913	26.09				
200% - 299%	541	15.46				
300% or more	846	24.17				
Maternal relationship with baby's father	3500					0
Married	915	26.14				
Cohabiting	1262	36.06				
Other[1]	1323	37.80				
Maternal age	3500		25.13	5.98	15-43	0
Number of biological children	3484		1.09	1.27	0-10	16 (0.46)
Pre-pregnancy BMI	2138		26.43	6.66	14.76-65.84	1362 (38.91)

Note. W1: wave 1 (baseline). BMI: body mass index.

[1] Mothers whose relationship with the baby's father was other than married or cohabiting were included in this category. Thus, this category was made up of fathers whose relationship with the mother was: visiting, friends, hardly talking, or never talking.

Measures

The variables and constructs of interest included in this study were: childhood overweight and obesity status, maternal depression, sleep, television viewing, outdoor play, and dietary intake, maternal involvement and breastfeeding. Demographic variables from mother and child included: child age, child sex, maternal race/ethnicity, age, education, poverty level, relationship with baby's father, number of biological children, and pre-pregnancy BMI. The operationalization of these variables is explained below.

Childhood overweight/obesity

Commonly, childhood overweight is defined as children and adolescents between the ages of 2 and 19 with a body mass index (BMI) equal or over the 85th percentile for sex and age, and obesity is defined as BMI equal or over the 95th percentile based on the CDC sex-and-age specific growth charts (Ogden et al., 2014). BMI percentiles are the most common method to investigate childhood overweight and obesity because of their easy interpretability. However, for this study I decided that child BMI z-score was the best option given that it is a standardized way of measuring child growth and it can be used to observe child growth over time (Reilly et al., 2011; Wang & Chen, 2012). Although BMI percentiles are easier to interpret and explain, they are not a good measure to be used over time because they have ceiling effects that prevent researchers from fully detecting differences in child's growth over time (Wang & Chen, 2012). BMI z-scores also offer cut-offs to identify overweight and obesity status (Wang & Chen, 2012). The BMI z-score cut-off for overweight status is +1.04 and for obesity status is +1.64 (Wang & Chen, 2012). These BMI z-score cut-offs are equivalent to the 85th and 95th percentiles (Wang & Chen, 2012). These BMI z-score cut-offs have been validated and previously used before on empirical studies (Reilly et al., 2011; Spruyt, Molfese, & Gozal, 2011).

BMI z-scores were provided as a constructed variable and were calculated using the child's weight, height (kg/m^2), sex, and age using the CDC growth curve charts. Child's anthropometric information was collected during the in-home visits by trained interviewers. Anthropometric information was measured at waves 3 through 5 on the child. First, the interviewers attempted to weigh the child by himself/herself; however, if it was not possible, the interviewers would weigh the mother, followed by weighing the mother and child together, and subtracting the difference to obtain child's weight. Participants were instructed to remove their shoes prior to obtaining height and weight. The instruments used were: SECA 840 Bella Digital Scales to measure weight and SECA 214 Road Rod Stadiometer to measure height. A constructed variable from FFCWS for child's BMI z-score is available. This constructed variable was built using the CDC 2000 growth curves for each wave where BMI is available. BMI z-scores were used as a continuous variable and also as a categorical variable (weight status categories) depending on the research question.

BMI z-scores were not assessed by the research team at wave 1. However, information regarding weight and length/height was obtained from medical records and thus available. Child BMI z-scores were constructed using a macro in SAS 9.4 available from the World Health Organization (for more information, see: https://www.cdc.gov/nccdphp/dnpao/growthcharts/resources/sas-who.htm). This macro allows researchers to calculate different anthropometric measurements, including BMI z-score for children under the ages of 2 using the WHO growth charts (CDC, 2017). The WHO growth charts were used at wave 1 rather than the CDC growth charts since the CDC recommends that among children under the age of 2 WHO growth charts be used because they provide a better estimate (CDC, 2010b).

One aspect of particular attention about children's anthropometric measurements is that although they were available at waves 3 through 5, the sample sizes were different for each wave and they were larger for wave 5, contrary to what would be expected due to attrition. The case for the differences in sample sizes across waves had to do with funding and implementation. For waves 3 and 4, the in-home visits were *scheduled* and completed *after* the parent phone interviews, decreasing participation. On the other hand, during wave 5, the parents were consented for and *scheduled* an in-home interview *during* the phone interview (no time elapsed between the phone interview and scheduling the home visit), which increased the number of in-home visits.

Maternal depression

Maternal depression was assessed using the Composite International Diagnostic Interview- Short Form (CIDI-SF). The CIDI-SF is a standardized instrument developed from the larger CIDI, which uses questions that are consistent with the Diagnostic and Statistical Manual of Mental Disorders- Fourth Edition (Association, 2013)**.** The CIDI-SF provides the probability of a respondent being a "case" (i.e. having major depression) if the full CIDI would have been administered. Therefore, the CIDI-SF provides a categorical assessment of probable major depression ("caseness" coded as 0, 1) rather than level of depressive symptoms. Symptoms are assessed via the protocol described below. Overall, a cut point of three or more was coded as a "probable depression" case, as suggested by Fragile Families protocol and by Kessler and colleagues (Kessler, Andrews, Mroczek, Ustun, & Wittchen, 1998). Table 3.2 shows the waves at which depression was assessed.

The CIDI was administered as follows: there were 10 total possible items in the CIDI-SF. The first step consisted on assessing the participants for feelings of dysphoria or anhedonia. The

question to assess feelings of dysphoria was: “During the past twelve months, has there ever been a time when you felt sad, blue, or depressed for two or more weeks in a row?”. If the participant answered no to feelings of dysphoria, then a question to assess feelings of anhedonia was asked: “During the past twelve months, has there ever been a time lasting two weeks or more when you lost interest in most things like hobbies, work, or activities that usually give you pleasure?” If the participant answered yes to either of these questions, then daily reoccurrence and severity were assessed by asking how many days over the past two weeks (i.e. every day) and for how long (i.e. all day long) the person had those feelings. If the respondent answered yes to the dysphoria or anhedonia questions and in addition mentioned that these feelings had taken place almost every day and lasted at least half of the day, then another seven questions were asked. These follow-up questions included: losing interest, feeling tired, change in weight (at least 10 pounds), trouble sleeping, trouble concentrating, feeling down, and having thoughts about death. If the participants said no to the dysphoria or anhedonia questions, then the question would be coded as not having depression (depression = 0). On the other hand, if the respondent answered positively to the anhedonia or the dysphoria questions and to some of the seven follow-up questions, a sum was generated and a score from 0 to 8 was created. Per the literature, scores of 3 and above were coded as probable depression (depression = 1) (Kessler et al., 1998). In the case that a participant was taking anti-depressant medication, no more questions were asked but the person received a positive score for probable depression (depression = 1). It is important to bear in mind that the CIDI-SF provides the *probability* of being diagnosed with major depression, but it requires a confirmatory clinical follow-up interview to confirm the diagnosis (Kessler, 2007).

Recurrent maternal depression

Given that FFCWS is a longitudinal study, it allows for maternal depression to be assessed at different waves. A categorical measure was developed using depression information from waves 2 and 3. Those without depression at any wave were coded as depression = 0, those with depression at *any* wave were coded as depression = 1, and those with depression at *both* waves were coded as depression = 2 to indicate recurrent depression.

Child-level variables

Sleep

Child sleep duration was assessed with a single item during the in-home interview at wave 4 through maternal report. The mothers were asked: "How many hours of sleep does [child] usually get"? The mothers reported the number of hours the child slept in whole numbers. This variable was categorized using the recommendation from the National Sleep Foundation where preschool-aged children should sleep between 10 and 13 hours (Hirshkowitz et al., 2015). Thus, sleep was categorized as those children having less than 10 hours and those having 10 hours or more.

Television viewing

Television viewing among children was gathered during the in-home interview through maternal report at waves 3 and 4. A separate question was asked for weekdays and for weekends. The question about weekday television viewing was: "Think for a moment about a typical **weekday** for your family, including daytime and evening hours. How much time would you say [child] spends watching television or watching videos on TV, either in your home or somewhere else?" The question was then repeated using "a typical **weekend day** (Saturday or Sunday)." Weekday and weekend television viewing were combined through a weighted average by

multiplying the number of hours of television viewing during the weekday by five and during the weekend by two and then dividing the total by seven. A categorical variable was then developed based on the previous recommendations from the American Academy of Pediatrics that children between the ages of 2-5 should not watch more than two hours of television per day ("American Academy of Pediatrics: Children, adolescents, and television," 2001; "Media and Young Minds," 2016). Thus, television viewing was categorized into children who watched less than two hours of television per day and children who watched two hours or more per day. Given that information on television viewing was gathered at waves 3 and 4, I obtained an average of television viewing and then I created a categorical variable for those children who watched television for two hours or more per day (coded as 1) and those children who watched television for less than two hours (coded as 0).

Outdoor play

Information on outdoor play among children was gathered during the in-home interview through maternal report at waves 3 and 4. This question was also asked separately for weekdays and weekends. The questions asked to determine the number of hours the child played outside were: “Think for a moment about a typical **weekday** for your family. How much time would you say [child] spends playing outdoors, either at your home or somewhere else?”. The question was then repeated but using “a typical **weekend day** (Saturday or Sunday)”. Weekday and weekend outdoor play were combined through a weighted average by multiplying the number of hours of outdoor play during the weekday by five and during the weekend by two and then dividing the total by seven. This variable was used as a continuous variable. Given that information on outdoor play was gathered at waves 3 and 4, I obtained and used the average for analysis.

Dietary intake

Dietary intake refers to the diet that the child had at wave 4 and it encompassed three different questions/variables. The overall aim of this construct was to quantify foods the child typically ate that were "healthful" or high in nutrition but low in calories (fruits and vegetables) and those that were "unhealthful" or low in nutrition but high in calories (soda and fast food). To assess dietary intake mothers were asked: "On a typical day, about how many servings of the following foods does [child] eat?" for each of the following foods: soda, fresh fruits or vegetables, and frozen or canned vegetables. Responses were presented in a Likert scale from 0 to 5 with "0" meaning none and "5" meaning having five or more servings per day. Since there are two questions for fruit and vegetable consumption, these two questions were combined to yield servings/day. Information on fast food consumption was obtained asking: "About how many times a week does [child] eat a meal from a "fast food" restaurant (e.g., McDonald's, KFC, etc.)?". This question was asked per *week* rather than per day. This study used dietary intake data from wave 4 since this is the first wave where this information is available. Dietary intake variables were used as continuous.

Clan-level variables

Maternal involvement

Maternal involvement was assessed at waves 3 and 4, but the questions differed somewhat from wave to wave in order to reflect the child's developmental stage. Maternal involvement was assessed by asking: "Please tell me how many days you do **each** of these activities in a typical week?". In wave 3 the mother was asked to report the number of days/week she engaged with her child in each of 13 activities such as: sing songs, hug child, tell him/her that you love him/her, let the child help with chores, play imaginary games, read stories, tell

stories, play inside with toys, tell the child that you appreciate something s/he did, take him to visit relatives, go out to eat, assist with eating, and put the child to bed. At wave 4, mothers reported the number of days/week they engaged with their child in the following eight activities: singing songs, reading stories, telling stories, playing inside with toys, telling child that you appreciated something s/he did, playing outside, going out, and watching television together. Although this information was available at waves 3 and 4, I only used information from wave 3 given that I wanted to understand the effect of maternal involvement early in life and around the same time as maternal depression was being measured. Since maternal depression was assessed at wave 3 and maternal involvement was measured at the same time, I decided to only use maternal involvement from wave 3. Scale analysis was conducted to obtain Cronbach's alpha internal reliability scores for the maternal involvement scale. Then, in order to reduce the number of items that were included from, a principal components analysis (PCA) was performed to determine the underlying structure of this variable (PCA procedures are described in detail under the "Principal Components Analysis" section).

Breastfeeding

Breastfeeding was measured at wave 2 by asking: "Did you ever breastfeed [child]? and "How old was [child] when you stopped breastfeeding (him/her)"?. The first question was used to assess breastfeeding initiation, and was coded as 0 or 1. The second question was used to assess breastfeeding duration and was dichotomized into less than 6 months or 6 months or more in order to follow the recommendation by the American Academy of Pediatrics (Eidelman et al., 2012).

Control variables

Some demographic variables, along with variables that have been shown to be associated

with childhood obesity in the literature, were added as controls. These variables include maternal age, maternal education, pre-pregnancy BMI, poverty level, race/ethnicity, and child sex and age. Maternal age was obtained through self-report at the time of the birth of the child (wave 1). Maternal education and poverty level were gathered at wave 1. Maternal education was gathered asking the mother: "What is the highest grade or year of regular school that you have completed"?. This variable was used as a categorical variable with two levels; women with high school or less and women with some college or more. Pre-pregnancy weight and height were collected from maternal medical records and pre-pregnancy BMI was calculated using weight (Kg)/Height(m)^2. Pre-pregnancy maternal BMI was used as a continuous variable. Child sex was gathered through maternal report at wave 1. Sex was used as a dichotomous variable; male and female. Child race was not reported; therefore, following the National Center for Health Statistics (NCHS), the race of the mother was used and assumed to be that of the child (NCHS, 2015).

Maternal race/ethnicity was used as a categorical variable with four levels; Non-Hispanic White, Non-Hispanic Black, Latino, and Other. Since this method has been criticized for not taking into account the role of the father's race in child outcomes (Mason, Nam, & Kim, 2014), I constructed a race category for the child using the mother's and the father's race into a single construct. I constructed this variable following the recommendation from Mason et al. (2014) where children with two parents of the same race/ethnicity were coded with the same race/ethnicity and children of parents with different race/ethnicity were coded as biracial/biethnic, including those of Latino heritage. For example, if one parent was Latino and the other was white, the child was coded as biracial/biethnic. Whenever one parent was coded as Other and the other parent was coded as *something besides* Other, the race of the child was coded

as Other since it was impossible to determine a specific race for the parent under the Other category. Using this method, I ended up with a total of five categories: Non-Hispanic White, Non-Hispanic Black, Latino, Biracial/Biethnic, and Other. I then tested whether there were any differences on the outcome measurements (i.e. Child BMI z-score) by using maternal race or the newly constructed child race and there were not any significant differences. Thus, I used maternal race since this variable was more parsimonious and it offered more complete and interpretable information. Table 3.2 provides information on the variables that were included in the study and the wave in which they were measured.

Table 3.2: Information included by Waves

Variable of Interest	Wave 1 (birth)	Wave 2 (age 1)	Wave 3 (age 3)	Wave 4 (age 5)	Wave 5 (age 9)
Dependent Variable					
Child BMI z-score	x		x	x	x
Independent Variable					
Maternal depression		x	x		
Child-level variables					
Hours of sleep				x	
Television viewing			x	x	
Outdoor play			x	x	
Dietary intake				x	
Clan-level variables					
Maternal-involvement			x	x	
Breastfeeding		x			
Control variables					
Maternal age	x				
Poverty level	x				
Education	x				
Race/ethnicity	x				
Number of biological children	x				
Pre-pregnancy BMI	x				
Child's sex	x				
Child's age	x	x	x	x	x

Note. BMI: Body Mass Index.

Data Screening Procedures

Data screening and data cleaning procedures were conducted prior to data analysis. First, univariate descriptive statistics on each variable were obtained (i.e. means, standard deviation, range, kurtosis, skewness, and missing values) using SPSS 24. Variables were recoded as necessary. In many cases, the variables were labeled by the Fragile Families research team with negative numbers (-9, -6, -2, etc.) as a way to indicate the reason they were missing. All these numbers were recoded into missing to avoid taking them into account as part of the answers

and/or avoid them from skewing the data. The variables on breastfeeding duration merit particular attention. Breastfeeding duration was not asked to the moms who decided not to breastfeed, and was therefore missing due to a legitimate skip pattern. Those mothers with a legitimate skip pattern were recoded into 0 in the continuous breastfeeding variable. The continuous variable was then used to create a dichotomous variable of those women who breastfed for 6 months or more.

I then examined the distribution of all variables to determine whether they were normally distributed. In order to assess normality, I used information on skewness, kurtosis, as well as histograms and scatterplots. Multicollinearity among independent variables was assessed using the Pearson's product-moment correlation coefficient between variables. A couple variables showed potential multicollinearity with each other by having a medium to high correlation coefficient. Maternal pre-pregnancy BMI and gestational weight gain were highly correlated with each other. To address this issue of multicollinearity, gestational weight gain was not included in the analysis. Maternal pre-pregnancy BMI was kept in the analysis instead of gestational weight gain given that the literature suggests that one of the main predictors of childhood adiposity and obesity is pre-pregnancy BMI (Yu et al., 2013). There was also high collinearity between breastfeeding initiation and breastfeeding duration. In this case, making the decision on which variable to keep was difficult because studies have found that breastfeeding initiation and duration are strong predictors of childhood overweight/obesity (Arenz et al., 2004; Eidelman et al., 2012; Gibbs & Forste, 2014; Yan et al., 2014). However, I decided to keep breastfeeding duration since recent studies have found that breastfeeding duration has a stronger effect on childhood obesity development than breastfeeding initiation (Yan et al., 2014).

Variables Assessed

The variables that were cleaned and prepared for use in this study included demographic variables (Table 3.1), child variables (Table 3.3), and maternal variables (Table 3.4). Other steps for variable assessment included examining the distribution of the variable, conducting principal components analysis (PCA), recoding and constructing summed scales, and checking for directionality of variables.

I also made decisions about cut points for certain variables such as maternal education, maternal relationship with baby's father, poverty level, and television viewing. Regarding maternal education, I created a two level variable of high school or less and at least some college. For the relationship of mother with baby's child, I created a three level categorical variable of married, cohabiting, and other. This three level variable was created in order to capture the nature of the Fragile Families dataset. Given that the FFCWS targeted unmarried mothers particularly, it was necessary to create distinct categories between married and cohabiting mothers, and distinguish these two groups from those mothers who are in a different relationship with the father of the baby (i.e. friends, not talking, etc.). For poverty level, I created a three-level category by collapsing the five-level variable on poverty provided by FFCWS. The three-level category included families who were between 0-99% of the federal poverty line, the next level included families between 100-199% of the poverty line, and the last level included families who were over 200% of the poverty line. Finally, for television viewing, I created a dichotomous variable with children who watched less than two hours of television per day and those who watched two hours or more.

Outliers were assessed as having implausible values. These were determined as having cases that were beyond the possible limits of the variables. For example, the variables that were

measured on an hourly basis (e.g. television viewing, hours of sleep, etc.) should not have more than 24 hours or less than 0 hours. If there were any observations that included more than 24 hours or less than 0 hours, they would be deemed as implausible. It is important to note that there were cases in which certain activities demonstrated a particular number that seemed to be an outlier (e.g. sleeping three hours per day). However, there were only a few of these cases and they were not implausible so these observations were kept.

Table 3.3: Child characteristics of the analytic sample Waves 2-5

Variable	n	*M (%)*	*SD*	Min - Max	Missing n (%)
Child BMI z-score W5	2629	0.78	1.10	-4.08 - 2.92	871 (24.89)
Child BMI z-score W4	1763	0.62	1.13	-4.16 - 4.74	1737 (49.63)
Child BMI z-score W3	1508	0.58	1.24	-4.27 - 4.83	1475 (42.14)
Child OW/OB status W5	2629				871 (24.89)
Normal and underweight	1508	57.36			
Overweight	451	17.15			
Obese	670	25.48			
Child OW/OB status W4	1763				1137 (49.62)
Normal and underweight	1145	64.95			
Overweight	323	18.32			
Obese	295	16.73			
Child OW/OB status W3	2025				1475 (42.14)
Normal and underweight	1305	64.44			
Overweight	351	17.33			
Obese	369	18.22			
Child age W5	2662	111.49	4.71	104-143	838 (23.94)
Child age W4	1910	63.76	3.03	34 - 76	1590 (45.43)
Child age W3	2745	37.46	3.20	31.3 - 53.4	755 (21.57)
Child age W2	3494	14.86	3.38	9 - 30	6 (0.17)
Sleep W4	2365				1135 (32.43)
Less than 10 hours	1199	50.70			
At least 10 hours	1166	49.30			
Television viewing W4	2332				1168 (33.37)
Less than 2 hours	694	29.76			
At least 2 hours	1638	70.24			
Television viewing W3					782 (22.34)
Less than 2 hours	748	27.52			
At least 2 hours	1970	72.48			
Outdoor play W4	2341	2.42	1.88	0-18.57	797 (22.77)
Outdoor play W3	2703	2.98	2.10	0-18.6	1672 (34.1)
Soda intake W4	2355	0.75	1.15	0-5	1145 (32.71)
Fruit and vegetable intake W4	2344	3.50	1.35	0-5	1156 (33.03)
Fast food intake W4	2365	1.22	1.03	0-5	1135 (32.43)
Breastfeeding ≥ 6 months	2770				730 (20.86)
No	2222	80.22			
Yes	548	19.78			

Note. BMI: body mass index. W1: wave 1 (birth). W2: wave 2 (age 1). W3: wave 3 (age 3). W4: wave 4 (age 5). W5: wave 5 (age 9).

Table 3.4: Maternal characteristics

Variable	n	*M (%)*	*SD*	Min-Max	Missing n (%)
Maternal involvement W3	3500	5.38	1.40	0-7	0.00
Maternal depression W3	3500			0-1	0.00
Yes	708	20.23			
No	2792	79.77			
Maternal depression W2	3500			0-1	0.00
Yes	535	84.71			
No	2965	15.29			
Maternal recurrent depression	3500				0.00
No maternal depression	2532	72.34			
Maternal depression at W2 *or* W3	693	19.80			
Maternal depression at W2 *and* W3	275	7.86			

Note. BMI: body mass index. W1: wave 1 (birth). W2: wave 2 (age 1). W3: wave 3 (age 3). W4: wave 4 (age 5). W5: wave 5 (age 9).

Principal Components Analysis (PCA)

The variables on maternal involvement were reduced using principal components analysis (PCA). PCA is a method in which many correlated variables are reduced into a small number of variables that still capture the information gathered by the original variables (Abdi & Williams, 2010). PCA was chosen as the method for data reduction because the main purpose was to identify and compute the least number of variables that could capture the main information of maternal involvement. Initially, there were 13 variables for maternal involvement at wave 3. However, only five variables were chosen to be included in the PCA: "How many days a week do you: 1) sing songs or nursery rhymes with [child] 2) Read stories to [child] 3) Tell stories to [child] 4) Play inside with toys such as blocks or legos with [child] 5) tell [child] that you appreciated something (he/she) did". I decided to use these five variables because they were related to activities carried out with the child (i.e. reading stories, playing with toys, etc.) rather than demonstrating display of affection (i.e. hugging child, saying "I love you").

After deciding to use only these five variables, I ran a PCA in SPSS 24. Before proceeding with PCA, I checked for three assumptions that need to be met before carrying out PCA. The first assumption is that the variables included need to be correlated with each other. All five variables had a correlation coefficient of at least 0.24 with each other and no variables had a correlation coefficient greater than 0.55. The second assumption is that the Kaiser-Meyer-Olkin measure of sampling adequacy needs to be at least 0.60 (Abdi & Williams, 2010). The KMO was 0.75, well above the minimum recommended. The final assumption is to reject the null hypothesis of having an identity matrix (Abdi & Williams, 2010). This is measured by the Barlett's test of sphericity. In this study the Barlett's test was significant (χ^2 (10) = 3005.13, $p <$ 0.01), indicating that the correlation matrix is not an identity matrix. After having checked and met all the assumptions, I proceeded with PCA.

The PCA eigen values indicated that the first component alone explained 46.26% of the variance. The two and three components explained 17.01% and 14.88% of the variance and four and five components explained even less. A one component solution was chosen because: 1) there was only one eigen value greater than 1, indicating only one principal component 2) the scree plot "leveled off" after one component. No item was eliminated from the component because all items contributed to the component structure and they all had a component loading of at least 0.54.

Since all five items/variables contributed to the component structure, a composite score of all five variables was created. These composite scores were based on the mean of the items of the five items. A higher score on the composite score indicated higher maternal involvement. Given the similarity of the questions, this composite score of maternal involvement was related to spending quality time with the child.

Missing Data

Missing data were inspected and reported for each single variable (see tables 3.5 and 3.6). Missing information on the main outcome, child BMI z-score at each wave, was not listwise deleted. The methodology that I used, group-based trajectory modeling, accommodates missing data, thus avoiding individuals with missing observations to be dropped (Nagin, 1999). Unless there was only one data point available (from all four time points), group-based trajectory modeling was able to identify the trajectory group membership. In case there were only one or less data points available, the observation was dropped from the trajectory modeling analysis. Missing information on maternal depression was deleted as part of the analytic sample strategy. Only nine observations were deleted because they had missing information on maternal depression at either wave 2 or 3.

Missing patterns were inspected among those who completed all waves and those who did not. I found that those participants who were present during all waves were more likely to be Non-Hispanic White (23.18% vs. 21.79; $\chi 2= 36.86$, $p <0.01$), Non-Hispanic Black (48.84% vs. 39.79%; $\chi 2= 36.86$, $p <0.01$) and less likely to be Latino (24.36% vs. 34.40%; $\chi 2= 36.86$, $p <0.01$), than those who were not present at all five waves. There were also significant differences on the pre-pregnancy BMI mean score among those participants who completed all waves and those who did not. The pre-pregnancy BMI mean score of those who completed all waves was higher than the mean score of those who did not complete all waves ($p = 0.01$). Finally, there were significant differences on maternal depression. Those participants who were present at all waves were more likely to be depressed at wave 2 *or* wave 3 (20.65 vs. 17.26; $\chi 2= 8.92$, $p = 0.01$) and at wave 2 *and* wave 3 (8.30 vs. 6.51; $\chi 2= 8.92$, $p = 0.01$), thank those not present at all

waves. There were no significant differences on any other variables, including child BMI z-score.

Table 3.5: Assessing missingness patterns in categorical variables

Variable	Participants present at all waves	Participants not present at all waves	χ^2	*p-value*
Child OW/OB status W3			1.02	0.60
Normal	1108 (64.91)	197 (61.95)		
Overweight	292 (17.11)	59 (18.55)		
Obese	307 (17.98)	62 (19.50)		
Child OW/OB status W4			0.16	0.92
Normal	1024 (64.97)	121 (64.71)		
Overweight	290 (18.40)	33 (17.65)		
Obese	262 (16.62)	33 (17.65)		
Child OW/OB status W5			0.71	0.70
Normal	1403 (57.50)	105 (55.56)		
Overweight	420 (17.21)	31 (16.40)		
Obese	617 (25.29)	53 (28.04)		
Breastfeeding ≥ 6 months			0.36	0.55
No	1655 (79.95)	567 (81.00)		
Yes	415 (20.05)	133 (19.00)		
Maternal recurrent depression			8.92	0.01
No maternal depression	1865 (71.05)	667 (76.23)		
Maternal depression at W2 or W3	542 (20.65)	121 (17.26)		
Maternal depression at W2 and W3	218 (8.30)	57 (6.51)		
Sleep W4			1.00	0.32
Less than 10 hours	1044 (50.31)	155 (53.45)		
At least 10 hours	1031 (49.69)	135 (46.55)		
Television viewing W3			0.91	0.34
Less than 2 hours	619 (27.91)	129 (25.80)		
At least 2 hours	1599 (72.09)	371 (74.20)		
Television viewing W4			0.00	0.99
Less than 2 hours	609 (29.77)	85 (29.72)		
At least 2 hours	1437 (70.23)	201 (70.28)		

Note. BMI: body mass index. W1: wave 1 (birth). W2: wave 2 (age 1). W3: wave 3 (age 3). W4: wave 4 (age 5). W5: wave 5 (age 9).

Table 3.6: Assessing missingness patterns in continuous variables

Variables	Mean score of participants present at all waves	Mean score of participants not present at all waves	t	*p-value*
Maternal age	25.48	25.04	1.76	0.08
Number of biological children	1.08	1.13	0.99	0.32
Pre-pregnancy BMI	26.61	25.73	-2.45	0.01
Child BMI z-score W1	-0.22	-0.25	0.62	0.53
Child BMI z-score W3	0.57	0.68	1.30	0.19
Child BMI z-score W4	0.62	0.61	-0.05	0.96
Child BMI z-score W5	0.78	0.84	0.46	0.64
Outdoor play W3	2.97	3.05	0.69	0.49
Outdoor play W4	2.43	2.31	-1.05	0.29
Maternal involvement W3	5.38	5.38	0.05	0.96
Soda intake W4	0.75	0.82	1.04	0.30
Fruit and vegetable intake W4	3.51	3.45	-0.70	0.49
Fast food intake W4	1.21	1.31	1.53	0.13
Maternal involvement W4	4.94	5.00	0.92	0.36

Note. BMI: body mass index. W1: wave 1 (birth). W2: wave 2 (age 1). W3: wave 3 (age 3). W4: wave 4 (age 5). W5: wave 5 (age 9).

The variables that were missing observations were listwise deleted, including maternal pre-pregnancy BMI. However, maternal pre-pregnancy BMI was missing more data than desirable (38.91%). Carrying out multiple imputation of this variable was not logical given that these are objectively measured responses. Thus, listwise deletion was used in models that included this variable because it was only assessed at one time point.

Data Analysis

Bivariate analysis

Bivariate analysis was carried out using different correlation types. In order to be true to the nature of the data I ran correlations based on the type of variables. When both variables were continuous and normally distributed, I ran Pearson correlations. When both variables were continuous but at least one of them was not normally distributed I ran Spearman correlations. In

case one variable was continuous and the other one was categorical, I ran point biserial correlations. Whenever one variable was ordinal and another categorical, I ran rank biserial correlations. When there were two categorical variables, I ran tetrachoric correlations and when the two variables were ordinal, I ran polychoric correlations. One problem that I encountered when running these correlations was that point biserial, rank biserial, tetrachoric, and polychoric correlations did not provide a *p*-value. To address this issue, I ran all correlations using Spearman and I used this *p*-value to indicate significant correlations.

Multivariable analysis

Multivariable assessment of relationships among variables was done using four main analytic strategies. First, general linear models (GLM) were used to examine the relationship between the main predictor variables (maternal depression) against the main outcome variable (child BMI z-score at wave 5). The next strategy was the use of logistic regression to test the associations between maternal depression and child overweight or obesity status at wave 5. The third strategy was the use of group-based trajectory modeling in order to identify different trajectories of growth (groups) over time. The next strategy was the use of logistic regression in order to examine the associations between maternal depression and childhood growth trajectories, and also to examine the relationships between the independent variables and childhood growth trajectories. Finally, analyses were stratified by race/ethnicity and group-based trajectory modeling was used to identify trajectories of growth over time. The following section provides an overview of the hypotheses proposed for this study and the statistical methods that were used to test them. See table 3.7 for a summary of the hypotheses and the analytic strategy used.

Table 3.7: Summary of Hypotheses and Analytic Strategy

Hypotheses	Analytic strategy
1a. Maternal depression at W2 and/or W3 will be associated with higher BMI z-score among children at W5, after adjusting for demographics (i.e. age, race, education).	1a. GLM to obtain associations between maternal depression and child BMI z-score at W5, controlling for demographic factors.
1b. Maternal depression at W2 and/or W3 will be associated with overweight (OW) and obesity (OB) status among children at W5, after adjusting for demographics (i.e. age, race, education).	1b. Logistic regression to obtain associations between maternal depression and child overweight and obesity status at W5, controlling for demographic factors.
2. Child growth patterns will follow multiple trajectories.	2. Group-based trajectory modeling to determine the best number of groups that fit the data over time.
3. Maternal depression at W2 and/or W3 will be associated with at-risk growth trajectory development.	3a. Chi-square analyses to determine whether maternal depression is associated with at-risk growth trajectory membership.
	3b. Logistic regression to test whether maternal is associated with at-risk growth trajectory membership.
4. Fewer hours of sleep and outdoor play and less fruit and vegetable consumption; and more television viewing, fast food, and soda intake will be associated with at-risk growth trajectory development.	4. Logistic regression to test whether fewer hours of sleep, outdoor play, and fruit and vegetable consumption; and more television viewing, fast food, and soda intake are associated with at-risk growth trajectory membership.
5. Lower levels of maternal involvement and breastfeeding for less than 6 months will be associated with at-risk growth trajectory development.	5. Logistic regression to test whether lower levels of maternal involvement and breastfeeding for less than 6 months are associated with at-risk growth trajectory membership.
6. Trajectories of growth will differ by race/ethnicity.	6. Group-based trajectory modeling to determine the best number of groups that fit the data over time stratified by race/ethnicity.
7. Significant predictors of the most at-risk trajectory will differ by race/ethnicity.	7. Logistic regression to test predictors associated with at-risk growth trajectory membership for each racial/ethnic group.

Note. OW: overweight; OB: obesity. W2: wave 2 (age 1). W3: wave 3 (age 3). W5: wave 5 (age 9).

Hypothesis 1: Testing the association between maternal depression and childhood BMI z-score and overweight/obesity status

Hypothesis 1a and 1b were tested using two different models: general linear models (GLM) when using a continuous BMI z-score outcome at wave 5, and logistic regression when using dichotomous outcome at wave 5. For logistic regression I combined the overweight and obesity categories into one and compared it to the underweight/normal weight category. Overall, hypotheses 1a and 1b aimed to determine the association between maternal depression and child BMI z-score at wave 5, controlling for child sex, maternal education, poverty level, race/ethnicity, relationship with baby's father, age, maternal pre-pregnancy BMI, and number of biological children. All control variables come from wave 1 (see figures 3.2 and 3.3). General linear models are an extension of ordinary linear regression, but in SAS they allow categorical variables to be included in the model, unlike ordinary linear regression models.

Figure 3.2: Conceptual model hypothesis 1a

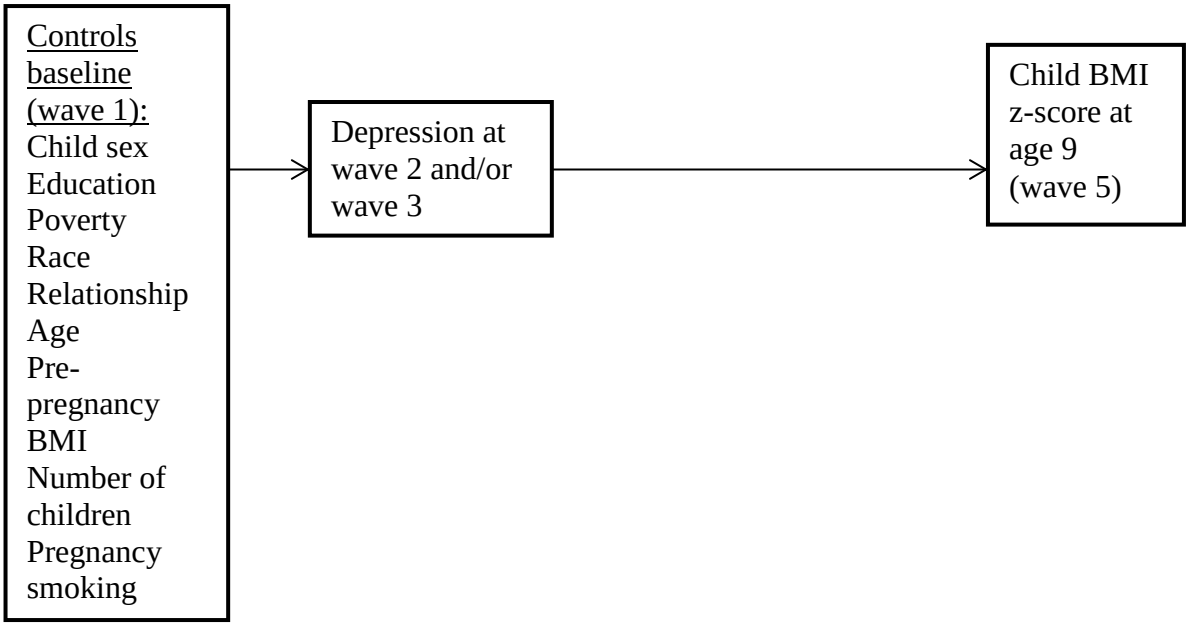
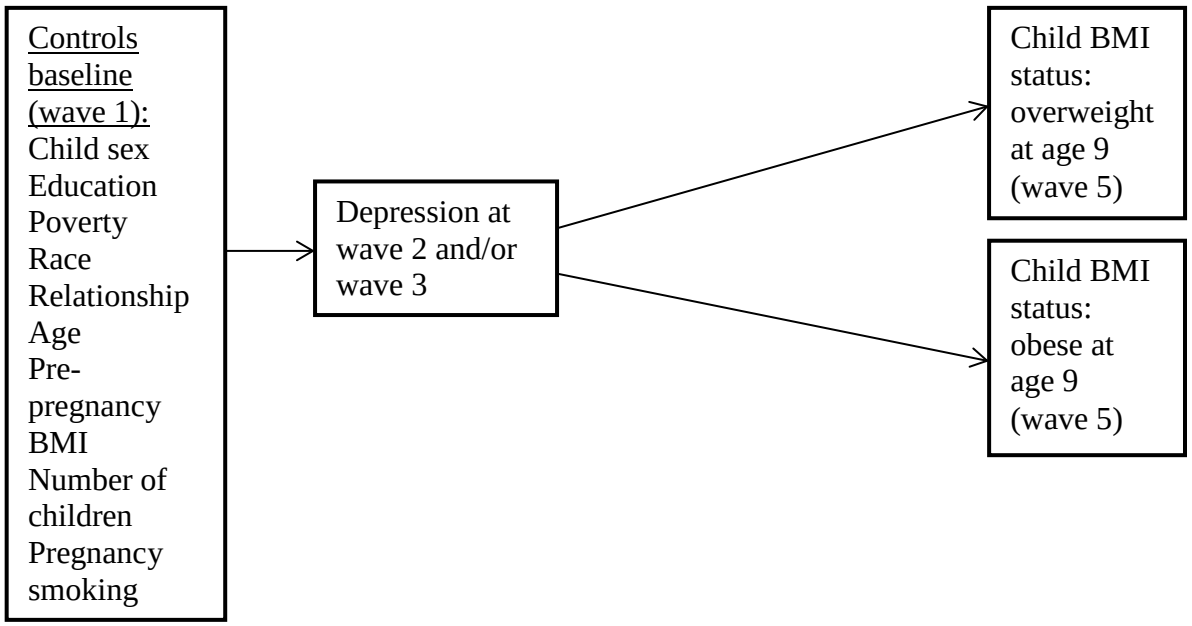


Figure 3.3: Conceptual model hypothesis 1b



Hypothesis 2: Child BMI z-scores growth trajectories

Group-based trajectory modeling (Jones, Nagin, & Roeder, 2001; Nagin, 1999, 2010) was used in order to identify the number of growth trajectories that best fit the data. Group-based trajectory modeling was designed to model developmental trajectories (Nagin, 1999, 2010). The main assumption of group-based trajectory modeling is that the population, in this case the children, have different growth patterns (Nagin, 1999, 2010). Thus, the main objective of this type of modeling is to identify specific and homogeneous clusters of trajectories (Nagin, 1999, 2010).

In order to determine the most optimal number of groups that best fit the data, a number of approaches were used (Nagin, 1999). The first approach was to determine model fit was the use of the Bayesian information criterion (BIC). Usually, a better BIC is one that is smaller. However, the BIC obtained by the SAS macro is negative; therefore, the smallest absolute value (or the least negative value) suggests a better fitting model (Jones et al., 2001; Nagin, 1999). The

second approach used was to check group membership probabilities (Nagin, 1999), which indicates the percentage of the sample that belongs to each group trajectory. The last approach that I used to determine model fit was to run a Wald test to determine whether the slopes were significantly different from each other (Jones & Nagin, 2007).

After identifying the most optimal number of groups, I added four risk factors into the model; child sex, maternal age, federal poverty line, and pregnancy smoking. These risk factors were selected because they have been shown to be associated with childhood overweight/obesity but they were not part of the main research questions, so I wanted to control for their effect on the modeling of the trajectories. In some cases the most optimal number of groups changed after adding risk factors to the model (Jones & Nagin, 2007). Thus, the fit of the trajectories was assessed before and after adding risk factors into the model and the number of optimal groups was changed whenever necessary to optimize the fit. After identifying the best model, each child was assigned to a group based on the growth pattern s/he had. The group that each child was assigned to was used as the outcome variable for hypotheses 3-5.

Hypothesis 3: Maternal depression and at-risk growth trajectories

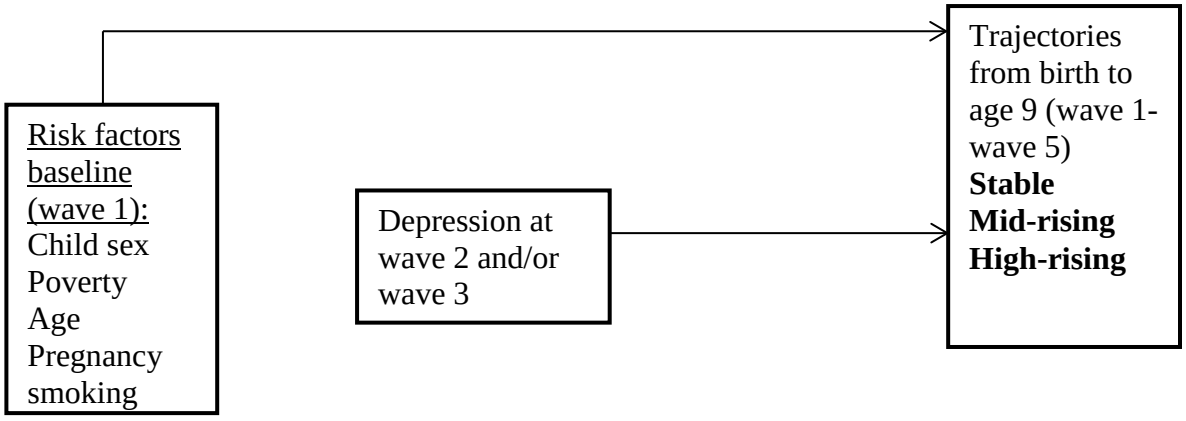
Hypothesis 3a was assessed using chi-square tests. The chi-square tests demonstrated whether there were any differences between the identified growth trajectories and maternal depression at wave 2 and/or wave 3.

Hypothesis 3b was assessed using logistic regression to determine the association between maternal depression and child BMI z-score trajectory, controlling for child sex, maternal education, federal poverty line, and pregnancy smoking.

Another method that could have been used to test this association was mixed effects models because in theory we have repeated measures over time and mixed effects models

provide an effective tool to address the correlation between variables over time. Another reason to use mixed effects models is that data from FFCWS could have a three-level nesting effect since children are nested within hospitals and hospitals are nested within cities. However, because I used the group trajectory as the outcome, the outcome was not a repeated measure that changed over time. Thus, using mixed effects models without having changing repeated measures over time would not make theoretical sense. In order to address the nesting effects; however, I did perform a mixed effects model and I found a covariance for hospital of zero, which indicated that there was no variability due to hospital selection. A covariance of zero was also found for city, indicating that there was no variability due to city selection. The covariances of zero for hospital and city are consistent with the random sampling design used by FFCWS. Given this information, I concluded that it was not necessary to use mixed effects models. Therefore, I decided to use logistic regression. See figure 3.4 for conceptual model.

Figure 3.4: Conceptual model for hypothesis 3.

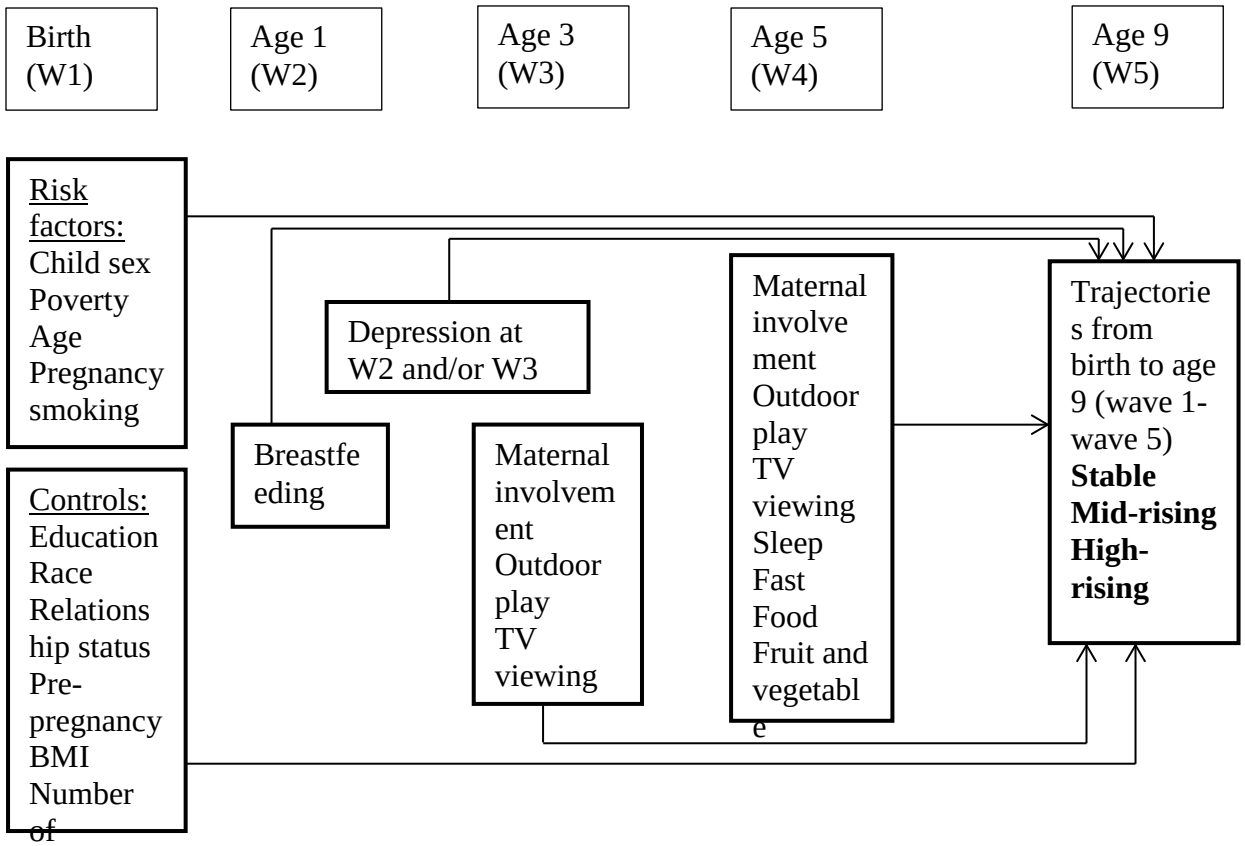


Hypothesis 4 and 5: Factors associated with growth trajectories

Logistic regression was used to assess whether fewer hours of sleep and outdoor play; more television viewing, fast food, soda intake, less maternal involvement, and breastfeeding duration were associated with at-risk trajectory group development. Two different models were

tested; one for hypothesis 4 and one for hypothesis 5. Two of the variables tested by these hypotheses were measured at two different time points; television viewing and outdoor play. Thus, an average of these variables was included in the model. See figure 3.5 for conceptual model.

Figure 3.5: Conceptual model for hypotheses 4 through 7.



Hypothesis 6: Child BMI z-score growth trajectories stratified by race/ethnicity

Group-based trajectory modeling (Jones et al., 2001; Nagin, 1999, 2010) was used in order to identify the number of growth trajectories that best fit the data by race/ethnicity. Data were analyzed separately in order to identify whether children from different race/ethnic backgrounds had different growth patterns. I fit separate group-based trajectory models for each group and identified the best number of trajectories using BIC, group membership probabilities,

and testing whether there were significant differences on the trajectories. All the models were adjusted for the same risk factors: child sex, maternal age, poverty level, and smoking during pregnancy. Each child was then assigned to a group depending on the growth trajectory s/he followed. Thus, the trajectory became the outcome variable to test hypothesis 7.

Hypothesis 7: Predictors of the most at-risk trajectory by race/ethnicity

Logistic regression was used to assess whether fewer hours of sleep and outdoor play; more television viewing, fast food, soda intake, more maternal involvement, and breastfeeding for 6 months or more were associated with at-risk trajectory group membership. All these factors were run in the sample stratified by race/ethnicity. Figure 3.5 shows the conceptual model that was used to test this hypothesis.

For this hypothesis the most at-risk group was chosen because not all racial/ethnic groups had the same number of trajectories; thus, choosing the most at-risk group would provide a good reference point.

CHAPTER 4: RESULTS

In this chapter I first present the results of bivariate analysis and then multivariable analysis to address each of the seven research questions outlined in chapter three. I will address each hypothesis separately. At the end of this chapter I will provide a summary of the main results. Overall, the research questions aimed to better understand the association, if any, between maternal depression and childhood obesity. Moreover, this study aimed to determine the predictors of at-risk trajectory group membership. Finally, this study aimed to understand whether there were differences on the growth trajectories and their predictors when stratifying by race/ethnicity.

Results of Bivariate Analysis

Among the main study variables, child BMI z-score at wave 5 was not correlated with maternal depression at wave 2 or wave 3. Maternal pre-pregnancy BMI was positively correlated with child BMI z-score at all time points. Maternal smoking during pregnancy was negatively correlated to child BMI z-score at wave 1 ($r = -0.05$, $p < 0.05$) and it was positively correlated to recurrent maternal depression ($r = 0.12$, $p < 0.01$). Number of biological children was positively correlated to child BMI z-score at wave 1 ($r = 0.10$, $p < 0.01$) and recurrent depression ($r = 0.05$, $p < 0.01$). Maternal age was positively correlated to child BMI z-score at wave 1 ($r = -0.05$, $p < 0.01$) and it was negatively correlated to recurrent depression ($r = 0.10$, $p < 0.01$).

Maternal education was negatively correlated with child BMI z-score at wave 5 ($r = -0.09$, $p < 0.01$). Being between 0-99% of the federal poverty line was positively correlated with child BMI z-score at wave 5 ($r = 0.06$, $p < 0.01$) and it was also positively correlated with maternal recurrent depression ($r = 0.06$, $p < 0.01$). Being on the 100-199% of the federal poverty line was positively associated with child BMI z-score at waves 3 ($r = 0.06$, $p < 0.05$), 4 ($r = 0.05$,

$p < 0.05$), and 5 (r = 0.05, $p < 0.05$). On the contrary, being on or over 200% of the federal poverty line was negatively correlated with child BMI z-score at wave 5 (r = -0.10, $p < 0.01$), and with maternal recurrent depression (r = -0.09, $p < 0.01$). Regarding race/ethnicity, White race was negatively correlated with child BMI z-score at wave 3 (r = -0.05, $p < 0.05$) and wave 5 (r = -0.14, $p < 0.01$). Moreover, Black race was negatively correlated with child BMI z-score at wave 1 (r = -0.09, $p < 0.01$) and wave 3 (r = -0.09, $p < 0.01$), but it was positively correlated with child BMI z-score at wave 5 (r = 0.06, $p < 0.01$). Finally, Latino ethnicity was positively correlated with child BMI z-score at all waves and it was negatively correlated with recurrent depression (r = -0.05, $p < 0.01$). Table 4.1 provides a summary of the correlations between the main study variables and the covariates from wave 1.

Table 4.1: Correlation Matrix of wave 1 study variables

Measure	1	2	3	4	5	6	7	8	9	10	11
1. Child BMI z-score (wave 1)	_										
2. Child BMI z-score (wave 3)	0.14**	_									
3. Child BMI z-score (wave 4)	0.19**	0.49**	_								
4. Child BMI z-score (wave 5)	0.11**	0.46**	0.68**	_							
5. Maternal recurrent depression	0.00	0.00	0.04	0.00	_						
6. Maternal pre-pregnancy BMI	0.09**	0.13**	0.22**	0.33**	0.06**	_					
7. Maternal smoking during pregnancy	-0.05*	-0.03	-0.01	0.01	0.12**	-0.02	_				
8. Number of biological children	0.10**	0.02	0.02	0.03	0.05**	0.20**	0.11**	_			
9. Maternal age at child's birth	0.08**	0.00	0.00	-0.01	-0.06**	0.19**	-0.02	0.40**	_		
10. Maternal education: HS or less	0.00	-0.04	-0.03	-0.09**	-0.04*	-0.01	-0.28**	-0.12**	0.07**	_	
11. Maternal federal poverty line: 0-99%	0.00	-0.02	0.00	0.06**	0.06**	0.05*	0.17**	0.20**	-0.18**	-0.55**	_
12. Maternal federal poverty line: 100-199%	0.02	0.06*	0.05*	0.05*	0.03	0.05*	0.06	0.02	-0.10**	-0.17**	_
13. Maternal federal poverty line: 200% or more	-0.01	-0.03	-0.04	-0.10**	-0.09**	-0.10**	-0.23**	-0.21**	0.26**	0.6**	_
14. Maternal relationship: Married	0.04	-0.03	-0.03	-0.10**	-0.11**	-0.03	-0.37**	0.03	0.03	0.52**	-0.54**
15. Maternal relationship: cohabiting	0.01	0.03	0.03	0.04**	0.05**	0.01	0.13**	0.04*	-0.12**	-0.24**	0.08**
16. Maternal relationship: Other	-0.05*	0.00	-0.01	0.05*	0.05**	0.02	0.14**	-0.07**	-.25**	-0.26**	0.34**
17. Race: White	0.01	-0.05*	-0.05	-0.14**	-0.02	-0.12**	0.23**	-0.09**	0.18**	0.41**	-0.48**
18. Race: Black	-0.09**	-0.09**	-0.04	0.06**	0.06**	0.11**	0.05	0.11**	-0.13**	-0.16**	0.26**
19. Race: Latino	0.11**	0.15**	0.11**	0.07**	-0.05**	0.03	-0.28**	-0.02	-0.05**	-0.28**	0.13**
20. Race: Other	-0.04	-0.01	-0.04	-0.02	-0.01	-0.07**	-0.13	-0.05**	0.06**	0.26**	-0.18**
21. Child sex	-0.01	0.01	-0.01	0.01	-0.02	-0.01	-0.03	-0.01	0.01	0.01	0.00

Table 4.1: (continued)

Measure	12	13	14	15	16	17	18	19	20	21
1. Child BMI z-score (wave 1)										
2. Child BMI z-score (wave 3)										
3. Child BMI z-score (wave 4)										
4. Child BMI z-score (wave 5)										
5. Maternal recurrent depression										
6. Maternal pre-pregnancy BMI										
7. Maternal smoking during pregnancy										
8. Number of biological children										
9. Maternal age at child's birth										
10. Maternal education: HS or less										
11. Maternal federal poverty line: 0-99%										
12. Maternal federal poverty line: 100-199%	–									
13. Maternal federal poverty line: 200% or more	–	–								
14. Maternal relationship: Married	-0.21**	0.59**	_							
15. Maternal relationship: cohabiting	0.07*	-0.13**	_	_						
16. Maternal relationship: Other	0.11**	-0.43**	_	_	_					
17. Race: White	-0.15**	0.51**	0.51**	-0.11**	-0.40**	_				
18. Race: Black	0.03	-0.27**	-0.45**	-0.07*	0.44**	_	_			
19. Race: Latino	0.11**	-0.22**	-0.07*	0.21**	-0.16**	_	_	_		
20. Race: Other	-0.03	0.18**	0.3**	-0.16**	-0.16**	_	_	_	_	
21. Child sex	0.01	-0.01	-0.01	-0.02	0.03	-0.01	-0.01	0.00	0.08	-

Note: All correlations used pairwise deletion. Thus, sample size varies for each correlation. **<0.01 *<0.05.

Correlations among the main study variables and independent variables from waves 2 through 5 are shown in Table 4.2. Television viewing for two hours or more at wave 3 was positively correlated with maternal recurrent depression ($r = 0.05$, $p < 0.05$). Maternal involvement at wave 3 was found to be negatively correlated with maternal depression at wave 3 ($r = -0.05$, $p < 0.01$). Sleeping less than the recommended amount of hours per night was negatively correlated with child BMI z-score at wave 5 ($r = -0.05$, $p < 0.05$) and also with recurrent depression ($r = -0.04$, $p < 0.05$). Finally, fast food consumption at wave 4 was positively correlated with child BMI z-score at wave 3 ($r = 0.05$, $p < 0.05$).

Table 4.2: Correlation Matrix of waves 2 through 5

Variable	1	2	3	4	5	6	7	8	9	10	11
1. Child BMI z-score (wave 1)	_										
2. Child BMI z-score (wave 3)	0.14**	_									
3. Child BMI z-score (wave 4)	0.19**	0.49**	_								
4. Child BMI z-score (wave 5)	0.11**	0.46**	0.68**	_							
5. Maternal recurrent depression	0.00	0.00	0.04	0.00	_						
6. Breastfeeding ≥ 6 months	0.00	-0.01	0.00	-0.07	-0.06**	_					
7. Television viewing => 2 hours W3	0.02	0.02	0.05	0.03	0.05*	-0.17**	_				
8. Outdoor play (per day) W3	0.00	-0.01	0.04	0.02	0.01	-0.05*	0.02	_			
9. Maternal involvement W3	-0.01	0.00	0.02	0.01	-0.05	0.01	-0.04	0.06**	_		
10. Television viewing => 2 hours W4	-0.03	0.00	0.00	0.04	-0.05	-0.19**	0.45**	0.03	-0.04	_	
11. Outdoor play (per day) W4	0.06**	-0.01	-0.04	-0.01	0.01	0.01	0.00	0.22**	0.06**	0.09**	_
12. Hours of sleep (per night) W4	0.01	-0.02	-0.04	-0.05*	-0.04*	0.17**	-0.21**	0.03	0.06**	-0.20**	-0.03
13. Soda consumption (per day) W4	0.00	0.06*	0.06*	0.06*	0.01	-0.12**	0.12**	0.05*	-0.06**	0.17**	0.07**
14. Fruit and vegetable intake (per day) W4	-0.02	-0.01	0.02	-0.01	0.01	-0.03	0.00	0.09**	0.14**	0.00	0.10**
15. Fast food intake (per week) W4	0.02	0.02	0.05*	0.03	0.01	-0.08**	0.11**	-0.01	-0.04*	0.10**	0.04
16. Maternal involvement W4	0.01	0.00	0.03	0.00	-0.02	0.01	-0.03	0.05*	0.54**	-0.01	0.06**
17. Television viewing => 2 hours (W2 and W3)	0.00	0.04	0.03	0.03	0.04	-0.20**	0.92**	0.03	-0.07**	0.89**	0.07**
18. Outdoor play average (W2 and W3)	0.04*	0.00	0.00	0.02	0.00	-0.03	0.02	0.85**	0.06**	0.09**	0.77**

Table 4.2: (continued)

Variable	12	13	14	15	16	17	18
1. Child BMI z-score (wave 1)							
2. Child BMI z-score (wave 3)							
3. Child BMI z-score (wave 4)							
4. Child BMI z-score (wave 5)							
5. Maternal recurrent depression							
6. Breastfeeding ≥ 6 months							
7. Television viewing => 2 hours W3							
8. Outdoor play (per day) W3							
9. Maternal involvement W3							
10. Television viewing => 2 hours W4							
11. Outdoor play (per day) W4							
12. Hours of sleep (per night) W4	_						
13. Soda consumption (per day) W4	-0.14**	_					
14. Fruit and vegetable intake (per day) W4	-0.01	0.12**	_				
15. Fast food intake (per week) W4	-0.08**	0.22**	0.05*	_			
16. Maternal involvement W4	0.06**	-0.03	0.18**	-0.06**	_		
17. Television viewing => 2 hours (W2 and W3)	-0.22**	0.17**	0.01	0.10**	-0.03	_	
18. Outdoor play average (W2 and W3)	0.00	0.08**	0.12**	0.03	0.06**	0.06**	_

Note: All correlations used pairwise deletion. Thus, sample size varies for each correlation. **<0.01 *<0.05. W2: wave 2 (age 1). W3: wave 3 (age 3). W5: wave 5 (age 9).

Association between Maternal Depression and Childhood BMI Z-Score (Question 1)

The first and main question of this study was to determine whether maternal depression in the first three years of the child's life was associated with childhood BMI z-score at age 9 (wave 5). I tested this question using GLM. Contrary to hypothesis 1a, neither maternal depression at any time point (when child was 1 *or* 3 years of age) nor recurrent maternal depression (when child was 1 *and* when child was 3 years of age) was associated with child BMI z-score at age 9. This held true for the unadjusted and the fully adjusted models (table 4.3).

The fully adjusted model (table 4.3) showed that being of Latino background was associated with a 0.25 ($p < 0.01$) BMI z-score unit increase at the age of 9 (wave 5). Also, higher maternal pre-pregnancy BMI and having more biological children were associated with higher BMI z-score at wave 5. One unit increase in maternal pre-pregnancy BMI was associated with an increase of 0.05 ($p < 0.01$) child BMI z-score at the age of 9. Also, for each additional child, there was a 0.05 ($p = 0.04$) decrease in child BMI z-score at the age of 9. Moreover, it was found that smoking during pregnancy was associated with a 0.16 ($p = 0.03$) child BMI z-score increase at age 9. Finally, the fully adjusted model showed that one unit increase in BMI z-score at birth was associated with a 0.07 ($p < 0.01$) increase in child BMI z-score at the age of 9.

Table 4.3: Regression analysis summary for variables predicting child BMI z-score at age 9 (wave 5)

	Model 1				Model 2			
Variable	B	SE	β	*p*	B	SE	β	*p*
Maternal recurrent depression								
Maternal depression at W2 or W3	0.05	0.05	0.02	0.32	-0.01	0.06	0.00	0.84
Maternal depression at W2 and W3	-0.07	0.08	-0.02	0.35	-0.12	0.09	-0.03	0.21
Child Sex (reference: female)								
Male					0.07	0.05	0.04	0.13
Maternal education (reference: high school or less)								
At least some college					-0.08	0.06	-0.04	0.20
Federal poverty line (reference: 0-99% of federal poverty line)								
100-199%					0.00	0.00	-0.01	0.96
200 and more					-0.07	0.07	-0.03	0.31
Race (reference: White)								
Black					0.12	0.08	0.05	0.12
Latino					0.25	0.08	0.10	<0.01
Other					0.22	0.15	0.04	0.14
Relationship (reference: married)								
Cohabiting					0.06	0.08	0.03	0.41
Other					0.11	0.08	0.05	0.18
Smoking during pregnancy (reference: no)								
Yes					0.16	0.07	0.05	0.03
Maternal age					0.00	0.01	0.01	0.65
Pre-pregnancy BMI					0.05	0.00	0.32	<0.01
Number of biological children					-0.05	0.03	-0.06	0.04
Child BMI z-score at birth					0.07	0.02	0.07	<0.01

Note: SE: Standard error. Model 1: unadjusted model. Model 2: fully adjusted model.

Hypothesis 1b aimed to determine whether there was an association between maternal depression and child overweight or obesity status. I used logistic regression, which estimates the odds of a child whose mother was depressed at age 1, 3, or both ages with being overweight/obese at age 9 (table 4.4). Contrary to my hypothesis, neither maternal depression at any time point, nor recurrent maternal depression was associated with childhood overweight or obesity in the unadjusted or adjusted models.

The fully adjusted model (table 4.4) demonstrated that maternal pre-pregnancy BMI and Latino ethnicity were associated with childhood overweight/obesity status at the age of nine (wave 5). For a one-unit increase in pre-pregnancy BMI, there was an 8% increase in the odds of children being overweight/obese at the age of 9 (wave 5; $p < 0.01$). Children of Latino background had 1.50 times the odds of being obese at the age of nine (wave 5; $p < 0.01$) compared to White children.

Table 4.4: Logistic regression analysis for variables predicting child *overweight/obesity* status at age 9 (wave 5)

	Model 1				Model 2			
Variable	B	SE	OR	*p*	B	SE	OR	*p*
Maternal recurrent depression								
Maternal depression at W2 or W3	0.04	0.10	1.04	0.67	-0.07	0.13	0.94	0.62
Maternal depression at W2 and W3	0.03	0.15	1.03	0.86	-0.06	0.20	0.94	0.74
Child Sex (reference: female)								
Male					-0.09	0.11	0.91	0.38
Maternal education (reference: high school or less)								
At least some college					-0.04	0.13	0.96	0.73
Federal poverty line (reference: 0-99% of federal poverty line)								
100-199%					-0.05	0.14	0.96	0.74
200 and more					-0.14	0.15	0.87	0.36
Race (reference: White)								
Black					0.16	0.15	1.18	0.30
Latino					0.40	0.17	1.50	0.01
Other					0.18	0.31	0.65	0.56
Relationship (reference: married)								
Cohabiting					0.14	0.15	0.85	0.35
Other					0.13	0.16	0.82	0.44
Smoking during pregnancy (reference: no)								
Yes					0.21	0.15	1.24	0.15
Maternal age					0.01	0.01	1.01	0.54
Pre-pregnancy BMI					0.09	0.01	1.08	<0.01
Number of biological children					-0.07	0.05	0.84	0.16

Note: SE: Standard error. Model 1: unadjusted model. Model 2: fully adjusted model.

Group-Based Trajectory Modeling (Question 2)

The second question was in relation to the analysis of BMI z-score growth patterns over time. I used group-based trajectory modeling to determine the best number of groups that fit the data. Jones and Nagin (2001) recommend that different number of groups are tested and also different polynomials (i.e. linear, quadratic, cubic) to determine the best fit. Thus, I tested one, two, three, and four, five, and six groups. I first tested all the groups with the same quadratic polynomial following the recommendation by Nagin (2005). After having tested all the groups with the quadratic polynomial, I identified the groups with the most negative BIC value and changed the polynomials to improve the fit. For instance, if the quadratic polynomial was not significant for a particular group, I tested the model with a linear polynomial next. Once again, I checked the BIC and kept the number of groups and polynomials with the most negative BIC value. The third step consisted of adding the risk factors (child sex, maternal smoking during pregnancy, maternal age, and maternal poverty) to the different models and of testing their fit again. This step was necessary because adding the risk factors could change the model fit. During this step, the model that best fit the data became obvious through the use of the lowest negative BIC value, and through the visual inspection of trajectories (e.g. narrow confidence intervals or no overlapping trajectories).

After identifying the model that best fit the data, I then checked the group membership probabilities, which is the number of participants that followed each trajectory, and I also ran Wald tests to make sure that no two trajectories were parallel. After running the data with six groups, the initial BIC suggested that five and six groups fit the data best (table 4.5). However, after visually inspecting the trajectories, it became clear that there were overlapping trajectories. Nagin (2005) suggests that although a BIC might keep going down as more groups are added to

the model, the researcher should always aim for having less groups (more parsimonious solutions) and making it more interpretable. Thus, I decided to continue testing the three and four-group trajectories, since these offered a solution that was more parsimonious and interpretable (Nagin, 2005). I then tested the three and four-group models with different polynomials (table 4.6) to improve their fit. Lastly, I added the risk factors to the three and four-group models. The four-group trajectory still offered the best fit; however, after inspecting the trajectories visually, one of the groups had a confidence interval straddling between -1.6 and 2 BMI z-scores, demonstrating this group did not provide useful information. To make sure that the four-group model was not the best model, I also ran a Wald test for the four-group solution. As expected, the Wald test showed that the slopes for two of the groups were not significantly different ($\chi^2 = 0.05$, $p = 0.47$), indicating that it was not necessary to have a fourth group (Jones & Nagin, 2007).

Table 4.5: Group-based trajectory modeling fit statics for one to six groups

Number of groups	Order of polynomials	BIC (observations)	BIC (subjects)	AIC
1	2	-13788.65	-13786.65	-13774.46
2	2, 2	-13350.51	-13346.53	-13322.14
3	2, 2, 2	-13277.90	-13271.93	-13235.35
4	2, 2, 2, 2	-13245.39	-13237.42	-13188.65
5	2, 2, 2, 2, 2	-13223.39	-13213.43	-13152.47
6	2, 2, 2, 2, 2, 2	-13208.88	-13196.93	-13123.77

Note: AIC: Akaike information criterion. BIC: Bayesian information criterion.

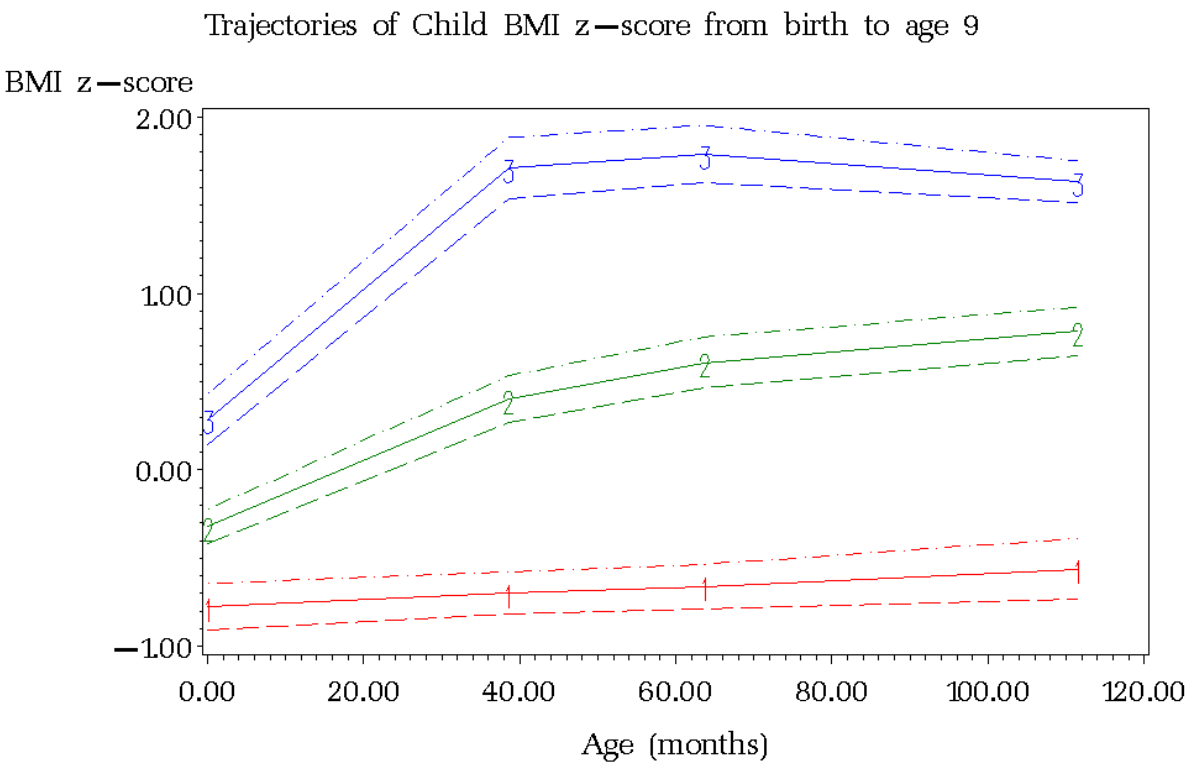
Table 4.6: Group-based trajectory modeling fit statics with risk factors

Number of groups	Order of polynomials	BIC (observations)	BIC (subjects)	AIC
3	1, 2, 2	-13275.00	-13265.54	-13207.65
3	1, 2, 3	-13265.69	-13255.72	-13194.78
4	1, 2, 1, 2	-13352.90	-13339.94	-13260.72

Note: AIC: Akaike information criterion. BIC: Bayesian information criterion.

As mentioned above, the best fitting model was a three-group solution. These trajectories were categorized as “stable”, “mid-rising”, and “high-rising” (see figure 4.1). The stable group started off around -0.8 BMI z-score and remained low throughout time. This group was characterized by 12.94% of the sample. The next group, the mid-rising group, started off closer to zero and it continued rising over time. At age 9, where the highest peak was, the BMI z-score was about 0.7. As discussed earlier, Wang and Chen (2012) have suggested that a BMI z-score of 1.04 translates to an 85^{th} percentile, thus placing children at risk for overweight. This mid-rising group was made up of 66.98% of the sample. Although children who followed this trajectory did not go over the 1.04 BMI z-score cut-off, the fact that this mid-rising trajectory did not stabilize is of great concern because it is possible that over time the BMI z-score of these children keeps increasing, thus placing them in a higher risk group. The last group, the high-rising group, started off around 0.3 BMI z-score but it grew rapidly until age five, where it reached a BMI z-score of 1.8 and then it started to plateau and even go down. This group was made up of 20.07% of the sample and it is the most at-risk group because their BMI z-score is well over the 1.04 cut-off, thus placing children at a high risk of overweight/obesity. Even after the decrease in BMI z-score at age 9, this trajectory showed a BMI z-score of 1.6.

Figure 4.1: Trajectories of child BMI z-score from birth to age 9 (wave 1 – wave 5).



Group-based trajectory modeling also allows one to model the groups controlling for risk factors. Thus, I added four risk factors into the model; child sex, maternal age, poverty level, and smoking during pregnancy. After adding the risk factors, the BIC improved, demonstrating a better fit (see tables 4.7 and 4.8). The results showed that being a male was a significant risk factor of developing a mid-rising trajectory (B = 0.36, p = 0.03; see table 4.7). No other risk factor was statistically significant for developing a mid-rising trajectory. Regarding the high-rising trajectory, only maternal poverty was a significant risk factor for being part of the high-rising trajectory (B = -0.24, p = 0.01; see table 4.8).

Table 4.7: Risk factors for the mid-rising trajectory group membership

Risk factor	Estimate	*p*-value
Child sex	0.36	0.03
Maternal smoking	0.09	0.65
Maternal age	-0.01	0.38
Maternal poverty	-0.14	0.13

Table 4.8: Risk factors for the high-rising trajectory group membership

Risk factor	Estimate	*p*-value
Child sex	0.18	0.22
Maternal smoking	-0.22	0.26
Maternal age	0.01	0.33
Maternal poverty	-0.24	0.01

Maternal Depression and its Association with Growth Trajectories (Question 3)

The third question aimed to investigate whether maternal depression was associated with the identified growth trajectories. Hypothesis 3a was first tested by using chi-square tests, since all variables of interest were categorical. The results from the chi-square test demonstrated that there was no association between maternal depression and growth trajectories ($\chi^2(4) = 2.19$; $p = 0.70$; table 4.9). Thus, hypothesis 3a was not supported.

Table 4.9: Chi-square test of maternal depression and group trajectory membership

	Group 1		Group 2		Group 3			
Variable	n	%	n	%	n	%	χ^2	*p*
Depression							2.19	0.70
No depression	332	73.45	1689	72.21	504	71.90		
Depression at any time point	80	17.70	468	20.01	145	20.68		
Depression at W2 and W3	40	8.85	182	7.78	52	7.42		

Hypothesis 3b consisted of testing the association between maternal depression and child growth trajectories using logistic regression. Because the high-rising trajectory was the only

category that was considered to be at risk, I combined the stable and the mid-rising groups and compared the high-rising to the other groups. The unadjusted model demonstrated that neither maternal depression when the child was one or when the child was three, nor recurrent maternal depression, were associated with increased risk of developing a high-rising trajectory (table 4.10). After adjusting for maternal education, race, relationship with baby's father, pre-pregnancy BMI, and number of children; maternal depression was not associated with developing a high-rising trajectory. However, the results showed that children of Latino background had 2.09 times the odds of developing a high-rising trajectory (95% CI = [1.50 – 2.90]), compared to children of White background. It was also found that maternal pre-pregnancy BMI was strongly associated with the high-rising trajectory. A one-unit increase in pre-pregnancy BMI was associated with a 0.07 increase in the odds of developing a high-rising trajectory. Finally, it was found that the number of children was associated with the high-rising trajectory. Each additional child in the home was associated with a 0.12 increase in the odds of the focal child developing a high-rising trajectory (95% CI = [1.03 – 1.22]).

Table 4.10: Logistic Regression Predicting High-Rising Growth Trajectory

	Model 1				Model 2			
Predictor	*B*	*SE*	*OR*	95% CI	*B*	*SE*	*OR*	95% CI
Maternal depression at W2 or W3	0.06	0.11	1.06	0.86 - 1.31	0.09	0.14	1.09	0.84 - 1.43
Maternal depression at W2 and W3	-0.06	0.16	0.94	0.68 – 1.29	-0.02	0.21	0.98	0.65 - 1.48
Education: at least some college					0.05	0.12	1.05	0.83 - 1.34
Race/ethnicity								
Black					0.12	0.17	1.13	0.82 - 1.57
Latino/a					0.74	0.17	2.09	1.50 - 2.90
Other					0.42	0.31	1.52	0.83 - 2.77
Relationship								
Cohabiting					-0.05	0.15	0.95	0.71 - 1.28
Other					-0.03	0.15	0.97	0.72 - 1.31
Pre-pregnancy BMI					0.07	0.01	1.07	1.05 - 1.09
Number of biological children					0.11	0.04	1.12	1.03 - 1.22

Note: SE: Standard error. Model 1: unadjusted model. Model 2: fully adjusted model.

Child-Level Factors and Their Association with Risky Growth Trajectories (Question 4)

The fourth research question aimed to determine whether child-level factors, such as sleep, television viewing, outdoor play, soda intake, fast food consumption, and fruit and vegetable consumption, were associated with the risky growth trajectory. In order to test this question, I used logistic regression. In the unadjusted model only soda consumption was associated with child BMI z-score; each additional soda consumed per day was associated with a 0.09 increase in the odds of developing a high-rising trajectory (95% CI = [1.00 – 1.19]). In the fully adjusted model, the results demonstrated that none of the child-level factors were associated with the high-risk trajectory development (table 4.11). Thus, hypothesis four was not supported.

Table 4.11: Child-level Factors and their Association with High-Rising Growth Trajectory

	Model 1				Model 2			
Predictor	*B*	*SE*	*OR*	95% CI	*B*	*SE*	*OR*	95% CI
Sleeping 10 hours or more	-0.07	0.10	0.93	0.76 - 1.14	0.13	0.14	1.14	0.87 - 1.49
Viewing TV two hours or more	0.15	0.12	1.16	0.92 - 1.46	-0.06	0.16	0.94	0.69 - 1.28
Outdoor play	-0.03	0.03	0.97	0.91 - 1.04	0.04	0.04	1.04	0.96 - 1.14
Fast Food	0.01	0.05	1.01	0.91 - 1.12	0.06	0.07	1.07	0.94 - 1.22
Soda	0.09	0.04	1.09	1.00 - 1.19	0.05	0.06	1.04	0.94 - 1.19
Fruit and vegetable	0.03	0.04	1.03	0.95 - 1.11	0.01	0.05	1.01	0.91 - 1.12
Education: at least some college					-0.01	0.15	0.99	0.73 - 1.34
Race/ethnicity								
Black					0.25	0.21	1.28	0.85 - 1.93
Latino/a					0.84	0.21	2.32	1.54 - 3.50
Other					0.15	0.45	1.16	0.48 - 2.81
Maternal relationship								
Cohabiting					-0.01	0.19	0.99	0.69 - 1.44
Other					-0.03	0.19	0.97	0.67 - 1.42
Pre-pregnancy BMI					0.06	0.01	1.06	1.04 - 1.08
Number of biological children					0.06	0.06	1.06	0.95 - 1.18

Note: SE: Standard error. Model 1: unadjusted model. Model 2: fully adjusted model.

Clan-Level Factors and Their Association with Risky Growth Trajectories (Question 5)

The objective of the fifth question was to identify whether clan-level variables such as maternal involvement and breastfeeding for six months or more, were associated with the development of the high-rising growth trajectory. This question was tested using logistic regression. The results demonstrated that neither breastfeeding duration, nor maternal involvement were significantly associated with the high-rising growth trajectory, either in the

unadjusted or the fully adjusted models in the full analytic sample (table 4.12). Thus, hypothesis 5 was not supported.

Table 4.12: Child-level Factors and their Association with High-Rising Growth Trajectory

	Model 1				Model 2			
Predictor	*B*	*SE*	*OR*	95% CI	*B*	*SE*	*OR*	95% CI
Breastfeeding ≥ 6 months	-0.13	0.12	0.88	0.65 - 1.05	0.19	0.16	1.21	0.89 - 1.64
Maternal involvement	0.00	0.03	1.00	0.94 - 1.01	0.02	0.04	1.02	0.94 - 1.11
Education: at least some college					-0.10	0.14	0.91	0.68 - 1.20
Race/ethnicity								
Black					0.10	0.19	1.10	0.76 - 1.61
Latino/a					0.84	0.19	2.32	1.59 - 3.40
Other					0.19	0.39	1.21	0.57 - 2.58
Maternal relationship								
Cohabiting					-0.08	0.17	0.93	0.66 - 1.29
Other					-0.07	0.18	0.93	0.66 - 1.31
Pre-pregnancy BMI					0.06	0.01	1.06	1.05 - 1.08
Number of biological children					0.12	0.05	1.13	1.03 - 1.24

Note: SE: Standard error. Model 1: unadjusted model. Model 2: fully adjusted model.

Child BMI Z-Score Growth Trajectories Stratified by Race/Ethnicity (Question 6)

The sixth question aimed to determine whether there were any differences on the growth patterns by race/ethnicity. In order to achieve this, I stratified the data and ran different models by race/ethnicity. Out of the 3500 participants, there were 797 (22.83%) participants who were White, 1626 (46.58%) who were Black, 938 (26.87%) who were Latino, 130 (3.72%) who were considered as other race/ethnicity and 9 participants were missing information on race/ethnicity. Thus, information from 3361 participants (96.28% of the total sample) was analyzed. Participants who had missing information on race/ethnicity or who were identified as other

race/ethnicity were not included in the analysis since it was impossible to determine their racial/ethnic background. Sample characteristics broken down by race/ethnicity can be found on Appendix B.

I tested one, two, three, four, five, and six groups, all using quadratic polynomials (Nagin, 2005). After having tested all the groups, I identified the groups with the most negative BIC value and adjusted their polynomials to improve the fit. I then checked the BIC again, and kept the models that had the most negative BIC value. I then added the risk factors to the different models and tested their fit again and assessed the accuracy of the trajectories visually. Ultimately, I checked the group membership probabilities to make sure that no group was too small, and I also ran Wald tests to assess that the slopes of the trajectories were significantly different from each other.

White children

Among White children, I found that a three-group solution was the best number of groups that fit the data (see table 4.13 and figure 4.2). Initially, a four-group trajectory offered the best fit. In fact, the BIC started to worsen when five and six groups were tested, demonstrating that four groups or less fit the data better. Because two, three, and four-group models offered a similar BIC, I tested all three models with different polynomials. After identifying the best polynomials for these models, I added the risk factors. In this step, it became evident that the three-group model fit the data best since it had the most negative BIC of all three models (table 4.14). None of the risk factors included in the model were significant for developing a high-rising trajectory (table 4.15).

The final three-group model showed a similar pattern as the one found for the overall sample with a “stable”, “mid-rising”, and “high-rising” group trajectories. There was a stable

group that started off around -0.6 BMI z-score and remained stable over time. This group was characterized by 17.09% of the sample. The second group started off around -0.4 BMI z-score but rose to about 0.4 BMI z-score at age 3 and it kept rising until 0.6 BMI z-score at age 5, and then it stabilized. This group was characterized by 70.23% of the sample. The last and most at-risk group started off with a BMI z-score of 0.6 and it kept increasing until age 5, where it decreased slightly until age 9. At its highest peak, at age 5, the BMI z-score of this trajectory reached a BMI z-score of 1.8, but at age 9, it decreased to 1.5. This group was characterized by 12.69% of the sample. Overall, in the sample of White children, two trajectories demonstrated a pattern that should not be of concern since the children are under the 1.04 BMI z-score cut-off (Wang & Chen, 2012). However, almost 13% of White children developed a risky growth trajectory that would place them in an overweight and even obese category (Wang & Chen, 2012).

Table 4.13: Group-based trajectory modeling fit statics for one to six groups

Number of groups	Order of polynomials	BIC (observations)	BIC (subjects)	AIC
1	2	-2866.16	-2864.26	-2855.08
2	2, 2	-2781.47	-2777.68	-2759.31
3	2, 2, 2	-2779.51	-2773.83	-2746.27
4	2, 2, 2, 2	-2767.41	-2759.83	-2723.08
5	2, 2, 2, 2, 2	-2773.49	-2764.01	-2718.08
6	2, 2, 2, 2, 2, 2	-2772.51	-2761.14	-2706.02

Note: AIC: Akaike information criterion. BIC: Bayesian information criterion.

Table 4.14: Group-based trajectory modeling fit statics with risk factors

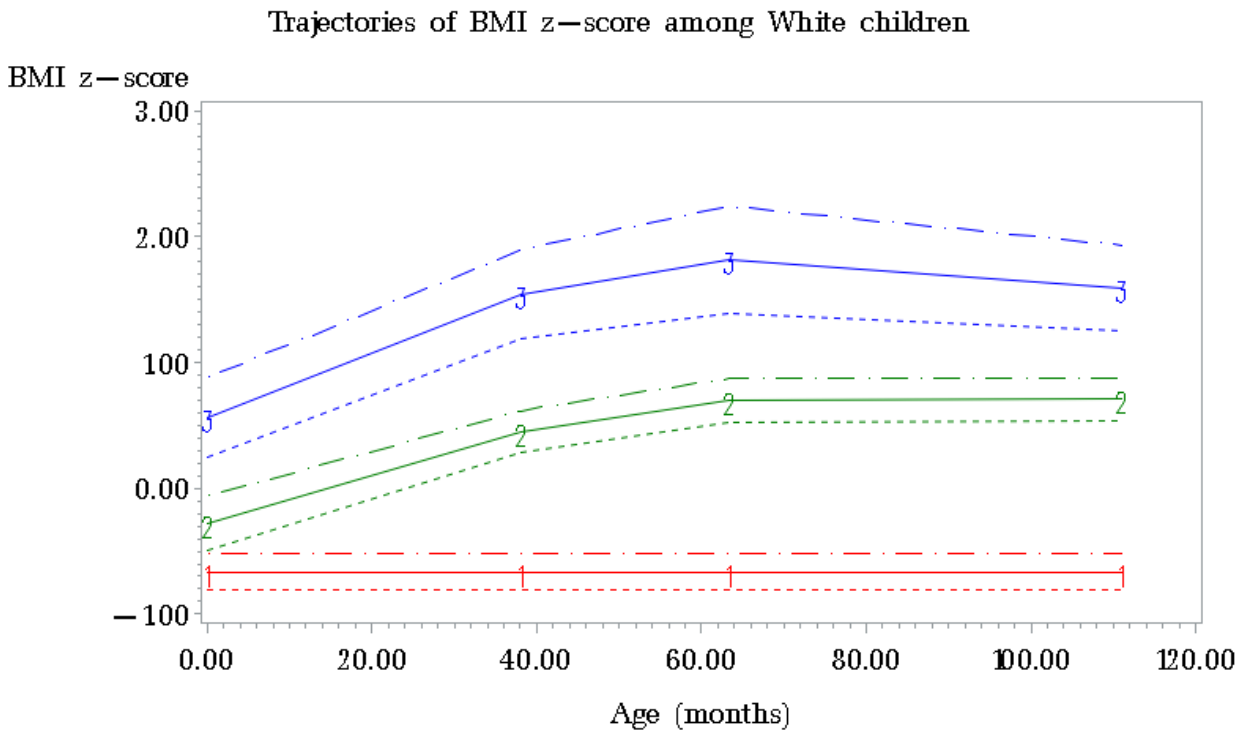
Number of groups	Order of polynomials	BIC (observations)	BIC (subjects)	AIC
2	2, 1	-2825.86	-2820.65	-2795.40
3	0, 2, 2	-2794.31	-2785.77	-2744.45
4	0, 2, 0, 1	-2805.08	-2794.18	-2741.37

Note: AIC: Akaike information criterion. BIC: Bayesian information criterion.

Table 4.15: Risk factors for developing high-rising trajectory

Risk factor	Estimate	*p*-value
Child sex	0.42	0.20
Maternal smoking	0.30	0.43
Maternal age	0.02	0.57
Maternal poverty	-0.03	0.88

Figure 4.2: Trajectories of child BMI z-score from birth to age 9 among White children.



Black children

Among Black children, a two-group trajectory was the best solution where there was a "slow-rising" group and a "mid-rising" group (table 4.16 and figure 4.3). Initially, the two, three, and four-group trajectory with quadratic polynomials showed a good fit. I then tested all these models with different polynomials to improve their fit even more. Then, I added the risk factors to the models and found that the two and four group trajectories provided a good fit (table 4.17). However, upon further inspection of the four-group trajectory, I found that one of the groups was

made up of 3% of the sample. Following Nagin's advice of choosing the number of groups that offers more parsimony and interpretability, I decided to keep the two-group model. The risk factors added to the model showed that higher maternal age was a significant predictor of developing a mid-rising trajectory (B = 0.03, p = 0.04; table 4.18). No other risk factor was a predictor of the mid-rising trajectory.

The final two-group model showed a "slow-rising" and a "mid-rising" trajectory. Children in the slow-rising group started off with a BMI z-score of -0.7-well below a normal BMI z-score- and their BMI z-score increased slowly over time, reaching a normal BMI z-score at age 9. Thus, the slow-rising trajectory was not of concern. Children in the mid-rising trajectory started off with a normal BMI z-score but grew until age 5, where they reached a BMI z-score of 1.2 and then the growth slowed down but reached a BMI z-score of 1.4 at age 9. This mid-rising trajectory is of concern given that children are over the 1.04 BMI z-score cut off recommended (Wang & Chen, 2012), thus being placed in an overweight (but not obese) trajectory. The majority of Black children were assigned to the mid-rising trajectory (64.96%), and 35.04% were assigned to the slow-rising trajectory.

Table 4.16: Group-based trajectory modeling fit statics for one to six groups

Number of groups	Order of polynomials	BIC (observations)	BIC (subjects)	AIC
1	2	-6747.73	-6745.66	-6734.98
2	2, 2	-6560.19	-6556.04	-6534.68
3	2, 2, 2	-6535.62	-6529.40	-6497.36
4	2, 2, 2, 2	-6532.44	-6524.14	-6481.42
5	2, 2, 2, 2, 2	-6528.79	-6518.41	-6465.02
6	2, 2, 2, 2, 2, 2	-6534.59	-6522.14	-6458.06

Note: AIC: Akaike information criterion. BIC: Bayesian information criterion.

Table 4.17: Group-based trajectory modeling fit statics with risk factors

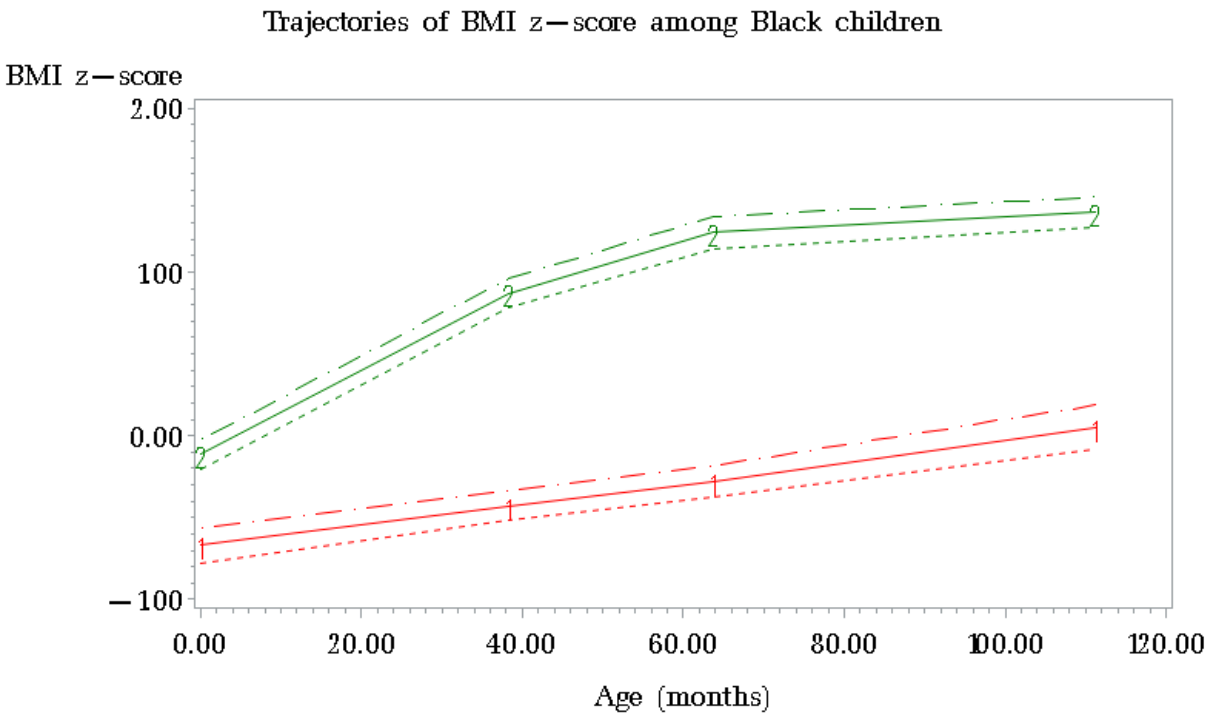
Number of groups	Order of polynomials	BIC (observations)	BIC (subjects)	AIC
2	1, 2	-6562.65	-6556.94	-6527.58
3	0, 2, 1	-6583.06	-6574.24	-6528.86
4	0, 2, 0, 2	-6554.71	-6542.26	-6478.2

Note: AIC: Akaike information criterion. BIC: Bayesian information criterion.

Table 4.18: Risk factors for developing mid-rising trajectory

Risk factor	Estimate	p-value
Child sex	-0.22	0.13
Maternal smoking	-0.02	0.92
Maternal age	0.03	0.04
Maternal poverty	-0.02	0.82

Figure 4.3: Trajectories of child BMI z-score from birth to age 9 among Black children.



Latino children

Among Latino children the best solution was a two-group solution where there was a "slow-rising" group and a "high-rising" group (table 4.19 and figure 4.4). Initially, the two,

three, and four-group trajectories with quadratic polynomials showed a good fit. I then tested all these models with different polynomials and added risk factors to the models (table 4.20). The BIC was the most negative for the two-group trajectory model with linear and quadratic polynomials, thus demonstrating that this was the best fit. The risk factors added to the model showed that there were not any significant predictors of developing a high-rising trajectory (table 4.21).

Children in the slow-rising group started off with a BMI z-score of -0.3 and there was a small increase over time, but the increase was subtle and not of concern. In fact, this increase put children at a normal BMI z-score over time. On the other hand, the high-rising trajectory was characterized by a normal BMI z-score at birth and a rapid growth until age 5, where it started to level off. At age 5, the trajectory reached a BMI z-score of 1.7 and then it went down to a BMI z-score of 1.5 at age 9. This category is of concern because it shows a very rapid growth over time and it places children on the overweight and obese categories. One of the main concerning aspects, however, is that the slow-rising group is made up of 35.22% of Latino children, whereas the high-rising group is made up of 64.78% of children. Thus, almost two-thirds of the Latino sample shows a concerning growth pattern.

Table 4.19: Group-based trajectory modeling fit statics for one to six groups

Number of groups	Order of polynomials	BIC (observations)	BIC (subjects)	AIC
1	2	-3612.14	-3610.19	-3600.63
2	2, 2	-3483.84	-3479.95	-3460.82
3	2, 2, 2	-3481.15	-3475.32	-3446.62
4	2, 2, 2, 2	-3477.77	-3469.99	-3431.73
5	2, 2, 2, 2, 2	-3480.62	-3470.9	-3423.07
6	2, 2, 2, 2, 2, 2	-3483.79	-3472.13	-3414.73

Note: AIC: Akaike information criterion. BIC: Bayesian information criterion.

Table 4.20: Group-based trajectory modeling fit statics with risk factors

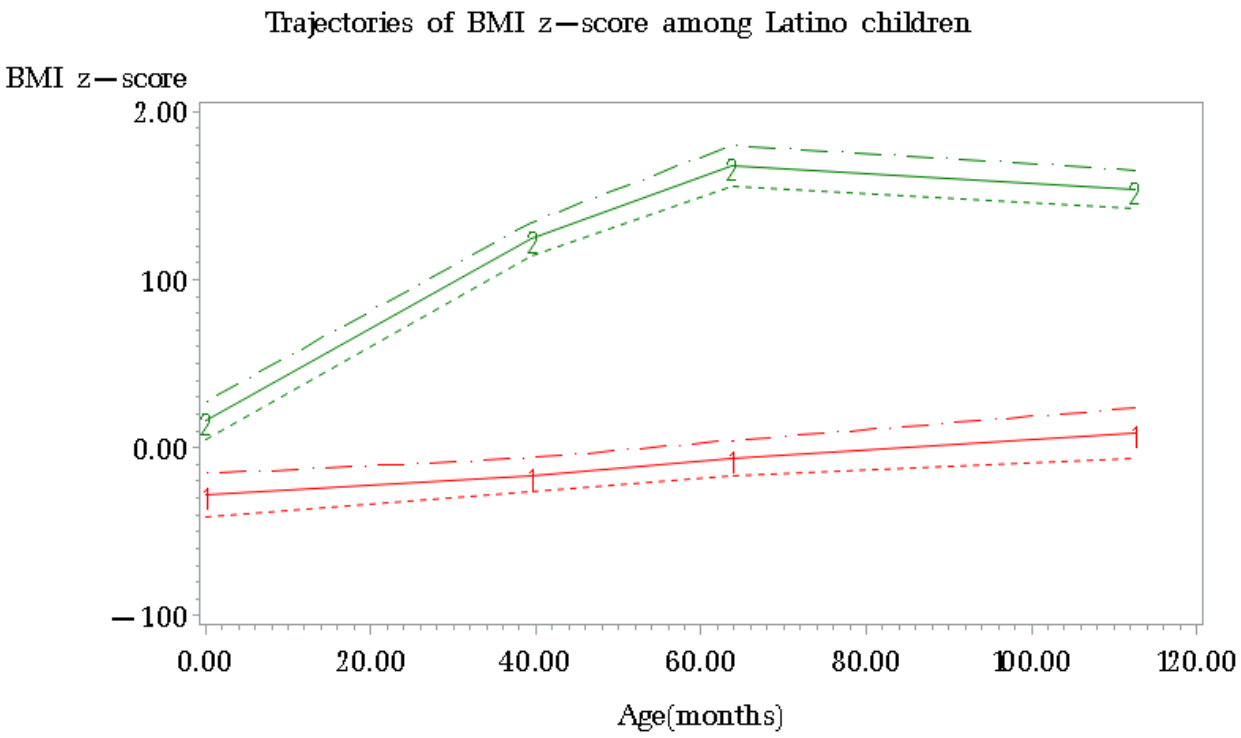
Number of groups	Order of polynomials	BIC (observations)	BIC (subjects)	AIC
2	1, 2	-3480.31	-3474.97	-3448.68
3	0, 1, 2	-3488.58	-3480.32	-3439.69
4	0, 0, 2, 0	-3491.25	-3480.57	-3427.99

Note: AIC: Akaike information criterion. BIC: Bayesian information criterion.

Table 4.21: Risk factors for belonging to mid-rising group

Risk factor	Estimate	p-value
Child sex	0.29	0.13
Maternal smoking	-0.18	0.58
Maternal age	0.00	0.92
Maternal poverty	-0.12	0.29

Figure 4.4: Trajectories of child BMI z-score from birth to age 9 among Latino children.



Overall, the findings demonstrated that there are qualitatively different growth trajectories for each racial/ethnic group. Among White children, the majority of them (87.31%)

developed a trajectory that was normal. On the other hand, 12.69% of them had a trajectory that was characterized by rapid growth and that placed children in an overweight/obese category, since they crossed the 1.04 and 1.64 BMI z-score cut offs that translate into the 85th and 95th percentiles, respectively (Wang & Chen, 2012).

Among Black children, overweight and obesity seemed not to be of concern, but instead undernutrition, since 35% of the sample started off with a BMI z-score of -0.7 and did not reach a normal BMI z-score of zero until age 9. Among Black children, the mid-rising group was characterized by rapid growth until age 5 and then the growth slowed down. The highest BMI z-score reached by this group was 1.4, which places children over the 85th percentile but under the 95th percentile, meaning that their growth is at risk of overweight but not obesity.

The group with the most striking results was for children of Latino background. Among this ethnic group, almost 65% of the population showed a risky growth trajectory with a very rapid increase in BMI from birth until age 5. This growth trajectory was of particular concern because the highest BMI z-score reached at age 5 was 1.7, which places children over the 95th percentile. Although there is no test of significance to test whether the differences between these three sets of trajectories are statistically significant, descriptively, I can conclude that hypothesis 6 is supported. There are qualitatively different growth trajectories by racial/ethnic groups, with Latino children having the greatest proportion of children in high-risk growth trajectories.

Factors that Predicted At-Risk Growth among Children Will Differ by Racial/Ethnic Group (Question 7)

The final research question was to determine the common and unique factors that placed children from different race/ethnicities at an increased risk of following at-risk growth trajectories. The purpose of this question was to identify risk factors that could be addressed

through tailored interventions. In order to test this hypothesis more systematically, I tested the same factors that I tested for the overall sample (maternal depression, child-level, and clan-level factors). Each one of these factors will first be discussed independently and then a summary will be presented for the risk factors of all the racial/ethnic groups.

White children

In order to test hypothesis 7 among White children, logistic regression was carried out. Given that among White children only the high-rising group placed children at risk for overweight/obesity, groups one and two were combined to compare the high-rising and most at risk trajectory to the other two non-at-risk groups.

The first model I tested was to determine whether maternal depression was associated with developing a high-rising trajectory. The results demonstrated that neither maternal depression at one time point, nor recurrent maternal depression, was associated with developing a high-rising trajectory among White children. This was true for the unadjusted and the fully adjusted models (table 4.22). It was also found that maternal pre-pregnancy BMI (B = 0.05, 95% CI = [1.01 - 1.09]) and number of biological children (B = 0.29, 95% CI = [1.08 - 1.65]) were associated with developing a high-risk trajectory. More specifically, it was shown that a one-unit increase in maternal pre-pregnancy BMI was associated with a 5% increase in the odds of developing a high-rising trajectory. Also, it was found that for each additional child in the house, the child had 34% increased odds of developing a high-rising trajectory.

Table 4.22: Logistic Regression Predicting High-Rising Growth Trajectory

	Model 1				Model 2			
Predictor	*B*	*SE*	*OR*	95% CI	*B*	*SE*	*OR*	95% CI
Maternal depression at W2 or W3	0.17	0.27	1.19	0.70 - 2.02	0.28	0.33	1.32	0.70 - 2.50
Maternal depression at W2 and W3	-0.19	0.42	0.83	0.36 - 1.89	-0.68	0.62	0.51	0.15 - 1.72
Education: at least some college					-0.02	0.31	0.98	0.54 - 1.79
Maternal relationship								
Cohabiting					-0.07	0.35	1.07	0.54 - 2.10
Other					0.41	0.36	1.50	0.74 - 3.05
Pre-pregnancy BMI					0.05	0.02	1.05	1.01 - 1.09
Number of biological children					0.29	0.11	1.34	1.08 - 1.65

Note: SE: Standard error. Model 1: unadjusted model. Model 2: fully adjusted model.

The next model tested aimed to determine whether there was any association between the child-level factors and the high-rising trajectory (table 4.23). In the unadjusted model, none of the child-level factors were associated with the high-rising trajectory. In the adjusted model, fast food consumption was significantly associated with the high-rising trajectory. A one-unit increase in fast food consumption was associated with a 45% increase in the odds of developing a high-rising trajectory (95% CI = [1.01 – 2.07]). Maternal pre-pregnancy BMI and number of children were also significant predictors of the high-rising trajectory.

Table 4.23: Child-level Factors and their Association with High-Rising Growth Trajectory

	Model 1				Model 2			
Predictor	*B*	*SE*	*OR*	95% CI	*B*	*SE*	*OR*	95% CI
Sleeping 10 hours or more	-0.36	0.27	0.70	0.40 - 1.19	0.05	0.37	1.05	0.51 - 2.15
Viewing TV two hours or more	0.41	0.29	1.50	0.85 - 2.64	0.46	0.40	1.59	0.73 - 3.47
Outdoor play	0.01	0.08	1.01	0.86 - 1.19	0.19	0.11	1.20	0.96 - 1.51
Fast Food	0.14	0.14	1.15	0.87 - 1.52	0.37	0.18	1.45	1.01 - 2.07
Soda	0.05	0.14	1.05	0.81 - 1.37	-0.04	0.18	0.96	0.68 - 1.36
Fruit and vegetable	0.10	0.10	1.10	0.90 - 1.35	0.14	0.14	1.16	0.88 – 1.51
Education: at least some college					-0.10	0.40	0.90	0.42 - 1.96
Maternal relationship								
Cohabiting					-0.10	0.46	0.90	0.37 – 2.21
Other					0.47	0.46	1.60	0.65 - 3.92
Pre-pregnancy BMI					0.05	0.02	1.05	1.00 - 1.10
Number of biological children					0.38	0.17	1.46	1.04 - 2.04

Note: SE: Standard error. Model 1: unadjusted model. Model 2: fully adjusted model.

The last model tested aimed to determine whether breastfeeding for less than 6 months and lower maternal involvement were associated with the high-rising trajectory (table 4.24). In the unadjusted and adjusted models I found that neither breastfeeding for less than 6 months , nor maternal involvement were associated with the high-rising trajectory. Only the number of children was a significant predictor of high-rising trajectory membership (B = 0.35, 95% CI = [1.07 – 1.88]).

Table 4.24: Clan-level Factors and their Association with High-Rising Growth Trajectory

	Model 1				Model 2			
Predictor	*B*	*SE*	*OR*	95% CI	*B*	*SE*	*OR*	95% CI
Breastfeeding ≥ 6 months	-0.20	0.30	0.82	0.46 - 1.47	0.32	0.44	1.38	0.58 - 3.27
Maternal involvement	0.02	0.11	1.02	0.83 - 1.25	0.04	0.15	1.04	0.78 - 1.38
Education: at least some college					-0.53	0.40	0.59	0.27 - 1.29
Maternal relationship								
Cohabiting					-0.10	0.42	0.90	0.39 - 2.07
Other					1.06	0.51	2.88	1.05 - 7.87
Pre-pregnancy BMI					0.12	0.03	1.13	1.06 - 1.20
Number of biological children					0.52	0.19	1.68	1.20 - 2.36

Note: SE: Standard error. Model 1: unadjusted model. Model 2: fully adjusted model.

Black children

Among Black children, I carried out the same process that I did with White children. I used logistic regression to test the factors associated with the mid-rising trajectory. Regarding maternal depression, I found that neither maternal depression at one-time point, nor recurrent maternal depression was associated with developing a mid-rising trajectory (table 4.25). This was true for the unadjusted and the adjusted models. It was also found that maternal pre-pregnancy BMI was associated with developing a mid-rising trajectory (B = 0.07, $p < 0.01$).

Table 4.25: Logistic Regression Predicting Mid-Rising Growth Trajectory

	Model 1				Model 2			
Predictor	*B*	*SE*	*OR*	95% CI	*B*	*SE*	*OR*	95% CI
Maternal depression at W2 or W3	0.11	0.13	1.11	0.87 - 1.43	0.04	0.17	1.04	0.75 - 1.44
Maternal depression at W2 and W3	-0.14	0.18	0.87	0.61 - 1.25	-0.15	0.25	0.86	0.53 - 1.41
Education: at least some college					0.17	0.16	1.19	0.88 - 1.61
Maternal relationship								
Cohabiting					-0.11	0.23	0.9	0.57 - 1.42
Other					-0.18	0.22	0.93	0.54 - 1.29
Pre-pregnancy BMI					0.07	0.01	1.07	1.04 - 1.09
Number of biological children					0.05	0.06	1.06	0.94 - 1.18

Note: SE: Standard error. Model 1: unadjusted model. Model 2: fully adjusted model.

The next model tested whether child-level factors such as sleep, television viewing, outdoor play, fast food, soda consumption, and fruit and vegetable intake were associated with developing a mid-rising trajectory (table 4.26). The results from the unadjusted and adjusted models demonstrated that none of the child-level factors were associated with the mid-rising trajectory among Black children.

Table 4.26: Child-level Factors and their Association with Mid-Rising Growth Trajectory

	Model 1				Model 2			
Predictor	*B*	*SE*	*OR*	95% CI	*B*	*SE*	*OR*	95% CI
Sleeping 10 hours or more	-0.06	0.12	0.94	0.74 - 1.20	-0.14	0.17	0.87	0.63 - 1.21
Viewing TV two hours or more	-0.15	0.15	0.96	0.64 - 1.16	-0.04	0.20	0.70	0.47 - 1.05
Outdoor play	0.01	0.04	1.01	0.94 - 1.09	-0.02	0.05	0.98	0.89 - 1.09
Fast Food	-0.03	0.06	0.97	0.87 - 1.09	-0.02	0.08	0.98	0.84 - 1.14
Soda	-0.01	0.05	0.99	0.89 - 1.10	0.01	0.07	1.01	0.87 - 1.16
Fruit and vegetable	0.01	0.05	1.01	0.92 - 1.11	0.05	0.06	1.05	0.93 - 1.19
Education: at least some college					0.12	0.19	1.12	0.78 - 1.61
Maternal relationship								
Cohabiting					0.19	0.27	1.21	0.71 - 2.08
Other					-0.13	0.26	0.87	0.52 - 1.46
Pre-pregnancy BMI					0.06	0.01	1.06	1.04 - 1.09
Number of biological children					0.05	0.07	1.05	0.92 - 1.19

Note: SE: Standard error. Model 1: unadjusted model. Model 2: fully adjusted model.

The final model tested whether breastfeeding for less than 6 months and maternal involvement were associated with the development of the mid-rising trajectory (table 4.27). The results suggested that maternal involvement was significantly associated with the mid-rising trajectory. However, maternal involvement was no longer significant after adjusting for demographics.

Table 4.27: Clan-level Factors and their Association with Mid-Rising Growth Trajectory

	Model 1				Model 2			
Predictor	*B*	*SE*	*OR*	95% CI	*B*	*SE*	*OR*	95% CI
Breastfeeding ≥ 6 months	-0.12	0.16	0.88	0.64 - 1.21	-0.27	0.21	0.77	0.51 - 1.16
Maternal involvement	0.09	0.04	1.09	1.02 - 1.18	0.05	0.05	1.05	0.95 - 1.17
Education: at least some college					0.31	0.18	1.37	0.97 - 1.94
Maternal relationship								
Cohabiting					-0.06	0.25	0.95	0.58 - 1.55
Other					-0.27	0.24	0.76	0.47 - 1.23
Pre-pregnancy BMI					0.06	0.01	1.06	1.03 - 1.08
Number of biological children					0.07	0.06	1.08	0.95 - 1.22

Note: SE: Standard error. Model 1: unadjusted model. Model 2: fully adjusted model.

Latino children

Among Latino children I also used logistic regression to test the different models since the best fitting model for Latinos was a two-group trajectory model. The first model I tested for Latinos aimed to determine whether maternal depression was associated with the high-rising trajectory (table 4.28). The results demonstrated that among Latinos, neither depression at any time point, nor recurrent depression was associated with developing a high-risk trajectory. This remained true for the unadjusted and the fully adjusted models. The results also demonstrated that only maternal pre-pregnancy BMI was a significant factor associated with the high-rising trajectory. A one-unit increase in pre-pregnancy BMI was associated with a 0.09% increase in the odds of developing a high-rising versus a slow-rising trajectory.

Table 4.28: Logistic Regression Predicting High-Rising Growth Trajectory

	Model 1				Model 2			
Predictor	*B*	*SE*	*OR*	95% CI	*B*	*SE*	*OR*	95% CI
Maternal depression at W2 or W3	0.19	0.18	1.20	0.84 - 1.72	-0.06	0.23	0.94	0.60 - 1.47
Maternal depression at W2 and W3	-0.18	0.29	0.83	0.48 - 1.46	-0.48	0.35	0.62	0.31 - 1.23
Education: at least some college					-0.15	0.20	0.86	0.58 - 1.28
Maternal relationship								
Cohabiting					-0.15	0.22	0.87	0.56 - 1.34
Other					0.09	0.24	1.10	0.69 - 1.75
Pre-pregnancy BMI					0.09	0.02	1.09	1.06 - 1.13
Number of biological children					-0.05	0.07	0.95	0.82 - 1.10

Note: SE: Standard error. Model 1: unadjusted model. Model 2: fully adjusted model.

The next model for Latino children tested whether child-level factors were associated with the high-rising trajectory. The unadjusted and adjusted models showed that none of the child level factors were significantly associated with the high-rising trajectory (table 4.29).

Table 4.29: Child-level Factors and their Association with High-Rising Growth Trajectory

	Model 1				Model 2			
Predictor	*B*	*SE*	*OR*	95% CI	*B*	*SE*	*OR*	95% CI
Sleeping 10 hours or more	0.03	0.17	1.03	0.73 - 1.44	0.09	0.22	1.09	0.71 - 1.67
Viewing TV two hours or more	0.27	0.19	1.31	0.90 - 1.91	0.13	0.25	1.14	0.70 - 1.84
Outdoor play	-0.04	0.05	0.96	0.87 - 1.06	-0.01	0.07	0.99	0.87 -1.12
Fast Food	-0.05	0.09	0.95	0.81 - 1.13	0.02	0.11	1.02	0.83 - 1.27
Soda	-0.04	0.07	0.96	0.83 - 1.11	-0.05	0.09	0.95	0.79 - 1.14
Fruit and vegetable	-0.01	0.06	0.99	0.87 - 1.11	-0.03	0.08	0.97	0.83 - 1.13
Education: at least some college					-0.38	0.25	0.69	0.42 - 1.13
Maternal relationship								
Cohabiting					-0.18	0.28	0.83	0.48 - 1.44
Other					0.26	0.30	1.30	0.73 - 2.33
Pre-pregnancy BMI					0.09	0.02	1.09	1.05 - 1.14
Number of biological children					-0.18	0.10	0.84	0.70 - 1.01

Note: SE: Standard error. Model 1: unadjusted model. Model 2: fully adjusted model.

The final model tested among Latino children aimed to identify whether clan-level factors, such as maternal involvement and breastfeeding less than 6 months were associated with developing a high-rising trajectory (table 4.30). The results demonstrated that neither maternal involvement nor lower breastfeeding duration were significantly associated with the high-rising trajectory. This was true for the unadjusted and the fully adjusted models. Maternal pre-pregnancy BMI was the only factor that was associated with developing a high-rising trajectory. For each one-unit increase in pre-pregnancy BMI, Latino children had 11% higher odds of developing a high-rising versus a slow-rising trajectory (95% CI = [1.07 – 1.16]).

Table 4.30: Clan-level Factors and their Association with High-Rising Growth Trajectory

	Model 1				Model 2			
Predictor	*B*	*SE*	*OR*	95% CI	*B*	*SE*	*OR*	95% CI
Breastfeeding ≥ 6 months	-0.19	0.19	0.83	0.57 - 1.19	0.05	0.24	1.06	0.66 - 1.70
Maternal involvement	-0.01	0.05	0.99	0.89 - 1.10	-0.05	0.07	0.95	0.83 - 1.08
Education: at least some college					-0.07	0.24	0.93	0.58 – 1.49
Maternal relationship								
Cohabiting					0.07	0.25	1.07	0.65 - 1.76
Other					0.16	0.27	1.17	0.69 - 2.00
Pre-pregnancy BMI					0.10	0.02	1.11	1.07 - 1.16
Number of biological children					-0.12	0.09	0.89	0.75 - 1.06

Note: SE: Standard error. Model 1: unadjusted model. Model 2: fully adjusted model.

After testing the same demographic, child-level, and clan-level factors for each racial/ethnic group separately, I found that there was one common factor associated with developing at-risk trajectories. The only common factor that was associated with developing at-risk growth trajectories was maternal pre-pregnancy BMI. Among White children, the number of biological children was also an important factor for developing an at-risk growth trajectory. Also, among White children, eating fast food placed them at a higher risk of developing an at-risk growth trajectory. Besides this factor, there were no other factors that differed by race/ethnicity, after adjusting for demographic variables. The factors tested have been previously found to be associated with overweight/obesity; however, in this study many of these risk factors do not place children at a higher risk of developing at-risk growth trajectories. To summarize, this hypothesis was partially supported. There were different predictors of at-risk trajectories for

White children and Black and Latino children. However, the variables tested did not differ for Black and Latino children. See table 4.31 for a summary of the hypotheses and the results.

Table 4.31: Summary of Hypotheses and Analytic Strategy

Hypotheses	Results
1a. Maternal depression over the first three years of the child's life is associated with higher BMI z-score when the child is 9 years, compared to children of mothers with no maternal depression.	1a. Not supported. Maternal depression at W2 and/or W3 was not associated with BMI z-score at age 9 (W5).
1b. Maternal depression over the first three years of the child's life is associated with overweight (OW) and obesity (OB) status when the child is 9 years, compared to children of mothers with no maternal depression.	1b. Not supported. Maternal depression at W2 and/or W3 was not associated with OW or OB status among children at age 9 (W5).
2. Child growth patterns will follow multiple trajectories.	2. Supported. The best fitting model had 3 growth trajectories; stable, mid-rising, and high-rising trajectories.
3. Maternal depression over the first three years of the child's life will be associated with at-risk growth trajectory development.	3. Not supported. Maternal depression was not associated with child growth trajectories.
4. Fewer hours of sleep and outdoor play and less fruit and vegetable consumption; and more television viewing, fast food and soda intake will be associated with at-risk growth trajectory development.	4. Not supported. None of the child-level factors were associated with at-risk trajectory development.
5. Lower levels of maternal involvement and breastfeeding for less than 6 months will be associated with at-risk growth trajectory development.	5. Not supported. Neither breastfeeding duration nor maternal involvement were significantly associated with at-risk trajectory development.
6. Trajectories of growth will differ by race/ethnicity.	6. Supported. The group-based trajectory solutions differed by race/ethnicity. Latino children had a high-rising trajectory. Black children had a mid-rising trajectory. White children had two trajectories; a high-rising and a mid-stable trajectory.
7. Significant predictors of the most at-risk trajectory will differ by race/ethnicity.	7. Partially supported. There were different predictors of at-risk trajectories for White children and Black and Latino children. However, the variables tested did not differ for Black and Latino children.

CHAPTER 5: DISCUSSION

This last chapter offers an interpretation of the results and integrates the study findings with current literature. This chapter will also discuss the strengths and the limitations of the study, the implications for theory, practice, and policy, and it will end with an overview of future research directions.

Maternal Depression and Childhood BMI Z-Score and Overweight/Obesity Status

Initially, I hypothesized that maternal depression, either when the child was one or three and also at both time points, would be associated with child BMI z-score and also with overweight/obesity status. However, contrary to my hypothesis, a general linear model regression showed no association between maternal depression and child BMI z-score. Moreover, a logistic regression also demonstrated that maternal depression did not increase the odds of overweight/obesity when children were 9 years of age. Although this hypothesis was not supported, the results from this study contribute to the mixed literature on maternal depression and childhood obesity. On the one hand, the results from this study are in line with a study of 4,287 children from Brazil (Santos, Matijasevich, Domingues, Barros, & Barros, 2010). In the Brazilian study, investigators gathered information on maternal depressive symptoms when the child was 1, 2, and 4 years of age using the EPDS. Mothers were categorized as never having elevated depressive symptoms, having elevated depressive symptoms at one or two time points, and having elevated depressive symptoms at all time points. Children's anthropometric measures included weight-for-age, weight-for-height, and height-for-age when they were 4 years of age. Their fully adjusted findings suggested that there was no association between maternal depression and any of the anthropometric measures gathered (Santos et al., 2010). On the other hand, the results from this study contradict findings from other studies that have found

significant positive associations such as the study by Gross and colleagues (2013). In said study, the researchers found that children of mothers with at least moderate depressive symptoms had 2.62 times the odds of being overweight/obese, compared to children of mothers without depressive symptoms (Gross et al., 2013). Below I discuss some potential reasons for the results found in this study.

Although previous studies have found null results on the association between maternal depression and childhood overweight/obesity, there are other studies that have found significant associations in US samples (e.g. Gross et al., 2013; Surkan, Kawachi, & Peterson, 2008; Wang et al., 2013). One potential reason why I did not find a significant association between maternal depression and childhood obesity was the measurement used. Most previous studies have used depressive symptomology as the measure of depression. In this study I used a measurement that is closer to depression diagnosis (CIDI-SF) than it is to depressive symptomology, thus providing a more rigorous analysis of the consequences of major maternal depression. Although the CIDI-SF is a strong and rigorous measure of depression, it does not provide information on severity of depressive symptoms, as previous studies have done. Because of the type of measurement used in my study, it is possible that only those women with severe symptoms were identified by the screening, leaving out women with mild or moderate symptoms. This is not a limitation per se, but it is necessary that more studies are conducted using depression diagnostic tools that can shed light on how *maternal depression* is influencing child development.

Another possible reason why I did not find an association between maternal depression and child BMI z-score or obesity/overweight status at age 9 is timing. This particular study investigated the effect that maternal depression during the first three years of life had on child BMI z-scores during elementary school years. Because there are about six years between

measurements, it is impossible to determine how maternal depression changed over the span of those years and the potential effect it had on child BMI. For instance, it is possible that maternal depression resolved during these years and the influence of maternal depression on children was ameliorated. This theory can be supported by cross-sectional studies that demonstrate a positive association between maternal depression and child BMI. For instance, a recent cross-sectional study used data on 2,965 children from the FFCWS at age 9 to determine the association between maternal depression and child overweight status (Dow-Fleisner & Hawkins, in print). This study found that maternal depression measured at age 9 increased children's odds of being overweight at age 9, after controlling for multiple demographic and health behaviors (Dow-Fleisner & Hawkins, in print). Another study, also cross-sectional, among 401 5 year-old children found a positive association between maternal depressive symptoms and childhood overweight and obesity (Gross et al., 2013). Specifically, the study found that children of mothers with moderate/severe depressive symptoms had 2.62 times the odds of being overweight and obese at age 5. Thus, it is possible that the repercussions that maternal depression has on childhood overweight and obesity are short-term, rather than long-term.

A third possible reason why this study did not find an association between maternal depression and child BMI z-score or overweight and obesity is that it is difficult to predict the effect that maternal depression has on the children, particularly because not many mechanisms explaining this association have been tested. In fact, there are a few studies that have found that maternal depressive symptoms are associated with lower BMI and lower weight gain (Duarte et al., 2012; Surkan et al., 2014; Wojcicki et al., 2011). For example, a study using data on 181 Latino children found that having depression during the antenatal and postpartum periods was associated with a lower weight-for-length z-score when the child was two years of age (Wojcicki

et al., 2011). Another study that used data from the Early Childhood Longitudinal Study-Kindergarten (ECLS-K), found that severe maternal depressive symptoms when the child was in kindergarten was associated with lower BMI z-score when the child was in third grade. This was only true among girls. Overall, these findings suggest that maternal depression might not automatically suggest higher BMI; the opposite might be true as well. In order to better understand how maternal depression is potentially impacting child health, it is necessary to look more into the mechanisms that can explain this association. Future studies should aim to understand the obesogenic behaviors that are associated with maternal depression.

A final potential reason is that indeed there is no association between early maternal depression and childhood obesity. This study particularly focused on maternal depression during the first three years of the child's life and subsequent childhood obesity (age 9). Thus, it is possible that the long-term consequences of maternal depression are not reflected on child's adiposity and BMI. Instead, it is possible that early maternal depression has repercussions that are more evident through children's behaviors than through their body composition. After all, studies that have found a positive association between maternal depression and childhood obesity have yet to explain the mechanisms for this association. In addition, not providing a theoretical or empirical explanation of the association between maternal depression and childhood obesity is a disservice to mothers. This lack of explanation is often (incorrectly) filled with finger-pointing at the mothers, blaming them for everything that happens to their children. It is then necessary to investigate the mechanisms that explain the association between maternal depression and childhood obesity to provide a more thorough understanding of the repercussions that maternal depression has on children. As mentioned earlier, future studies need to investigate how maternal

depression can influence children's behaviors, which can in turn increase the chances for developing childhood obesity.

Child BMI Z-Scores Group-Based Trajectories

The second research question aimed to determine whether the data followed a single or multiple trajectories. As expected, the hypothesis that the data followed multiple trajectories was supported. Previous studies investigating child growth have also found multiple group trajectories, the majority have also found that a three-group solution was the most effective (Garden, Marks, Simpson, & Webb, 2012; Li, Goran, Kaur, Nollen, & Ahluwalia, 2007; Pryor, Tremblay, Boivin, & et al., 2011; Rzehak et al., 2017). For instance, a longitudinal study using anthropometric data on 370 children collected annually from birth to 11.5 years found a three-group trajectory solution (Garden et al., 2012). The authors ran different models for boys and girls and each group had a three-group trajectory solution. Among girls, the trajectories found by this study included a late increase group, which showed an increase in BMI after age 8; an early and persistent group, which had an increase in BMI after age 3; and a normal group, which showed normal growth over time (Garden et al., 2012). In this study, among girls the normal group included 62% of the sample, 12% of girls followed an early and persistent trajectory, and the late increase group included 26% of the sample (Garden et al., 2012). The trajectories found for boys were similar; 61% of boys followed a normal trajectory; 12% followed an early and persistent increase trajectory; and 27% followed a late increase trajectory (Garden et al., 2012). Another study using data on 1739 children from the National Longitudinal Survey of Youth 1979 found a three-group trajectory among children ages 2-12 (Li et al., 2007). The study found that 83.9% of the sample displayed a normal growth trajectory; whereas 10.9% displayed an early-onset growth trajectory and 5.2% showed a late-onset trajectory (Li et al., 2007). Another study

used data from 1,957 children from the Quebec Longitudinal study of child development (Pryor et al., 2011). The results demonstrated a 3-group trajectory modeling with a low-stable group, characterized by 54.5% of the sample; a moderate group, made up by 41.0% of the sample; and a high-rising group, characterized of 4.5% of the sample (Pryor et al., 2011).

Although the findings from the current study identified a three-group trajectory in the full sample – similar to previous research – the distribution of group membership was different from previous studies. This study found a high-rising group that was made up of 20% of the sample, which is unusually high. Previous studies have identified groups with risk trajectories that were characterized by no more than 12% of the sample (e.g. Li et al., 2007; Pryor et al., 2011; Rzehak et al., 2017). The differences might be due to the metric used by this study. Previous studies that have found a high-risk trajectory with a smaller percentage have used raw BMI score. On the contrary, this study used BMI z-score as the outcome. Using a different metric might present a different solution. Another explanation for the discrepancy on these findings is the population studied. Previous studies using group-based trajectory modeling or growth mixture modeling (the analogue of group-based trajectory modeling using MPlus software), have been conducted using data predominantly from White middle income participants (Li et al., 2007; Pryor et al., 2011; Rzehak et al., 2017). However, the current study used data from a predominantly low-income, minority sample and found that 42.63% of children at age 9 were either overweight or obese. Nationally, the rate of overweight and obesity among children between the ages of 6 through 11 is 34.2% (Skinner et al., 2018). The rates found in this study are much higher than they are at the national level. This suggests that in a predominantly low-income minority group, the rates of overweight and obesity are more concerning. Thus, low-income minority groups should be given more attention from researchers and health policy makers.

Maternal Depression and its Association with Growth Trajectories

The third hypothesis was related to maternal depression and its association with high-risk growth trajectories. Chi-square tests and logistic regression demonstrated that this hypothesis was not supported. The findings showed that there was not an association between maternal depression at any point or between recurrent maternal depression and at-risk growth trajectories. These results are surprising given that there is evidence suggesting an association between maternal depression and childhood obesity over time. For example, a longitudinal study using data on 1,090 children from the National Institute of Child Health and Human Development Study found that children of mothers with elevated depressive symptoms at three time points had 2.13 times the odds of being overweight over time, compared to children of mothers without depression (Wang et al., 2013).

One possibility for the discrepancy on the findings is that the study by Wang and colleagues measured maternal depressive symptoms when the children were 1, 2, and 3 years of age. On the other hand, my study only measured depression at two time points; 1 and 3 years. Thus, having recurrent depressive symptoms over three years might have a different effect than having recurrent depression two times in a row. Future studies should aim to address this issue by measuring depressive symptoms/depression over longer periods of time to really understand the effect that recurrent depressive symptoms/depression can have on children's BMI.

A second reason as to why this study did not find an association between maternal depression and child growth trajectories is that children in this low-income sample might have multiple sources of stress, including more systemic issues that are masking the effect of maternal depression. The sample of this study consisted of predominantly low-income minority children. For example, many of the children in this sample had mothers who were head of household and

worked a long number of hours and nonstandard shifts. A study investigated the effect that maternal work shifts had on children's overweight and obesity using data from FFCWS (Miller & Chang, 2015). The authors found that children of mothers who worked a standard primary shift and a nonstandard (e.g. evening or rotating shifts) secondary shift had a 0.09 increased probability of being overweight or obese, compared to children of mothers who only worked standard shifts (Miller & Chang, 2015). Another study also using data from FFCWS investigated the cumulative effect that intimate partner violence, food insecurity, housing insecurity, maternal substance use, depression, and paternal incarceration had on children's overweight and obesity status at age 5 (Suglia, Duarte, Chambers, & Boynton-Jarrett, 2012). The authors found that girls who had two or three risk factors had 1.77 times the odds of being obese and girls with three or more risk factors had 1.89 times the odds of being obese at age 5 (Suglia et al., 2012). These findings demonstrate that children in this sample experience many adverse conditions in life; thus, it is possible that other more prevailing conditions or the accumulation of them place children at risk for overweight and obesity, rather than maternal depression alone.

A third potential reason for not having found a significant association between maternal depression and child growth trajectories is that I did not separately investigate growth trajectories for males and females. Previous studies have demonstrated that there can be different effects by sex. For instance, the study by Suglia and colleagues (2012) mentioned above found that cumulative social risk factors were associated with obesity only among girls. It is possible that by conducting a separate analysis by sex, I would have seen differences. However, because I used BMI z-score as the outcome variable to identify the trajectories, conducting a stratified analysis by sex would have been redundant since the BMI z-scores are already adjusted for sex.

To further control for sex, the trajectories were adjusted for sex. However, future studies should stratify by sex and determine whether there are different outcomes for boys and girls.

A final potential reason is that the findings are accurate and early maternal depression does not have an impact on child's BMI. This study tested all different ways in which maternal depression could be associated with child BMI z-scores. This study used different methodologies; general linear models, logistic regression, and a stringent analysis of growth patterns over time and no association was found between early maternal depression and childhood overweight/obesity or growth trajectories. Therefore, it is possible that in this low-income mostly minority sample, early maternal depression does not increase the odds of overweight/obesity risk. Although no other study has predominantly focused on testing the association between maternal depression and child growth trajectories, the most similar study used mixed effects models to track the mean BMI trajectory of children from 9 months to 6 years (Surkan et al., 2014). In the study, Surkan and colleagues (2014) used data from 6,550 children from the Early Childhood Longitudinal Study (ECLS-B). Authors gathered information on maternal depressive symptoms when the child was 9 months of age and examined its association with the trajectory of child mean BMI from 9 months to 6 years (Surkan et al., 2014). The fully adjusted models showed that neither mild nor moderate/severe maternal depressive symptoms were associated with child BMI to age 6 (Surkan et al., 2014).

Two other studies that are somewhat similar to the present study used child growth trajectories to identify the role that maternal depression played as a control variable (Pryor et al., 2011) and as a mediator (Lane, Bluestone, & Burke, 2013). The study by Pryor and colleagues (2011) aimed to determine risk factors for at-risk trajectories during early childhood. A three-group trajectory solution was found, and a high-rising risky trajectory was identified. The

researchers measured maternal depressive symptoms when the child was five months and found that elevated depressive symptoms were not associated with the high-rising trajectory (Pryor et al., 2011).

Another study using BMI data on 1,238 children from ages 2 to 11 investigated the mediating role of elevated maternal depressive symptoms using a modified scale of the CESD (Lane et al., 2013). The researchers found a three-group trajectory solution; a stable, a steady increase, and an elevated group. It was found that maternal depression was not a predictor of the elevated group (Lane et al., 2013). However, it was also found that in the stable class, children of mothers who had elevated depressive symptoms had higher BMI, compared to children who were in the stable class but whose mothers did not have depressive symptoms (this was a within-class analysis). Similar to the findings from these previous studies, I did not find that maternal depression was associated with membership in the high-rising group.

Although the findings from this study suggest that there is no association between maternal depression and childhood growth patterns of overweight and obesity over time, it is important not to rule out maternal depression as a risk factor altogether. First of all, it is necessary to bear in mind that the scholarship on maternal depression and childhood obesity development is still in its infancy and there are mixed findings. Thus, it is necessary to carry out more studies to understand for whom and under which conditions maternal depression may have repercussions on children. More importantly, it is necessary to identify the sensitive windows when maternal depression can be a risk factor for childhood development. For example, it is possible that maternal depression during the first three years of life does not impact childhood obesity development, but maternal depression later in life might have a stronger impact on child's health. Second, it is necessary that future studies focus on better understanding maternal

depression and its relationship to other obesogenic behaviors, such as sleep, fast food consumption, and soda intake, among others. Although BMI is considered an objective measure of childhood adiposity and growth, it is not the perfect measure (Burkhauser & Cawley, 2008; Freedman, Wang, Thornton, & et al., 2009). Studies have argued against the use of BMI as the only outcome measurement because it favors body types from certain racial/ethnic groups such as Asian and White body types (Jackson, Ellis, McFarlin, Sailors, & Bray, 2009). Thus, BMI may not provide the most objective or culturally appropriate measurement. Instead, focusing increasingly on obesogenic behaviors may have more relevance to health outcomes. The majority of the literature on maternal depression has focused on understanding the effects it has on children's internalizing and externalizing behaviors (Ewell Foster, Garber, & Durlak, 2008). However, when it comes to children's health status, particularly overweight and obesity, there is very little literature on maternal depression and child obesogenic behaviors as the outcomes. Thus, whenever available, studying the effect of maternal depression on children's behaviors would be recommended.

Child-Level Factors Associated with Growth Trajectories

Surprisingly, this study found that child-level factors; television viewing, outdoor play, sleep, and dietary intake, were not associated with group trajectory membership in the overall sample. Although these child-level factors have previously been found to be associated with overweight/obesity, in this study many of these risk factors do not place children at a higher risk of developing an at-risk growth trajectory. Below is a discussion of each child-level factor.

Television viewing

Although there is robust evidence to suggest that television viewing is associated with childhood overweight and obesity (Zhang et al., 2015), the current study did not find an

association. Among the most compelling evidence of the association between television viewing and childhood obesity is a recent meta-analysis of 14 studies that found 47% increased odds of childhood obesity among children who watched the most amount of television, compared to children who watched the least amount (Zhang et al., 2015). Another study used data from 7,334 children to investigate the association between growth trajectories and television viewing among children between the ages of 5 and 11 (Danner, 2008). The unadjusted results demonstrated that hours of television were positively associated with BMI increase over time and also with BMI acceleration. However, after controlling for demographics and birth weight, only BMI acceleration remained positively associated with television viewing (Danner, 2008).

One potential reason for the lack of association between television viewing and group trajectory membership is that information is only available until age 9, since it is possible that the consequences of sedentary behaviors do not become evident until later in life. In fact, previous studies have not found associations between television viewing and childhood overweight and obesity among young children. A cross-sectional study using data on 3,141 children from FFCWS investigated the association between television viewing and childhood obesity among 5 year old children (Burdette & Whitaker, 2005a). This study found that television viewing was not significantly associated with childhood obesity (Burdette & Whitaker, 2005a). Another cross-sectional study using data on 354 preschool-aged children investigated the role of television viewing on child BMI and on dietary intake (Harrison, Liechty, & the STRONG Kids Program, 2012). Harrison and Liechty found no association between television viewing and child BMI among preschool-aged children. However, they did find that television viewing was associated with increased consumption of high-energy, low nutrient foods such as candy and soda and also with decreased intake of fruits and vegetables (Harrison, Liechty, & the STRONG

Kids Program, 2012). These previous studies suggest that an association is not evident among young children; but an association might become clear later in life and having information after age 9 would help investigate this issue. Thus, it is important to use longitudinal data that follows infants and children into adulthood to identify the age at which the effect of obesogenic behaviors on BMI becomes evident.

Another possible reason for the lack of association between television viewing and childhood obesity has to do with the information that was encompassed by this measure. In this study, over 70% of children ages three and five watched television for more than two hours per day. However, it is not clear how television viewing was measured. Especially in a time when there is so much technology available, it is not clear whether this question encompassed information regarding computer time, or other screen time. This is important because overall screen time can provide a more stringent measurement of sedentary activity among children, which in turn can provide more accurate information on childhood obesity development. Future studies that collect primary data should try to use a more holistic measurement of screen time.

Outdoor play

This study did not find an association between outdoor play and growth trajectory membership. This adds to previous mixed results from this dataset. Although a previous cross-sectional study found an inverse association between outdoor play and child BMI using data from 5 year-old children from FFCWS (Kimbro, Brooks-Gunn, & McLanahan, 2011), another study using the same dataset, but looking at 3 year-old children did not find such an association (Burdette & Whitaker, 2005b). There are different possibilities that can explain the lack of significant findings. First, it is possible that an association only becomes evident in cross-

sectional studies, rather than longitudinally. Finding longitudinal associations implies that the effects of outdoor play are persistent across time, which might not be the case for outdoor play.

The second potential reason is that outdoor play may not be a good proxy measure for physical activity. Although a study by Burdette and colleagues among 250 mostly White preschool-aged children (2004) found that physical activity was highly correlated with parental report of outdoor play during preschool years, it is possible that as children grow older, the outdoor play measurement is no longer valid. As children grow older they may spend more time playing outside on their own or with school friends and less time playing with their parents. Thus, a more stringent measurement of physical activity, such as accelerometers can be used in future studies to better understand the role of physical activities on growth patterns into middle childhood.

A third possibility for not finding a significant association between outdoor play and group membership is that I did not stratify by sex for reasons outlined earlier. Previous studies have found that the association between physical activity and BMI is different for boys and girls (Fernandes & Sturm, 2011; Riddoch et al., 2009). For example, a longitudinal study on 11,841 children investigated the association between physical activity at age 12 and fat mass at age 14 (Riddoch et al., 2009). The study found that for every 15 minutes of moderate-vigorous activity, boys had 12% less fat mass and girls had 10% less fat mass (Riddoch et al., 2009). It is possible that by not stratifying the data by sex, significant effects of outdoor play on growth trajectories were not observed.

The last possibility for not finding an association between outdoor play and group trajectory membership is that there is no *longitudinal* association between outdoor play and childhood BMI. To the best of my knowledge, this is the first study investigating the association

between outdoor play and growth trajectories. However, other studies have investigated other physical activity measurements (Fernandes & Sturm, 2011). For example, a study using data on 8,246 children examined the association between recess and physical education at school and BMI percentile from first to fifth grades (Fernandes & Sturm, 2011). The results from this study found that each additional hour of recess was associated with BMI percentile decreasing by 0.30 units (Fernandes & Sturm, 2011). Moreover, it was found that following the national guidelines for physical education was associated with a 1.56 unit BMI percentile decrease among boys.

Sleep

Although previous studies have found a significant inverse association between sleep and child BMI, this study did not find an association. To date, the most robust evidence demonstrating an association between sleep and child BMI comes from a recent systematic review of longitudinal studies (Magee & Hale, 2012). The systematic review compiled evidence from seven longitudinal studies among children under the age of 12 and found that sleeping for shorter duration predicted weight gain in children (Magee & Hale, 2012). Similarly, a more recent study using longitudinal data from preschoolers found that children who slept fewer hours at night had higher BMI percentile, demonstrating that sleep is an important predictor of child BMI and the repercussions of lack of sleep can be observed early in life.

One potential reason for the lack of significant findings in the present study is the time when sleep was measured. This study measured the number of hours of sleep when the child was 5 years of age (wave 4), which might have been too late to identify any effect on the growth trajectories. Although innovative techniques such as GBTM provide a reliable and accurate way to analyze longitudinal data, identifying predictors of group membership can be cumbersome. One reason it might difficult to identify predictors or factors that are associated with growth

trajectories is that they have to be measured as close to the start of the trajectory as possible or they also need to be measured at the same time points as the outcome measure. In this study however, the first time that the number of hours of sleep was reported for the child was at age 5 (wave 4), which might already be too late for finding associations. When using longitudinal data, future studies should make sure to use datasets that have recurrent measures available on the independent variables that need to be analyzed.

Another potential reason for the lack of association between sleep and group membership is that there is no true association. A study of 1,079 children used growth mixture modeling to identify different growth trajectories among children between the ages of 4 and 11 years (Magee, Caputi, & Iverson, 2013). The authors found a three-group solution; a healthy weight trajectory, an early onset obesity, and a later onset obesity trajectory. Hours of sleep were measured annually at the same time as child BMI. The results demonstrated that sleeping was inversely associated with the early onset obesity trajectory (Magee et al., 2013). However, there was no relationship found between sleep and the later onset obesity trajectory. The results from Magee et al. (2013) demonstrate that when it comes to the effect that sleep has on children's growth trajectories, sleep may not be a significant factor for all the at-risk trajectories.

Dietary intake

This study examined separately the effect of three different dietary behaviors; soda intake, fresh food and vegetable consumption, and fast food consumption. These three variables have been found to be independently associated with overweight and obesity among children (DeBoer et al., 2013; Malik et al., 2006). However, in this study I did not find any of these variables to be associated with the high-risk trajectory. It was particularly surprising to find that soda was not significantly associated with group membership because previous studies have

found a strong association (DeBoer et al., 2013). For soda intake, for example, a longitudinal study investigated the association between sugar-sweetened beverages (SSBs) and weight gain (DeBoer et al., 2013). This study found that compared to children who had less than one SSB per day, those who had at least one SSB per day had a higher BMI z-score at ages 4 and 5 (DeBoer et al., 2013). It is important to mention that soda was marginally not significant ($p = 0.05$; 95% CI = [0.99 – 1.19]). It is possible that the results were not significant because I used soda as a proxy for SSBs, rather than using a measurement that encompassed more sugary drinks, such as juice. However, the problem with including juice consumption is that it is impossible to determine whether it is 100% fruit or not; thus, the effect might be confounded. Future studies should try to discern the effect that sugary drinks and juice have on the development of childhood obesity.

The evidence for fast food and fruit and vegetable consumption is not as strong as it is for SSBs. Fast food has usually been thought to be a contributor of higher BMI and obesity because of its high caloric content (Boutelle et al., 2006; Bowman et al., 2004; Harrison & Liechty, 2012). On the other hand, fruits and vegetables have been perceived as a protective factor against childhood obesity since they are a healthy and filling food source (Makris & Foster, 2011). However, the literature around the association between fast food, fruit and vegetable consumption, and childhood obesity is not too robust. In fact, there are few studies that have investigated the association between fast food consumption and childhood obesity. The majority of studies use proximity to fast food restaurants as a proxy for fast food consumption (Currie, DellaVigna, Moretti, & Pathania, 2010). This study provides a measure of fast food consumption reported by the parents, which might provide better information than proximity to fast food outlets. Regarding fruit and vegetable consumption, there is also limited support to definitively

argue that eating more fruits and vegetables is associated with lower likelihood of overweight/obesity. In fact, a recent study comparing BMI trajectories by race/ethnicity found that neither fruit nor vegetable consumption was predictive of BMI growth for any racial/ethnic group (Guerrero et al., 2016). Thus, it is possible that no association exists between fast food or fruit and vegetable consumption and child growth trajectories.

It is also possible that an association between dietary intake and trajectory membership was not found in this study because all three variables were only available at age 5 (wave 4). It is possible that measuring behaviors this late in time might mask the true effect that they have on the development of childhood obesity. Whenever available, future studies should focus on using behaviors measured close to the start of the trajectory or use behaviors that are measured over time.

Clan-Level Factors Associated with Growth Trajectories

This study also tested the effect that clan-level variables – maternal involvement and breastfeeding duration – had on child trajectory membership. These variables aimed to tackle the emotional relationship and the bonding that was created between the mother and the child. I hypothesized that by having more bonding with the child through activities that foster emotional attachment, the child would be less likely to develop an at-risk growth trajectory. For instance, a study of 977 children investigated the effect that early maternal-child relationships had on the child at age 15 and found that obesity was more prevalent among children who had poor relationships with their mothers during the first 3 years of life (Anderson et al., 2011). Scholarship on breastfeeding and childhood obesity is even stronger, suggesting a strong inverse association between breastfeeding and childhood obesity (Yan et al., 2014). A meta-analysis of 25 studies showed that children who were breastfed for 7 months or more had a lower risk of

being overweight or obese (Yan et al., 2014). Although this previous literature suggests an association between clan-level factors and childhood obesity, this study did not find an association. One possibility is that although the measurement of maternal involvement was aiming to tap into the emotional relationship between the mother and the child, this measurement might not have captured emotional connection in its entirety. Previous studies using FFCWS data have also used this measurement to identify maternal involvement (Choi & Jackson, 2011; Walters, 2014). However, this measurement was adapted from a measure that was used in the Early Head Start Parent Interview, so it is not a validated instrument used to assess maternal involvement or attachment and it is possible that it captures other behaviors besides mother-child emotional connectedness.

As mentioned earlier, the literature on the association between breastfeeding duration and low risk of childhood obesity is robust. The fact that an association was not found in this study was surprising. However, there are a couple of reasons why an association was not observed in the study. First, the measurement used in this study addressed breastfeeding duration only, rather than breastfeeding duration accompanied by breastfeeding exclusivity. It is possible that breastfeeding duration alone did not have an effect on child BMI. In fact, previous studies have found that breastfeeding exclusivity is a strong predictor of childhood obesity (Musaad, Donovan, Fiese, & the STRONG Kids Team, 2016). For instance, a recent longitudinal study of 351 mother-infant dyads investigated the association between exclusive breastfeeding for the first 3 months and change in child weight-for-length z-score (WFLZ) from birth to 12 months (Musaad et al., 2016). This study found that not breastfeeding exclusively by month 3 was associated with larger change in WFLZ (Musaad et al., 2016). This study also used growth trajectories and found that children who were not exclusively breastfed by the third month had

2.24 times the odds of developing a low-rising (at-risk) trajectory instead of a mid-stable trajectory (Musaad et al., 2016). Whenever possible, future studies should test the role of breastfeeding exclusivity, in addition to breastfeeding duration.

The second potential reason for not finding an association between breastfeeding duration and child at-risk growth is that there is in fact no association over time. Two studies using latent growth mixture modeling (Garden et al., 2012; Rzehak et al., 2017), have found discrepant results on the association between breastfeeding and child BMI trajectory. The study by Garden and colleagues, based on a sample of Australian children with highly educated parents (2012), found a three-group trajectory solution for BMI from birth to 11.5 years. Different trajectories were fit for boys and girls. The results demonstrated that breastfeeding for six months or more was not associated with BMI trajectory group membership, either among boys or girls (Garden et al., 2012). However, a more recent study combined data predominantly from participants with high levels of education living in different European countries to investigate the relationship between infant feeding and growth trajectories (Rzehak et al., 2017). The authors found a three-group trajectory solution for the data and found that children who were exclusively breastfed for less than three months were more likely to develop rapid-growth trajectories than they were of developing a normal-growth trajectory (Rzehak et al., 2017). This information suggests that the effects of breastfeeding duration and exclusivity on children's growth *over time* is not conclusive. Future studies should continue investigating the relationship between breastfeeding initiation, duration, and exclusivity longitudinally. Moreover, although the cut-point of breastfeeding for 6 months or more has been the recommendation, other cut-points need to be explored as well. In fact the studies by Musaad (2016) and Rzehak (2017) suggest that exclusive

breastfeeding for 3 months or more can have a stronger effect on child BMI than nonexclusive breastfeeding for longer periods of time (Garden et al., 2012).

Overall, from this study I found that based on a national sample of low-income unmarried mothers, there is no association between child-level and clan-level factors with child trajectory membership. It is difficult to determine whether these associations were not significant because of a measurement or timing issue or because there is no association longitudinally. Only a few studies have investigated the effects that child-level and clan-level variables have on child *growth trajectories*. Although latent growth mixture modeling and group based trajectory modeling offer innovative ways to analyze longitudinal data, especially BMI data, it is not widely used by researchers in the field of childhood obesity. The variation in findings between this study and that of cross-sectional studies and the additional insights gained shows that this method should be considered more for future research.

Child BMI Z-Score Growth Trajectories Stratified by Race/Ethnicity

Previous research has demonstrated that childhood overweight and obesity disparities start early in life (Guerrero et al., 2016; Ogden et al., 2014). This study aimed to determine whether there were different growth patterns from birth to 9 years of age by race/ethnicity. Given the literature suggesting that childhood obesity disparities can be observed at different ages, it was hypothesized that the trajectories of growth would be qualitatively different. The results demonstrated that the best trajectory that fit White children was a three-group solution, and for Black and Latino children, the best solution was a two-group trajectory. To the best of my knowledge, this is the first study that fits separate growth trajectories for each racial/ethnic group using GBTM (or LGMM). The closest study found was one conducted by Guerrero and colleagues (2016). In this study the researchers used data on 15,418 children at ages 4, 5, and 6

from the ECLS-B to identify growth trajectories by race/ethnicity (Guerrero et al., 2016). In that study the authors identified growth trajectories among White, Black, Asian, Latino from English-speaking households, and Latino children from Spanish-speaking households. The purpose of the study was to examine average growth patterns by racial/ethnic group, so only one trajectory was fit for each group using a growth curve of mean BMI (Guerrero et al., 2016). The results showed that Latino children from Spanish-speaking households had a higher BMI at age 4 and their BMI remained high over time, compared to White children. Similarly, Black children started with higher BMI than White children at age four, and their BMI remained higher, but not as high as that of Latino children from Spanish-speaking households (Guerrero et al., 2016). Similar to these findings, the current study found that some Latino children have high BMI early in life, but not all. This study also found that higher BMI among Latino children can be found as early as birth. However, contrary to the findings by Guerrero and colleagues (2016), Black children in the current study did not show a high BMI at the start point. It is possible that the socioeconomic disadvantage of the sample used by the current study drives those different findings.

Another similar study was carried out using ECLS-B data on 7,200 predominantly White children and it aimed to identify growth trajectories among kindergarten children in the US (Isong, Richmond, Avendaño, & Kawachi, 2017). The study used mean BMI z-score to identify different trajectories among White, Latino, African-American, Asian, American-Indian, and Pacific-Islander children (Isong et al., 2017). The researchers ran different models for each sex. The results showed that among females, Black girls start with a lower BMI z-score than White girls, but by age 3 their BMI z-score surpasses that of White girls. American Indian and Pacific Islander girls started with the highest mean BMI z-scores of any other group, and their trend remained high until age 6. Among boys, Latino and American Indian children started with the

highest BMI z-score. However, among American Indian boys, the trend went down and stabilized at 45 months, whereas the trend among Latino boys continued increasing until age 6. This study further corroborates that there are disparities in childhood obesity by race/ethnicity that start very early in life.

Previous studies have suggested that there are different growth patterns by race/ethnicity. However, previous studies have not applied advanced methods to fit different group trajectories by race/ethnicity, nor have they looked at differences at birth. This study fills in this gap by providing group trajectories by race/ethnicity starting at birth. This statistical method has the benefit of highlighting variation within minority groups, rather than assuming the "average" pattern reflects the group accurately. Thus, this study can help differentiate normal growth versus unhealthy growth in different racial/ethnic groups and it can also help identify some of the early risk factors that are associated with at-risk trajectories.

Predictors of the Most At-Risk Trajectory by Race/Ethnicity

I also stratified by race/ethnicity, and found two differences in the predictors associated with membership in the at-risk trajectory. Since each of these factors have been discussed thoroughly during the discussion of hypotheses 4 and 5, in order to avoid repetition, the section below will summarize the findings for the child-level factors and the clan-level factors regarding their association with trajectory group membership and any differences by race/ethnicity.

Overall, this study found that among White children the only child-level factor associated with at-risk trajectory membership was fast food consumption. Among White children, none of the clan-level factors were associated with developing an at-risk trajectory. One of the most surprising findings was that among Black and Latino children, *none* of the child-level or clan-

level factors were associated with at-risk trajectory membership in this national low-income sample.

Contrary to what was expected, viewing television for two hours or more was not associated with an increased risk of developing an at-risk trajectory among any of the racial/ethnic groups. Previous studies have found that television viewing is strongly associated with childhood obesity, particularly among minority children. For example, a prospective study of 1,826 children found that having a television in the bedroom increased the odds of being obese by 7.7 times among Black children and by 7.9 times among Latino children by age 4 (Taveras et al., 2010). One possibility for not finding significant associations is that the association between television viewing and childhood overweight or obesity is only observed in cross-sectional studies. For example, a longitudinal study using data from 2,379 adolescent girls between the ages of 11 and 14 used BMI trajectories to identify the association between television viewing and BMI trajectories (Henderson, 2007). The study found that among White girls, more hours of television viewing were associated with a steeper BMI trajectory over time (Henderson, 2007). No associations were found among Black girls. These findings suggest that longitudinal associations using growth trajectories might be more difficult to detect than cross-sectional associations.

Contrary to expectations, sleeping less than the recommended amount of hours was not significantly associated with at-risk trajectory membership among any of the racial/ethnic groups. A previous study investigating the early risk factors for childhood obesity identified that Black and Latino children below the age of 2 years who slept less than 12 hours per day had 3.7 and 2.5 times the odds of being obese (respectively), compared to those children who slept the recommended amount of hours. One particular aspect of sleep, though, is that it might not only

be the number of hours that a child sleeps, but also the quality of sleep and having a specific time for bed (i.e. a routine) that can affect childhood obesity development (Spruyt et al., 2011). Thus, it is possible that by only measuring the number of hours of sleep, this study did not find significant associations. Future studies should use multiple measurements of sleep duration and quality that encompass more than one metric to assess sleep patterns among children.

It is possible that one of the reasons why I did not find any significant associations with child-level and clan-level factors among Latinos is that I did not stratify by nativity or language spoken at home. The study by Guerrero (2016) and colleagues identified different trajectories for Latino children from Spanish-speaking households and those from English-speaking households. Their results showed that Latino children from Spanish-speaking households had a trajectory of mean BMI that started high and remained high throughout the study (Guerrero et al., 2016).

It is surprising that dietary intake was not significantly associated with at-risk trajectory membership among Black and Latino children since previous studies have found an association (Guerrero et al., 2016; Taveras et al., 2010). A cross-sectional study of 1,826 children found that higher intake of SSBs and fast food increased the odds of obesity among Black and Latino children (Taveras et al., 2010). The study by Guerrero and colleagues (2016) also demonstrated that soda and fast food intake were associated with higher BMI trajectories. It is possible that this study did not find an association between soda and fast food consumption because they were only measured at age 5. On the other hand, the study by Guerrero (2016) had information on soda and fast food consumption at three different time points and was able to use these variables as time-varying, thus measuring their effect on the trajectories across time.

A final variable that was not associated with at-risk trajectory membership among any of the racial/ethnic groups was breastfeeding for six months or more. Although there is a large

amount of literature suggesting that breastfeeding duration and exclusivity protect children from different backgrounds from developing obesity, not much is known about the effects that breastfeeding has on children from specific racial/ethnic backgrounds. Only a couple of studies have investigated the associations between breastfeeding and childhood obesity by race/ethnicity (Burdette & Whitaker, 2007; Guerrero et al., 2016). A cross-sectional study used data from FFCWS on 2,146 children and found that Latino children who were breastfed had a lower prevalence of obesity, compared to Latino children who were not breastfed (Burdette & Whitaker, 2007). However, the association between breastfeeding and obesity was not found among White or Black children (Burdette & Whitaker, 2007). Another study using longitudinal data found that breastfeeding initiation was significantly associated with lower mean BMI score trajectories among White and Latino children from Spanish-speaking households (Guerrero et al., 2016). This relationship was not found among Black, Asian, and Latino children from English-speaking households (Guerrero et al., 2016). It is possible that in the present study an association between breastfeeding duration and child growth trajectories among low-income minority children was not found because in fact there is not one. However, it is highly possible that an association was not found in this study because I only had information available on duration rather than exclusivity. As mentioned earlier, previous studies have found that breastfeeding exclusivity can be a stronger predictor of childhood obesity and growth trajectories than breastfeeding duration (Musaad et al., 2016; Rzehak et al., 2017). Thus, whenever possible, future studies should include measurements on breastfeeding duration and exclusivity and determine whether there are differences in their association with growth trajectories by race/ethnicity.

Overall, the majority of child-level and clan-level factors were not associated with at-risk growth trajectories among children from different racial/ethnic groups. One potential reason for this lack of association in this low-income sample is that differences in child growth trajectories might be driven by more systemic issues. The sample in this study included low-income, mostly unmarried, and minority women. In fact, 60% of the sample lived under 200% of the federal poverty line; 74% of women were not married to the baby's father, and 77% were non-White women. Given the characteristics of the sample, it is possible that none of the child and clan-level factors were significant because these women and their children had more systemic and macro-level concerns that might play an important role in childhood obesity development. Thus, it is likely that some social determinants of health that were not tested in this study are driving some of these disparities in childhood obesity. Future studies should investigate the role that social determinants of health play in childhood obesity development among children from different racial/ethnic groups.

Study Strengths

This study had particular strengths worth mentioning. First of all, the literature on maternal depression has consistently relied on depressive symptoms. Although this study did not use an actual diagnosis of depression, it did provide probable depression caseness, which is a measure that is closer to depression diagnosis (Kessler et al., 1998). A second strength is that the population of study was a low-income mostly minority population. In fact, the largest racial group was Black children, followed by Latino children. Thus, this study provided a substantial sample size for groups that are usually underrepresented in the literature. A third strength of this study is the use of longitudinal data and methodology to investigate the relationship between maternal depression and child growth trajectories from birth to 9 years. Particularly, the use of

group-based trajectory modeling provides a novel and rigorous way to identify different growth patterns of BMI z-score over time. A fourth strength is investigating growth patterns within each racial/ethnic groups and whether there is a qualitatively different growth pattern by race/ethnicity. Although many studies have demonstrated disparities in child overweight and obesity by race/ethnicity, it is not well known how early these different growth patterns start. This study provides additional insight by creating different growth trajectories for White, Black, and Latino children. To the best of my knowledge, this is the first study investigating the relationship between early maternal depression and group membership and also the first to identify separate growth trajectories using group-based trajectory modeling for different racial/ethnic groups. Finally, this study controlled for pre-pregnancy BMI gathered from medical records. Pre-pregnancy BMI is a strong predictor of childhood obesity and the majority of studies cannot control for it; however, this study was able to do so, which strengthened the results. In fact, pre-pregnancy BMI is a stronger predictor than any of the factors tested here, thus not including this predictor can skew the analysis.

Study Limitations

This study also had several limitations worth mentioning. The first limitation is related to BMI measurement. The outcome of study was BMI z-score given that this measurement is commonly used in the literature and for longitudinal analysis (Wang & Chen, 2012). However, some researchers have proposed that other outcome measures of adiposity be used along with BMI, such as waist circumference or skinfold thickness in order to obtain a more accurate measurement of adiposity (Freedman & Sherry, 2009). One of the main limitations of the use of BMI is that it does not distinguish between muscle and bone mass; thus, some children are categorized as overweight or obese, when they should not be (Freedman & Sherry, 2009). Using

multiple measurements to increase reliability will always offer more accurate results; however, BMI information continues to be the most common measure collected and used to measure overweight and obesity, especially in large epidemiological studies. To mitigate the potential inaccuracy in this study, it is expected that using growth trajectories rather than cross-sectional BMI z-scores provided a more accurate measurement. This approach takes into account repeated measurements of BMI z-score and it develops groups based on growth patterns. Nonetheless, whenever available, researchers should use as much anthropometric information available to accurately identify body adiposity.

A second limitation is related to the number of repeated BMI measurements available. In growth trajectory modeling it is always better to have as many repeated measurements as possible in order to create accurate trajectories (Nagin, 2005); however, FFCWS did not collect anthropometric information at age 1 (wave 2), which would have strengthened the examination of growth trajectories. Also, there was no information available beyond age 9. Thus, there was information available from four different time points from birth to age 9. Having more measurements available would have been useful. The next wave of FFCWS contains information from children at age 15 and is coming out soon; thus, future studies should use that information to corroborate the trajectories found by this study.

A third limitation was the necessary use of two different metrics for weight status. For birth (wave 1) I used the WHO growth curves and for waves 3 through 5, I used the CDC growth curves. The decision to use different metrics follows the CDC recommendation to use the WHO growth curves before two years of age (CDC, 2017). Although it is the standard, it is possible that using different metrics in the same model could produce biased estimates.

A fourth limitation is related to temporality issues and the lack of repeated measurements over time for the child-level and clan-level variables. Some of the child-level variables such as sleep, soda intake, fruit and vegetable, and fast food consumption were gathered at age 5 (wave 4) only. Because the association of these variables was tested in relation to the group-based trajectories that started during birth and ended at age 9, it is possible that they were measured too late. When using group trajectories as the outcome, it is recommended to use variables that are measured as close as possible to the start of the trajectory or to include time-varying variables that are available at the same time points as the outcome measure (Jones & Nagin, 2007; Nagin, 2005).

The last limitation of the study is the limited generalizability of the results. This study used national data; however, it is not nationally representative. The decision of not using weights to make the data nationally representative had to do with the original Fragile Families researchers' sampling strategy. In order to use weights and make the data nationally representative, the participants from four cities (n = 1, 325) have to be dropped because these states were not randomly selected (Reichman et al., 2001). Dropping data on participants from four cities and also adding the restrictions for the analytic sample would have reduced the sample size substantially. Thus, like most researchers using FFCWS data, I decided not to make generalizations about a nationally representative sample.

Despite the limitations outlined above, there were many reasons to use the FFCWS dataset. First, FFCWS provides recent information on child's anthropometric data and maternal mental health information over time. Finding a dataset that included measurements on mothers and children was difficult, especially data that provide recent trends. A second reason for using this dataset was that it had information available from medical records about pregnancy

behaviors that could have influenced childhood obesity development, such as maternal pre-pregnancy BMI and smoking during pregnancy. Another reason for using this dataset was that it oversampled racial/ethnic minorities, which helped me answer questions related to within-group variation and disparities in rates of overweight and obesity among different groups. Thus, in spite of the limitations, this study offers rigorous and useful findings that can move the field of childhood obesity and mental health forward. In the next section I will discuss the implications that this study has for theory, practice, and policy.

Implications for Practice and Theory

The findings from this particular study offered little support to target maternal depression in interventions tailored to address childhood obesity. However, previous literature available on maternal depression and child obesity must be considered. Previous studies have demonstrated that there is an association between maternal depression and childhood obesity. For that reason, ruling it out altogether would be a mistake. Most importantly, it is necessary to continue investigating the role that maternal depression plays in childhood obesity development and also on the development of obesogenic behaviors. In this study I did not investigate the association between maternal depression and television viewing, sleep, dietary intake, or outdoor play. However, it is possible that children's behaviors are more susceptible to maternal depression than is a purely biological measure such as BMI. For example, previous studies have suggested that maternal depression is associated with increased television viewing among children and less healthy food consumption (Bank et al., 2012; Vericker, 2015). In this study, it was also shown that there was a positive correlation between television viewing at age 3 and maternal recurrent depression and there was also a negative correlation between sleep at age 5 and maternal depression. Thus, testing these associations in future studies is advised.

This study used the 6 C's model to determine whether factors from different levels influenced childhood obesity development differently. In the overall sample, neither the child-level nor the clan-level factors were significant, after adjusting for background factors, including maternal pre-pregnancy BMI. However, after stratifying the data, some child-level and some clan-level factors became significantly associated with developing an at-risk trajectory. This study provides further evidence about the importance of considering social determinants of health from multiple levels of the 6 C's model that can potentially influence childhood obesity development.

One of the main contributions of this study for practice purposes is the identification of some common and unique factors for different racial/ethnic groups. In the overall sample maternal pre-pregnancy BMI put children at an increased risk of being part of the most at-risk trajectory – the high-rising trajectory. Maternal pre-pregnancy BMI was also the only predictor that children from all racial/ethnic backgrounds had in common. Thus, maternal pre-pregnancy BMI is the main driver of differences in child obesity risk and growth trajectories. Future interventions that aim to *prevent* childhood obesity need to start as early as possible and need to work with mothers and families even before birth by promoting healthy weight before and during conception. Moreover, it is necessary that future culturally adapted interventions that aim to prevent childhood obesity among particular racial/ethnic groups address unique factors to the population of interest. For instance, this study found that among White children many factors should be considered when developing an intervention. For example, this study found that consuming more fast food placed White children at risk of developing a high-rising trajectory. Thus, it is important to develop interventions that encourage healthy eating and cooking at home. These are malleable factors that can be easily changed and can produce long-lasting effects.

From the point of view of Social Learning Theory, changing these types of behaviors early on in life can promote healthy behaviors among children that last a lifetime. If children observe early on in life that their parents cook at home and eat healthy, children might be more likely to observe and mirror these behaviors, which can eventually become routines that promote health.

Although breastfeeding duration was not found to be associated with group trajectory membership, there is vast literature demonstrating the benefits that breastfeeding has for infants and mothers. Thus, more needs to be done to promote breastfeeding initiation, duration, and exclusivity, particularly among low-income minority women (Marshall et al., 2013). Besides including breastfeeding initiation and duration as an intervention target, it is necessary that campaigns are created to reduce stigma. In this sample, only 60% of women ever tried to breastfeed and even more concerning, only 20% of them breastfed for six months or more. The *2020 Healthy People* goal is that 82% of women initiate breastfeeding and that 60% breastfeed for at least six months and this study suggests that low-income minority women are far from reaching that goal (Office of Disease Prevention and Health Promotion, 2010). There are many reasons behind the low initiation rates of low-income women, but previous studies have shown that stigma is definitely one of them (Acker, 2009). Thus, campaigns that promote a culture that values and encourages breastfeeding are necessary. Initial steps are being taken by creating baby-friendly hospitals where breastfeeding is promoted from birth (Pérez-Escamilla, 2007); however, if breastfeeding is not promoted after the mother leaves the hospital, these initiatives will not provide the expected results. Normalizing public breastfeeding through health promotion campaigns and encouraging family members to encourage breastfeeding duration should be a priority for social workers and public health practitioners.

A surprising finding was that among Latino children none of the child-level or clan-level factors increased the risk of children developing a high-risk trajectory. However, this does not mean that none of these factors should be included in culturally-adapted interventions. Instead, it is necessary to determine what is driving Latino children to have higher BMI z-scores at birth that can predispose them to develop a high-risk growth trajectory. For now, the results found in this study suggest that it is important to address maternal pre-pregnancy BMI. One way to do this in a culturally appropriate way is by promoting group exercise classes such as Zumba or folkloric dance among Latina women of reproductive age, who are pregnant, or had recently had a child (Racine, Coffman, Chrimes, & Laditka, 2013).

Implications for Policy

There are multiple opportunities for policy implementations to foster health and wellbeing among mothers and children. First, given that maternal pre-pregnancy BMI was found to be the main predictor of at-risk growth trajectories, it is imperative to promote preconception (before pregnancy) and interconception (in between pregnancies) care. This type of care can only be accessed if women have insurance. Between 2013 and 2016, the first years of the Affordable Care Act, rates of uninsured women of reproductive age fell by 41% (Guttmacher Institute, 2018). Back in 2013 there were 20% women of reproductive age (15-44) who were uninsured, and this number fell to 12% in 2016 (Guttmacher Institute, 2018). Through the ACA it is possible to provide significant improvements in the wellbeing of women and their children by providing quality care for reproductive age women. It is also necessary that policies are developed to prevent childhood obesity early on and to particularly focus on low-income minority women. The results of this study showed that low-income Latino children are

particularly at risk of developing unhealthy growth trajectories. Thus, developing and implementing policies that can prevent childhood obesity early on are critical.

Besides promoting childhood obesity prevention initiatives, increased attention needs to be given to maternal depression. Although this study among low-income mothers did not find a significant association between maternal depression and group trajectory membership, the detrimental consequences that maternal depression has on children have been consistently documented. For example, maternal depression can be a risk factor for the development of child depression (Garber & Cole, 2010; Hammen, Hazel, Brennan, & Najman, 2011), which in turn can be a psychosocial risk factor for the development of child and adolescent obesity (Blaine, 2008). Previous studies have linked adolescent depression and other psychosocial risk factors such as weight overestimation to weight gain and overweight/obesity status (Liechty & Lee, 2014; Blaine, 2008). Thus, the psychosocial consequences that maternal depression can have on the child might eventually become social risk factors for obesity development (Liechty & Lee, 2014). It is thus necessary to develop and implement policies that prevent and support treatment for maternal depression. While states have made noteworthy improvements on addressing postpartum depression by implementing universal depression screening (Rhodes & Segre, 2013), not much has been done on passing mandates to screen women for depression at other time points besides the perinatal period. Only three states have passed mandates requiring health care providers to screen women for depression during the postpartum period (Segre et al., 2012). It is then critical that more states propose legislation to implement universal depression screening over time. Carrying out depression screenings can take only five minutes and in turn can prevent detrimental consequences for the mother and the child (Gjerdingen, Crow, McGovern, Miner, & Center, 2009).

It is also important to reinforce programs that provide care to low-income pregnant women and their children. For instance, the Women, Infant, and Children (WIC) program is a federally-funded program that provides nutrition education, case management, and supplemental food among pregnant women and children up of the age of 5 who are under 185% of the poverty line ("Women, Infants, and Children (WIC)," 2014). Programs like these are vital for women and their children, particularly low-income families, since in many occasions WIC is the main place that provides women with nutrition and health information. Programs like WIC that provide case management and nutrition education can have a significant impact on the wellbeing of low-income families. Thus, it is necessary to maintain and even expand these programs to reach a wider audience of mothers who do not qualify for WIC but who cannot afford similar resources.

Policies also need to address paid maternal and paternal leave to promote breastfeeding. One of the reasons many women decide not to breastfeed or to stop breastfeeding early is because they have to go back to work (Guendelman et al., 2009). Creating policies that can allow women to access paid maternity leave can encourage breastfeeding initiation and duration, especially among unmarried low-income women, who are usually the ones who have fewer benefits and protections from employers. Moreover, developing policies that encourage paternal leave is also necessary. Previous studies have demonstrated that a strong predictor of breastfeeding initiation and duration is partner's support (Mannion, Hobbs, McDonald, & Tough, 2013; Rempel & Rempel, 2004). Thus, having the baby's father be available to provide support to the mother by accompanying on visits to the lactation consultant or other appointments, or even by providing emotional support and encouragement can be highly beneficial to the mother and the infant. Developing these policy initiatives can help women and children in the short and long-term.

Overall, it is important to raise attention on the topic of maternal and child health. If there is a true interest in preventing childhood obesity and other detrimental consequences, it is imperative to invest more resources and develop more policies that can ensure the healthy development of children during gestation and throughout the first years of life.

Future Research Directions

This study helped advance the scholarship available on the association between maternal depression and childhood obesity development and on racial/ethnic differences of growth. However, this is only a small piece to prevent childhood obesity development. Below, I present a few suggestions on how the field can continue to move forward. First, future longitudinal studies should use more outcome measures that identify obesogenic behaviors rather than BMI alone. It is true that BMI may provide an objective measurement of childhood obesity and growth trajectories can provide a good longitudinal measurement over time; however, as outlined previously, there are many flaws to using BMI and other anthropometric measurements only. Promoting healthy behaviors regardless of BMI and weight should be at the forefront, since these healthy behaviors can promote health and decrease morbidity and mortality (Loef & Walach, 2012). Moreover, by using healthy/unhealthy behaviors as the outcome measure, it may be possible to determine the mechanisms through which maternal depression has been previously found to be associated with BMI. Thus, future studies should focus on understanding the independent association between maternal depression and healthy/unhealthy behaviors among children. Another reason to use child behaviors as the outcome is that previous literature on maternal depressive symptoms shows that children's behaviors are more susceptible to external influences than are biological measurements.

Second, future studies using longitudinal data should maximize the benefits of using these data by investigating "causal" effects. Although only an experimental design could determine true causality, longitudinal data allows researchers to investigate potential "causal" pathways by using lagged-effect models. Since many of the studies that have found a significant association between maternal depression and childhood overweight and obesity are cross-sectional, it is not clear whether maternal depression truly influences child obesity development (i.e. directionality has not been established). Cross-lagged effect models could help researchers identify the nature of the association and determine whether maternal depression should remain a focus of study in relation to childhood obesity or whether other aspects, such as behaviors, should be the focus of investigation.

Third, future studies should stratify by sex. Because the primary aim of this study was to identify racial/ethnic differences in growth patterns, I limited my analysis to racial/ethnic stratification. However, previous studies suggest that male and female children have different growth patterns and it has also been demonstrated that child-level and clan-level factors can affect males and females differently (Boynton-Jarrett, Fargnoli, Suglia, Zuckerman, & Wright, 2010). Thus, it is necessary to stratify by sex to identify the ways in which child-level and clan-level factors can influence males' and females' growth patterns.

Fourth, this study found in the overall sample that child-level and clan-level factors were not associated with at-risk growth patterns; however, in the stratified analysis, it was shown not only that racial/ethnic groups follow different growth patterns, but also that child-level and clan-level factors influenced children of racial/ethnic backgrounds differently. Thus, it is necessary that future studies stratify by race/ethnicity to better understand their effect. Moreover, future studies should determine whether there are other amenable macro-level factors that influence

childhood obesity development. For example, future studies could investigate the role that neighborhood or immigration have on group trajectory membership.

Finally, future studies should focus on developing more qualitative studies that can help illuminate the mechanisms of association between maternal depression and childhood obesity. Also, qualitative studies could help identify differences in childhood obesity risk by race/ethnicity. Particularly, having a better understanding of barriers and facilitators to engaging in healthy or unhealthy behaviors could inform the development of culturally tailored interventions. Moreover, gathering information as to what families want to see in an intervention is imperative. Using a community-based participatory research lens can allow researchers to develop interventions that are useful and enjoyable to families, which in turn can increase intervention adherence.

Overall, this study shed light on the association between maternal depression and childhood overweight/obesity status and between maternal depression and growth trajectories in a low-income mostly minority sample. However, there is still a lot of work that needs to be done around maternal depression and childhood obesity. Because of the mixed literature on the field, it is necessary that the immediate next steps focus on identifying among who and under which conditions maternal depression can influence childhood obesity development. This can be achieved by using more diverse longitudinal samples and also by stratifying by sex and by race/ethnicity. More research in the area of maternal depression and childhood obesity can promote public health campaigns and policies devoted to increasing health and mental health care awareness and access across all populations.

Conclusion

The present study used different methodologies to identify the association between early maternal depression and childhood overweight/obesity and growth trajectories in a low-income mostly minority sample. The different statistical methodologies demonstrated that there was no association. Moreover, this study examined the role that child and clan-level factors had on child growth trajectories and found that none of these factors were significantly associated with child growth trajectories. All the child- and clan-level factors tested in this study had been previously identified either cross-sectionally or longitudinally with childhood overweight and obesity. However, in this low-income sample they were not found to be significantly associated with at-risk growth. This indicates that it is necessary to focus on systemic factors that can be contributing to childhood obesity development in this low-income sample. Future studies need to devote time and resources to investigate different social determinants of health that are affecting childhood obesity development in low-income, mostly minority children in the US. Finally, this study demonstrated that there are qualitatively different growth trajectories by racial/ethnic group, indicating that disparities in childhood obesity start early in life and remain at least through childhood. Specifically, Latino children have the highest prevalence in the most at-risk trajectory of any other racial/ethnic group. Moreover, among Latino children, only maternal pre-pregnancy BMI was a significant predictor of the risky growth trajectory. Thus, future interventions need to be provided before and during gestation to ensure healthy child development. Finally, future studies aiming to understand childhood obesity disparities also need to investigate the role that macro-level factors such as poverty and immigration play in childhood obesity development.

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APPENDIX A: IRB LETTER

UNIVERSITY OF ILLINOIS
AT **URBANA-CHAMPAIGN**

I

Office of the Vice Chancellor for Research

Office for the Protection of Research Subjects

RE: *longitudinal examination of maternal metabolic psychosocial and behavioral risk factors for childhood obesity in the Fragile Families and Child Wellbeing Study*
IRB Protocol Number: 16761

Dear Dr. Liechty:

This letter authorizes the use of human subjects in your project entitled *longitudinal examination of maternal metabolic psychosocial and behavioral risk factors for childhood obesity in the Fragile Families and Child Wellbeing Study.* The University of Illinois at Urbana-Champaign Institutional Review Board (IRB) approved, by expedited review, the protocol as described in your IRB application; as well as the Data Protection Plan for the use of data from The Fragile Families and Child Well Being Study. The expiration date for this protocol, IRB number 16761, is 06/08/2017. The risk designation applied to your project is *no more than minimal risk.*

Under applicable regulations, no changes to procedures involving human subjects may be made without prior IRB review and approval. The regulations also require that you promptly notify the IRB of any problems involving human subjects, including unanticipated side effects, adverse reactions, and any injuries or complications that arise during the project.

If you have any questions about the IRB process, or if you need assistance at any time, please feel free to contact me at the OPRS office, or visit our website at https://www.oprs.research.illinois.edu.

Sincerely,

LeaAnn Carson

LeaAnn Carson,
MS OPRS
Specialist

Attachment(s)

c: Maria Pineros Leano

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APPENDIX B: DATA CHARACTERISTICS STRATIFIED BY RACE/ETHNICITY

Table B.1: Sample characteristics stratified by race/ethnicity

Variables	Total n (%)	White n (%)	Black n (%)	Latino n (%)
Child sex				
Female	1611 (47.93)	384 (48.18)	781 (48.03)	446 (47.55)
Male	17.50 (52.07)	413 (51.82)	845 (51.97)	492 (52.45)
Child BMI z-score, SD	-0.24 (1.09)	-0.24 (1.10)	-0.35 (1.09)	-0.05 (1.06)
Maternal education				
High school or less	2135 (63.58)	325 (40.78)	1102 (67.82)	708 (75.64)
Some college or more	1223 (36.42)	472 (59.22)	523 (32.18)	228 (24.36)
Federal poverty level				
0-99%	1168 (34.75)	101 (12.67)	689 (42.37)	378 (40.30)
100-199%	880 (26.18)	157 (19.70)	438 (26.94)	285 (30.38)
200% or more	1313 (39.07)	539 (67.63)	499 (30.69)	275 (29.32)
Maternal relationship with baby's father				
Married	847 (25.20)	408 (51.19)	220 (13.53)	219 (23.35)
Cohabiting	1226 (26.48)	243 (30.49)	550 (33.83)	433 (46.16)
Other	1288 (38.32)	146 (18.32)	856 (52.64)	286 (30.49)
Maternal age, SD	25.05 (5.95)	27.25 (6.44)	24.28 (5.61)	24.52 (5.62)
Number of biological children, SD	1.10 (1.27)	0.87 (1.13)	1.24 (1.35)	1.04 (1.21)
Pre-pregnancy BMI, SD	26.50 (6.65)	25.10 (6.22)	27.33 (7.16)	26.35 (5.92)

Note. BMI: body mass index.

Table B.2: Child characteristics of Waves 2-5 of the sample stratified by race/ethnicity

Variable	Total n (%)	White n (%)	Black n (%)	Latino n (%)
Child BMI z-score W5, SD	0.78 (1.10)	0.50 (1.10)	0.85 (1.08)	0.90 (1.11)
Child BMI z-score W4, SD	0.63 (1.13)	0.52 (1.07)	0.56 (1.14)	0.84 (1.11)
Child BMI z-score W3, SD	0.59 (1.24)	0.48 (1.13)	0.49 (1.24	0.86 (1.26)
Child OW/OB status W5				
Normal and underweight	1448 (57.12)	392 (67.01)	703 (54.79)	353 (52.92)
Overweight	437 (17.24)	98 (16.75)	238 (18.55)	101 (15.14)
Obese	650 (25.64)	95 (16.24)	342 (26.66)	213 (31.93)
Child OW/OB status W4				
Normal and underweight	1105 (64.70)	254 (67.37)	596 (66.30)	255 (59.03)
Overweight	313 (18.33)	76 (20.16)	159 (17.69)	78 (18.06)
Obese	290 (16.98)	47 (12.47)	144 (16.02)	99 (22.92)
Child OW/OB status W3				
Normal and underweight	1259 (64.43)	284 (68.60)	694 (68.51)	281 (53.32)
Overweight	337 (17.25)	72 (17.39)	157 (15.50)	108 (20.49)
Obese	358 (18.32)	58 (14.01)	162 (15.99)	138 (26.19)
Child age W5	111.47 (4.66)	110.93 (4.18)	111.18 (4.76)	112.50 (4.73)
Child age W4	63.77 (3.02)	63.40 (2.78)	63.90 (2.95)	63.80 (3.36)
Child age W3	38.47 (3.20)	37.97 (2.86)	38.29 (3.20)	39.21 (3.33)
Child age W2	14.89 (3.38)	13.92 (3.04)	15.83 (3.57)	14.07 (2.83)
Sleep W4				
Less than 10 hours	1175 (51.35)	176 (32.96)	709 (60.55)	293 (49.83)
At least 10 hours	1113 (48.65)	358 (67.04)	460 (39.45)	295 (50.17)
Television viewing (average)				
Less than 2 hours	829 (28.39)	290 (42.34)	307 (21.39)	232 (29.00)
At least 2 hours	2091 (71.61)	395 (57.66)	1128 (78.61)	568 (71.00)
Television viewing W4				
Less than 2 hours	673 (29.82)	220 (41.59)	271 (23.67)	182 (31.22)
At least 2 hours	1584 (70.18)	309 (58.41)	874 (76.33)	401 (68.78)
Television viewing W3				
Less than 2 hours	720 (27.59)	242 (39.10)	271 (21.31)	207 (28.79)
At least 2 hours	1890 (72.41)	377 (60.90)	1001 (79.69)	512 (71.21)
Outdoor play (average)	2.73 (1.73)	2.93 (1.58)	2.54 (1.74)	2.90 (1.79)
Outdoor play W4	2.42 (1.88)	2.66 (1.72)	2.21 (1.88)	2.61 (1.97)
Outdoor play W3	2.99 (2.12)	3.17 (1.87)	2.83 (2.18)	3.12 (2.19)
Soda intake W4	0.77 (1.15)	0.47 (0.91)	0.84 (1.21)	0.89 (1.19)
Fruit and vegetable intake W4	3.50 (1.35)	3.36 (1.35)	3.66 (1.30)	3.32 (1.42)
Fast food intake W4	1.23 (1.03)	1.04 (0.93)	1.25 (1.06)	1.35 (1.04)
Breastfeeding ≥ 6 months				
No	2154 (80.70)	417 (72.65)	1167 (85.37)	570 (78.30)
Yes	515 (19.30)	157 (27.35)	200 (14.63)	158 (21.70)

Note. BMI: body mass index. W2: wave 2 (age 1). W3: wave 3 (age 3). W4: wave 4 (age 5). W5: wave 5 (age 9).

Table B.3: Maternal characteristics at W2 and W3 stratified by race/ethnicity

Variable	Total n (%)	White n (%)	Black n (%)	Latino n (%)
Maternal involvement W3	5.37 (1.42)	5.74 (1.21)	5.33 (1.42)	5.11 (1.51)
Maternal depression W3				
Yes	681 (20.26)	151 (18.95)	370 (22.76)	160 (17.06)
No	2680 (79.74)	646 (81.05)	1256 (77.24)	778 (82.94)
Maternal depression W2				
Yes	517 (15.38)	119 (14.93)	274 (16.85)	124 (13.22)
No	2844 (84.62)	678 (85.07)	1351 (83.15)	814 (86.78)
Maternal recurrent depression				
No maternal depression	2428 (72.24)	593 (74.40)	1126 (69.25)	709 (75.59)
Maternal depression at W2 *or* W3	668 (19.88)	138 (17.31)	356 (21.89)	174 (18.55)
Maternal depression at W2 *and* W3	265 (7.88)	66 (8.28)	144 (8.86)	55 (5.86)

Note. BMI: body mass index. W1: wave 1 (birth). W2: wave 2 (age 1). W3: wave 3 (age 3). W4: wave 4 (age 5). W5: wave 5 (age 9).