Does Genetic Conflict Contribute to Pregnancy Complications and Postpartum Health and Behavior?

by

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ABSTRACT

Pregnancy is often described as one of the most cooperative ventures that a woman can experience in her lifetime. But when one considers the biological changes that occur during pregnancy, it becomes clear that pregnancy is not as cooperative as it seems on the surface. The current research uses a genetic conflict framework to predict how underlying conflict between mother and fetus over resource transfers is expected to alter eating behavior and food preferences, and how these changes in eating behavior and preferences should then be associated with certain pregnancy complications. Across two studies, women who had recently had a baby (Study 1) or were currently pregnant (Study 2) recalled changes in their eating behavior during pregnancy as well as any pregnancy complications they experienced during that pregnancy. Providing partial support for the hypotheses, women who reported increased vomiting in response to maternal-favoring foods were more likely to experience preeclampsia during pregnancy. In addition, the results provided preliminary evidence that changes in pregnancy eating behavior were associated with an increased the likelihood of experiencing high blood pressure, gestational diabetes, and infections during pregnancy. Taken together, these studies show that the framework of genetic conflict makes testable predictions about the relationship between eating behavior in pregnancy and pregnancy complications, and that several pregnancy complications that are relevant to genetic conflict (high blood pressure, pre-eclampsia, gestational diabetes, and infection) are associated with changes in eating behavior in pregnancy. Future research should continue to investigate how genetic conflict influences the relationships between pregnancy eating behavior, pregnancy complications, and how these associations impact postpartum health.

DEDICATION

For the budding graduate students who are told that they are too loud, aggressive, rude, or need to change themselves. You aren't. Science is for everyone, so keep taking up the space you need.

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CHAPTER 1

INTRODUCTION

Pregnancy is often viewed as an extreme act of cooperation, where both the mother and fetus are peacefully working together to ensure that the fetus is born. This perception exists not only in the scientific community, where advances in reproductive technologies that aid in the creation of life are openly celebrated, but also in social contexts where gatherings are specifically designed to shower the new parents with love, affection, celebration, and support as they transition into a new phase of their lives. When we consider pregnancy and birth from an evolutionary perspective, where the ultimate goal for all organisms is to reproduce, these positive perceptions of the biological, psychological, and social role changes that happen during pregnancy align with the scientific thinking surrounding these topics.

However, there are also conflicts of interests between the mother and fetus during pregnancy. While the mother and fetus are related, they are not genetically identical. Mother and fetus, therefore, do not have perfectly aligned fitness interests. These unaligned fitness interests can ultimately be traced back to the underlying genetic conflict between maternal and paternal genes. Across all of the interactions between mother and fetus, these different sets of genes attempt to alter fetal growth and development to be slightly more in line with their interests as opposed to with those of the other parent (Fowden & Moore, 2012; Haig, 1993; Moore, 2012; Trivers, 1974). This means that while both mother and fetus ultimately want the fetus to be born and to thrive, there are disagreements on a genetic level over the optimal level of investment of resources in the fetus during pregnancy.

Broadly, the different strategies that maternal and paternal genes pursue can be classified into one of two categories. In one category, the optimal strategy for fetal growth and development prioritizes what is in the mother and her genes' best interest (less growth while still in utero). In the other category, the optimal strategy for fetal growth and development prioritizes what is in the fetus and its *paternal* genes' best interest (more growth while still in utero). Whenever there is a biological event that can impact fetal growth and developmental trajectories (e.g., placental implantation, remodeling of spiral arteries, resource transfers between mother and fetus), the maternal and fetal optimal strategies are in conflict. However, while both sides are actively pursuing strategies that are in their best interests, this conflict of interest happens within the constraints of an overarchingly cooperative relationship.

Using Hamilton's rule (1964) to describe this phenomena, Haig (1993) explained that these disagreements over the optimal trajectory of growth and development persist throughout the course of pregnancy as long as the fitness benefits the fetus receives are twice as large as the fitness costs to any of the mother's other children. In these situations, fetuses have slightly more bargaining power since mothers are selected to provide sufficient biological resources for growth and development while the fetus is in utero. Mothers, therefore, only face a tradeoff between current and future reproductive opportunities when fetuses try to take more resources from the mother than the mother is selected to provide (i.e., benefit to the current fetus > 2 x the cost to the mother's other children) because these additional resources, from the mother's perspective, could be invested into the growth and development of other children.

While research on the effects of genetic conflict is a growing area of study in the biological sciences (Rice, 2013; Werren, 2011), researchers studying the psychological and behavioral changes that accompany these biological processes have yet to incorporate this framework into their investigations. This is true even for researchers who study biologically and psychologically entwined processes such as pregnancy, birth, and postpartum health. Given that gestation and birth are considered to be universal human experiences (Held, 1989), it is surprising that the interactions between biological and psychological changes during this time are not fully understood. To address this gap in the literature, I investigated if genetic conflict during pregnancy is related to a commonly discussed pregnancy-induced behavioral change: changes in eating behavior. The current work seeks to connect these literatures by documenting that changes in pregnancy eating behavior are an index of genetic conflict and can be used to predict a woman's likelihood of experiencing pregnancy complications. This connection, in turn, provides a more comprehensive understanding of how genetic conflict influences pregnancy outcomes.

Cooperation and Conflict in Human Pregnancy

As discussed earlier, there are two perspectives regarding the changes that happen during pregnancy: one that explains maternal changes during pregnancy as the result of maternal and fetal systems actively cooperating to ensure the fetus can be born, and one that explains maternal changes during pregnancy as the result of delicate conflict negotiations between maternally- and paternally-expressed genes over the growth and development of the fetus that occur within an overarchingly cooperative relationship. Neither perspective endorses the notion that genes are consciously making decisions

about when and how to cooperate or compete, but rather that these processes are happening as the result of selfish genes advocating for their own "interests" in order to ensure that they successfully replicate themselves into the next generation (Dawkins, 1976). Below, I discuss how these different perspectives have been applied in research on changes in pregnancy eating behavior.

Building from the cooperative perspective of pregnancy, changes in maternal behaviors during pregnancy are conceptualized as a suite of adaptative changes designed to protect and accommodate the changing developmental needs of the fetus over the course of its gestational development. Some examples of these pregnancy related changes include nesting during late pregnancy (Anderson & Rutherford, 2013; Ketterman et al., 2022), increased disgust towards and avoidance of potential disease vectors during pregnancy (Fleischman & Fessler, 2011), and increased social monitoring for potential threats during pregnancy (Anderson & Rutherford, 2010; Maner & Miller, 2014). The most salient example of this cooperative perspective, however, can be found in the literature on nausea and vomiting during pregnancy. In this body of literature, nausea and vomiting during pregnancy have been proposed as spontaneous maternal behavioral changes that ensure the mother avoids potentially teratogenic foods that may harbor plant toxins or pathogenic microorganisms, and can disrupt the development of fetal organs, neurological systems, and potentially lead to adverse pregnancy outcomes (Crystal et al., 1999; Day, 1992; Fessler, 2002; Flaxman & Sherman, 2000, 2008; Hook, 1976; Profet, 1988, 1992; Sherman & Flaxman, 2001; Tantibanchachai, 2014).

One specific adverse pregnancy outcome that has been heavily investigated in the literature on nausea and vomiting during pregnancy is the experience of miscarriage.

Researchers using this cooperative perspective of pregnancy behavioral change have documented a robust relationship between nausea and vomiting during early pregnancy and lowered miscarriage rates. This relationship, often referred to in the literature as the prophylaxis or "maternal and embryo protection" hypothesis, documents that women who experienced more nausea and vomiting during early pregnancy also experienced lower rates of miscarriage when compared with women who did not experience nausea or vomiting during pregnancy (Flaxman & Sherman, 2008; Sherman & Flaxman, 2002; Weigel et al., 2006, 2011; Weigel & Weigel, 1989). Interestingly, this relationship is believed to be attributable to the positive relationship between nausea and vomiting during pregnancy and placental weight (Weigel et al., 2006) such that more nausea and vomiting during pregnancy leads to larger placentas, and these larger placentas are actually what decreases the risk of experiencing a miscarriage.

A contrasting but related perspective on this literature comes from research suggesting that the relationship between nausea and vomiting during early pregnancy and the lowered rate of miscarriage is better described as a byproduct of a properly functioning placenta. In this research, the nausea and vomiting that an expectant mother experiences during early pregnancy is not thought to be an adaptive response from the maternal body but instead is the tolerated consequence of the placenta releasing appropriate quantities of human chorionic gonadotropin (hCG) to facilitate placental development in early pregnancy and enable the maternal body to sustain a viable pregnancy (Lin et al, 1995; Vaitukaitis, 1974). Even so, this view of nausea and vomiting during pregnancy still describes the inherent cooperation in this relationship since the

mother tolerates the symptoms of this byproduct in order to promote fetal growth and development.

It is important to note, however, that the research documenting protective effects of nausea and vomiting during pregnancy has only been found in association with lowered miscarriage rates. There are many other complications, such as hyperemesis gravidarum and gestational diabetes (Kuru et al., 2012; Ohara et al., 2016), where nausea and vomiting actually indicates the existence of pregnancy complications as opposed to providing protection against them. The cooperative view of pregnancy behavioral change has yet to incorporate theoretical framework showing how changes in eating behavior may provide some protection against other pregnancy complications such as these.

But, as discussed earlier, there is conflict throughout the course of pregnancy since the mother and fetus do not have completely aligned fitness interests (Fowden & Moore, 2012; Haig, 1993; Moore, 2012; Trivers, 1974). How exactly does this conflict manifest during pregnancy? To understand this conflictual perspective of pregnancy, we have to re-contextualize sexual reproduction and mammalian pregnancy in the light of genetic conflict theory (Haig, 1993). Traditionally, human reproduction is discussed as the union of a sperm and an egg to create a new individual. But this is not the complete story. The genes we inherit from each parent carry notably different instructions for our development and behavior, which means there are contrasting—and sometimes conflicting—blueprints for building and maintaining our bodies even though both sets of genes need to cooperative with one another in order to accomplish their goal of making it into future generations. This is the essence of genetic conflict.

The effects of genetic conflict are endemic and observable in pregnancy, as pregnancy is the only time where a single individual is responsible for balancing the health, well-being, energetic needs, and fitness "interests" of two or more related, but not genetically identical, individuals. Since the fetus inherits half of its instructional information from its mother and half of its instructional information from its father, this creates a unique situation where researchers are able to see the extent to which maternal and paternal genes have differing optimal trajectories for fetal growth and development by assessing how maternal biological processes that are normally stable during non-pregnancy (e.g., blood pressure, blood sugar levels) change while she is pregnant. One of the clearest biological processes where researchers can investigate the influence of unaligned fitness interests between maternal and paternal genes is in the resource transfers that the mother makes to her fetus (see Bowman et al., 2021, for a general overview of conflict over resource transfers during pregnancy).

All resources transfers that occur during pregnancy are transferred from mother to fetus through the placenta. The placenta is a fetally-derived endocrine organ (Power & Schulkin, 2012) that contains approximately 100 genes where the maternal and paternal optima for growth and development are not the same (Trivers & Burt, 2006; Wang et al., 2013). The lack of "agreement" between maternal and paternal genes allows researchers to see the footprints of genetic conflict during pregnancy (Fowden & Moore, 2012; Moore, 2012). As the placenta is the first organ to develop during pregnancy, starting even before the conceptus implants itself into the uterine lining, biologists often begin their investigations of genetic conflict by studying how conflict over placental development may manifest. In addition, once the conceptus implants into the uterine

lining, the placenta negotiates all other physiological changes to the uterine environment, enables all resource transfers throughout the remainder of the pregnancy, and sets the stage for how other "disagreements" between maternal and paternal optima may manifest.

But *why* is there conflict over the amount of resources transferred from the mother to her fetus? Shouldn't both sides want there to be an adequate transfer of resources so the fetus can grow properly? One reason for this conflict is that mothers have a limiting pressure on the number of children they may have during their lifetime. This is the case not only because mothers are born with the total number of gametes they will have in their lifetime (Trivers, 1972) but also because mothers must use the biological resources within their bodies (e.g., calcium, zinc, iron) to sustain each pregnancy. Some of these resources are limiting meaning that if the mother uses these resources for one pregnancy, she cannot use them in a subsequent pregnancy. Other resources can be replenished through her diet during or between pregnancies. This means, for every resource transfer, the mother has to assess the trade-offs associated with investing resources in the current pregnancy, investing resources in future pregnancies, or investing resources in her already existing children.

All else being equal, it is in the mother's best interest to equally distribute her resources across all pregnancies so she can invest her resources equally across her current and potential future children (Haig, 1992; 1993). This strategy of equal investment limits the extent of the trade-offs between current and future reproduction that she then experiences. However, because the fetus has genes from both mom and dad and there is no guarantee that the paternal genes will be in any other children the mother may have, it

is in the fetus's best interest to extract as many of the mother's resources as possible so it can grow as large as possible while still in utero (Moore, 2012) even if this is to the detriment of maternal genes, resources stores, and future reproduction.

A clearer way to picture this conflict is to consider an analogy from Haig (1992) that describes how the differences in optimal resource transfers manifest during pregnancy. Pretend that all of the resources a mom has to give her current and future children come in the form of a milkshake. Once the milkshake runs out, the mom will not be able to provide her children with the resources they need to survive. Mom, and her genes, know that there is this limiting pressure and therefore want all of mom's current and future children to share the resource milkshake equally. This means that maternal genes advocate for each child to "drink" only as much of the resource milkshake as they need to grow and develop properly. The fetus, and more specifically the genes it inherited from its father, do not have the same constraint since there is no guarantee that any other children the mother has will have the same paternal genes as the current child. The best strategy for paternal genes in the fetus, therefore, is to "drink" as much of the resource milkshake as they can (or take as many resources as possible) because these genes do not have the same pressure to save shares of the resource milkshake for guaranteed siblings[1].

But the disagreements caused by genetic conflict do not stop there. This conflict over resource transfers then extends to conflicts over growth and development across pregnancy, where the maternal optimum is for the fetus to have slightly less growth and development in utero while the paternal optimum is for the fetus to have slightly more

[1] The way the milkshake problem is derived assumes an evolutionary history of non-monogamy. It is possible that the evolution of monogamous relationships facilitated a reduction in costs due to genetic conflict and the costs associated with escalated conflict.

growth and development in utero (McKeown & Record, 1953). Now, instead of dealing with solely with conflict over resource transfers, genetic conflict between maternal and paternal genes begins to influence pregnancy outcomes. Maternal genes are aware that an offspring that is born too large to pass through the birth canal will not only extract more resources during pregnancy, but these larger babies will also require that the mother spend more resources in labor, delivery, healing, and feeding postpartum (Stanford Children's Hospital, 2020; University of Rochester Medical Center, 2022) while paternal genes do not have this same limiting selection pressure.

Eating Behavior as an Index of Genetic Conflict

The inherent conflict of interests that exist during pregnancy suggests that, even though the mother's body provided for the fetus as it develops in utero, fetuses have the ability to actively interact with their mother's physiology and advocate for their paternally-derived fitness interests. On a practical level, this means that fetuses directly change maternal physiology to extract more resources from the maternal body than is optimal for the maternal body to provide. Some of the ways that fetuses are able to advocate for their paternally-derived interests from a purely biological standpoint include investing more resources in placental development early in gestation, altering concentrations of reproductive hormones in the maternal circulatory system to increase blood pressure, and increasing concentrations of insulin-like growth factors in the maternal circulatory system to elevate blood sugar (Fowden & Forhead, 2009, 2013; Furneaux et al., 2001; Hay, 1994; Huxley, 2000; Lin et al., 1995; Murphy et al., 2006;

Reik et al., 2003). All of these strategies allow the fetus to increase the resources transferred from its mother to itself during pregnancy.

Another mechanism that fetuses can use to upregulate resource transfers during pregnancy is changing their mother's taste perception through placentally-induced changes to the maternal body (Bowen, 1992; Brown & Toma, 1986; Macedo & Diez-Garcia, 2014; Sipiora et al., 2000). As stated earlier, the placenta is a major endocrine organ that directly interacts with the maternal systems to facilitate resource transfers from mother to fetus (Burton & Jauniaux, 2015; Fowden & Moore, 2012; Murphy et al., 2006). But, by the end of pregnancy, the placenta is also responsible for producing many of the hormones needed to sustain pregnancy, such as progesterone and human chorionic gonadotropin (Costa, 2016; Donnelly & Campling, 2014). In addition to producing the hormones necessary to sustain pregnancy, the placenta also takes over the production of appetite-regulating hormones leptin and ghrelin (Allbrand et al., 2018; Ashworth et al., 2000; Forhead & Fowden, 2009; Fuglsang et al., 2005; Pérez-Pérez et al., 2018) and alters maternal taste preferences during pregnancy (Bowen, 1992; Brown & Toma, 1986). This means that fetuses can directly manipulate maternal perceptions of hunger, appetite, and what tastes good or not during pregnancy.

One such change in taste preferences during pregnancy is referred to as cravings during pregnancy. It is common for pregnant women to develop cravings for sweet foods such as candy and fruits (Hook, 1976; McKerracher et al., 2016; Orloff & Hormes, 2014; Weigel et al., 2011) as a result of fetal manipulation of eating behavior. Using genetic conflict theory, I hypothesize that one explanation why sweet foods become the most commonly craved food during pregnancy is that placentally-produced ghrelin and leptin

advocating for fetal interests by manipulating the maternal diet to include more simple sugars. These simple sugars are then easily transferred across the placenta and to the fetus to support more growth in utero. However, another explanation for cravings during pregnancy is that the cravings are a compensatory avenue for the maternal body to "make up" for any nutrients that are missing from the mother's diet and sometimes resulting in conditions such as pica during pregnancy (Mills, 2007). Regardless of the proximate reason for these changes in eating behavior, the conflict over eating behavior is one example of where maternal and fetal interests are directly at odds with one another.

Two Optima for Maternal Eating Behavior

As with other conflicts over resource transfers during pregnancy, the genetic conflict framework predicts that there should be two optimal diets: one that prioritizes the fitness interest of the maternal genes and one that prioritizes the fitness interests of the fetus and its paternal genes (referred to as the fetal optimum from here on). While there are different optimal diets, this does not imply that the diets consist of entirely different nutrients. Rather, these optimal diets differentially prioritize the needs and interests of the mother and the fetus while still ultimately ensuring that there are sufficient nutrients in the diet for both individuals. Using this genetic conflict perspective on pregnancy eating behavior, which I have developed here, it is clear that the fetus's interests are best served when the maternal diet consists for many simple sugars so that there is a rapid transfer of these sugars across the placenta. With this diet, all other limiting resources needed for development (e.g., proteins, calcium, iron, zinc) are extracted from the existing stores in

the mother's body. An example of a diet that prioritizes the fetus's interests would be one full of sugars, sweets, fruits, and other foods high in simple sugars.

The maternal interests, on the other hand, are best served when the maternal diet is not manipulated to increase simple sugars and instead involves a wide array of foods. With this diet, the fetus acquires all of the nutrients that it needs from the maternal bloodstream and therefore does not need to extract resources from the mother's existing resource stores unless it is absolutely necessary. An example of a diet that prioritizes the maternal interests would be a diverse diet full of a mixture of necessary nutrients that facilitate fetal growth and development. Returning to the milkshake analogy, a diverse diet allows mom to protect her resources stores, ensure that this fetus isn't taking more than its fair share of her resource milkshake, and ultimately lowers the trade-offs that she experiences when investing in the current pregnancy.

Overview of Studies

As a major goal of many public health organizations is increasing the health and well-being of mothers and their children (Center for Disease Control and Prevention, 2022; Office of Disease Prevention and Health Promotion, 2014), it seems necessary that researchers and public health officials alike fully understand the biological and psychological changes that occur during pregnancy in order to achieve this goal. To that end, these studies address the relationships between genetic conflict, pregnancy eating behaviors, and pregnancy complications.

Due to the lack of research on eating behavior as an index of genetic conflict, I designed these studies to assess how changes in pregnancy eating behavior gives insight

into the conflict over resource transfers during pregnancy and increases the risk of experiencing pregnancy complications. To do this, I complied an extensive list of foods for which there could be genetic conflict over eating behavior and created a classification system that focuses on how the foods prioritize maternal or fetal interests during prgnancy. Prior food lists have centered on very specific groupings, such as meats and vegetables that may contain teratogens (Fessler, 2002; Profet, 1992) or the number of foods that were associated with changes in eating behavior without identifying specific types of food (Crystal et al., 1999), so this new classification system was necessary for me to focus on how these foods could favor maternal and/or fetal interests during pregnancy. I created five categories of foods where maternal and fetal interests could be differentially prioritized during pregnancy: foods that favor the fetus in resource transfers, foods that favor the mother in resource transfers, foods that favor both individuals, foods that cultivate healthy microbes (probiotic foods), and foods that prevent the growth of pathogenic microbes.

If food choice is an index of fetal manipulation during pregnancy, then we should see that specific changes in eating behaviors are associated with specific pregnancy complications. For example, if the maternal system is actively trying to lower blood pressure (something that results in less sugar transfer to the fetus) then the fetus may respond by manipulating eating behavior, so the mother eats more fetal-favoring foods and, as a result, has elevated blood sugar. This increased blood sugar affords the fetus with more possibilities to extract simple sugars and grow larger in utero even if the maternal system successfully lowers blood pressure. But this conflict between maternal and fetal interests can lead to complications such as gestational diabetes, high blood

pressure, or preeclampsia. As such, I created a list of common pregnancy complications (derived from National Institute of Child Health and Human Development, 2017) that included complications known to be influenced by genetic conflict as well as other complications that currently have not been linked to genetic conflict theory. Across two studies, I surveyed women who have been (Study 1) or are currently pregnant (Study 2) and asked them to recall details about their most recent pregnancy including any eating behavior changes and pregnancy complications.

The purpose of the first study was to show that changes in eating behavior can be used as an index of genetic conflict and predict the likelihood of experiencing pregnancy complications. Additionally, the first study allowed me to assess whether the prophylaxis hypothesis (Flaxman & Sherman, 2008) is applicable to other pregnancy complications, or if this explanation of changes in eating behavior during pregnancy does not extend beyond its association with lowered miscarriage rates. General predictions were derived from the literature on nausea and vomiting during pregnancy being "protective" against pregnancy complications.

> Hypothesis 1: Based on the prophylaxis hypothesis, cravings during pregnancy should be associated with experiencing pregnancy complications since cravings increase the chances on consuming potentially teratogenic foods.
>
> Hypothesis 2: Cravings for fetal-favoring foods should be associated with experiencing pregnancy complications because fetal-favoring foods raise blood sugar. Cravings for other food groups should not be associated with pregnancy complications because these other food groups do not raise blood sugar.

After testing these hypotheses, I then explored the relationships between changes in eating behavior and specific pregnancy complications to assess if nausea and vomiting is protective against pregnancy complications other than miscarriage.

The purpose of the second study was to replicate the specific changes in eating behavior that were associated with specific pregnancy complications (high blood pressure, infections, and preeclampsia) from the first study. In addition, I also wanted to perform confirmatory factor analyses on my five theoretically derived food groups to determine if this model parsimoniously explains the variance in changes in eating behaviors.

Hypothesis 1: The five-factor model of food groups should adequately represent the variance of changes in eating behavior during pregnancy.

Hypothesis 2: Stronger cravings for or aversions towards fetal-favoring foods should be associated with experiencing high blood pressure.

Hypothesis 3: Stronger cravings for antimicrobial foods should be associated with experiencing infections during pregnancy.

Hypothesis 4: Stronger aversions towards or nausea and vomiting in response to fetal-favoring foods should be associated with experiencing preeclampsia.

I also investigated how changes in eating behavior influence the likelihood of experiencing gestational diabetes, as this is a pregnancy complication known to be caused by genetic conflict.

CHAPTER 2

STUDY 1

The purpose of this study was to provide preliminary evidence that changes in eating behaviors during pregnancy are an index of underlying genetic conflict and can be used to predict pregnancy complications. As this was an exploratory study, I did not have specific hypotheses about how changes in eating behavior would influence pregnancy complications. However, I expected that cravings, and more specifically cravings for fetal-favoring foods, would be associated with experiencing pregnancy complications. I also predicted that aversions, nausea, and vomiting should not be associated with pregnancy complications because, according to the prophylaxis hypothesis, these changes in eating behavior decrease the possibility that teratogenic substances will be ingested (i.e., Flaxman & Sherman, 2008; pre-registration of hypotheses available at https://osf.io/ydgrn/).

Method

Participants. Women ($N = 200$) were recruited through Prolific.co to participate in a 10-15 minute survey about dietary changes during pregnancy and were compensated $1.00 USD for their time. The only inclusion criteria were that the participants must be 1) female, 2) fluent in English, and 3) have given birth within the last five years or were currently pregnant. Of the participants who completed the survey, two were removed from the analyses. One was removed because they indicated that they were male, and one was removed because they indicated that they had never been pregnant before. This left a final sample of 198 women ($M_{age} = 39.16$ years, $SD_{age} = 11.39$ years; 72.7% White;

78.2% had some college education; average of 3 to 4 years since their most recent pregnancy). Roughly 72% of participants reported experiencing a pregnancy complication during their most recent pregnancy with some women reporting that they experienced more than one complication during their most recent pregnancy (see Table 1 for the number of women who experienced each pregnancy complication).

Table 1. Pregnancy complication frequencies for Study 1.

Pregnancy complication	*N*	Percent of sample
High blood pressure	49	24.7%
Gestational diabetes	21	10.6%
Infections	10	5.1%
Preeclampsia	23	11.6%
Hyperemesis gravidarum	9	4.5%
Preterm labor	27	13.6%
Pregnancy loss/ miscarriage	52	26.3%
Stillbirth	1	0.5%
Other complications	30	15.2%
Prefer not to share	8	4.0%

Materials & Procedure. After consenting to participate in the study, participants answered demographics about themselves (i.e., age, sex, ethnicity, education) and their pregnancies (i.e., how many times they have been pregnant, what complications they experienced in their most recent pregnancy). The pregnancy complication list was derived from the most common pregnancy complications reported by the National Institute of Child Health and Human Development (2017) and included complications known to be influenced by genetic conflict as well as other complications that currently do not have clear links to genetic conflict theory.

After finishing the demographics sections, participants were then presented with descriptions of the changes in eating behavior (see Appendix A) as well as a

comprehensive list of foods and drinks that may have caused these changes in eating behavior. For questions regarding each change in eating behavior (e.g., cravings, aversions, and nausea/ vomiting), participants first indicated whether or not they experienced this change in their eating behavior during pregnancy and what foods and drinks caused the change (see Table 2 for the food and drink groupings).

Table 2. Theoretically derived food categories.

Type of food group	Examples of food
Fetal-favoring foods	Fruits Bread/cakes Sweets & candies Fast foods Soda Fruit juices
Foods that are good for both	Milk Ice cream & milkshakes Cheese
Maternal-favoring foods	Vegetables Meat & fish Eggs Nuts & nut butters Ice Water
Antimicrobial foods	Spicy foods Tea Coffee Alcoholic drinks Earthy non-food substances Starchy non-food substances Chocolate Cocoa and hot chocolate
Probiotic foods	Yogurt Umami Pickled food

After completing these sections, participants were given an open-ended text box where they could provide any additional information that they felt was relevant regarding the changes in eating behavior they experienced during pregnancy. Participants were then thanked for their time and participation.

Results

Cravings and Complications Analysis. To assess whether cravings were associated with experiencing pregnancy complications broadly, I conducted a chi-squared association test. I found that women who reported having aversions, χ^2 (1, N = 198) = 3.83, p = 0.05, or experiencing nausea and vomiting, χ^2 (1, N = 198) = 18.80, p < 0.001, at any point during pregnancy reported experiencing pregnancy complications. There was no association between experiencing cravings at any point during pregnancy and experiencing pregnancy complications, χ^2 (1, N = 198) = 0.31, p = 0.58. Together, these results do not support my hypothesis that cravings during pregnancy are associated with experiencing pregnancy complications. Also, these results do not support the general predictions from the prophylaxis hypothesis that nausea and vomiting are protective, as those women who reported experiencing nausea and vomiting during pregnancy reported experiencing pregnancy complications.

Cravings for fetal-favoring foods and pregnancy complications. One possible reason why the hypothesis, derived from the prophylaxis hypothesis, that cravings should be associated with pregnancy complications was not supported is due to the way the data were analyzed. In the previous analysis, the foods that caused the cravings, aversions, and nausea and vomiting were combined into a single category of change in eating behavior when performing the analyses. This strategy could have obscured the effects of the

specific categories of food. Therefore, I next assessed the associations between cravings, aversions, and nausea and vomiting in response to specific food groups and experiencing pregnancy complications (see Table 3 for the associations).

For cravings, I found associations between cravings for fetal-favoring, maternal-favoring, and antimicrobial foods at any point during pregnancy and experiencing pregnancy complications. There were no associations with aversions to any of the food categories and pregnancy complications. For nausea and vomiting, I found associations between experiencing nausea and vomiting in response to fetal-favoring, good for both, and antimicrobial foods, and experiencing pregnancy complications. However, once I corrected for multiple tests (Bonferroni correction $p = 0.01$), these associations were no longer significant. Together, these results provide partial support for my second hypothesis but also do not support the prophylaxis hypothesis predictions.

Table 3. Associations between changes in eating behavior, specific food groups, and pregnancy complications.

	Fetal-favoring foods	Good for both foods	Maternal-favoring foods	Antimicrobial foods	Probiotic foods
Cravings	$\chi^2 = 3.27$, $p = 0.07$	$\chi^2 = 0.01$, $p = 0.93$	$\chi^2 = 4.13$, $p = 0.04$	$\chi^2 = 4.35$, $p = 0.04$	$\chi^2 = 1.76$, $p = 0.19$
Aversions	$\chi^2 = 2.10$, $p = 0.15$	$\chi^2 = 0.22$, $p = 0.88$	$\chi^2 = 0.06$, $p = 0.81$	$\chi^2 = 1.14$, $p = 0.29$	$\chi^2 = 0.09$, $p = 0.77$
Nausea and Vomiting	$\chi^2 = 2.87$, $p = 0.09$	$\chi^2 = 3.22$, $p = 0.07$	$\chi^2 = 0.43$, $p = 0.52$	$\chi^2 = 3.71$, $p = 0.05$	$\chi^2 = 0.004$, $p = 0.95$

**Note.* $df = 1$ and $N = 198$ for all tests.

Exploratory Analyses for Specific Complications. Finding mixed support for my preliminary hypotheses, I decided to explore the possibility that changes in eating behavior and their associations with pregnancy complications are specific to the pregnancy complication in question. To do this, I assessed whether the associations differed when I considered each pregnancy complication separately.

First, I assessed the associations between cravings and pregnancy complications. I found that women who reported craving fetal-favoring foods reported that they experienced high blood pressure, χ^2 (1, N = 198) = 5.25, p = 0.02. Extending the predictions made by the prophylaxis hypothesis where women were more likely to experience nausea and vomiting in response to what I have named maternal-favoring foods, women who reported craving maternal-favoring foods reported experiencing a pregnancy loss/ miscarriage, χ^2 (1, N = 198) = 6.73, p = 0.009. In addition, women who reported craving antimicrobial foods reported experiencing infections during pregnancy, χ^2 (1, N = 198) = 8.02, p = 0.005. Finally, women who reported having cravings for probiotic foods reported experiencing high blood pressure, χ^2 (1, N = 198) = 3.89, p = 0.05.

Next, I assessed the associations between aversions and pregnancy complications. I found that women who reported that they found fetal-favoring foods to be aversive also reported that they experienced high blood pressure, χ^2 (1, N = 198) = 8.27, p = 0.004, preeclampsia, χ^2 (1, N = 198) = 8.82, p = 0.003, or stillbirth, χ^2 (1, N = 198) = 4.85, p = 0.03. In addition, women who reported that they found foods that were good for both mom and fetus to be aversive reported that they experienced preterm labor, χ^2 (1, N = 198) = 5.58, p = 0.02, or stillbirth, χ^2 (1, N = 198) = 5.63, p = 0.02. Due to the low number of women who experienced a stillbirth, however, the results pertaining to stillbirths should be interpreted with great caution until they can be replicated in future research with a larger sample of women who have experienced a stillbirth.

Lastly, I assessed the associations between nausea and vomiting and pregnancy complications. I found that women who reported experiencing nausea and vomiting in

response to fetal-favoring foods reported that they experienced preeclampsia, χ^2 (1, N = 198) = 9.76, p = 0.002. Together, these results suggest that it is important for future investigations on this topic to consider the influence of genetic conflict on pregnancy complications separately instead of in aggregate as has been done in previous work testing the prophylaxis hypothesis (i.e., nausea and vomiting is protective against pregnancy complications).

CHAPTER 3

STUDY 2

The purpose of this study was to replicate the results from Study 1 and test whether changes in eating behavior during pregnancy are an index of genetic conflict that can be used to predict pregnancy complications. There were a few limitations to the design of Study 1 that need to be addressed in this study to fully assess the impact of changes in eating behavior on the likelihood of experiencing pregnancy complications. For example, in Study 1,changes in eating behavior were classified as dichotomous variables, so the chi-square association tests could detect associations between changes in eating behavior and pregnancy complications but not the direction of these relationships. In addition, the proportion of women who experienced these pregnancy complications was much smaller than the total sample. The small sample of women who experienced each complication may have caused the analyses in Study 1 to be underpowered and not accurately reflective of the underlying relationships. To address these limitations, Study 2 used the same survey as Study 1 with minor methodological modifications and a larger sample.

Given the associations found Study 1, Study 2 focuses on the relationships between changes in eating behavior and four pregnancy complications: high blood pressure, preeclampsia, infections during pregnancy, and gestational diabetes. While Study 1 did not find clear associations between changes in eating behavior and gestational diabetes, the best accepted explanation for why women experience gestational diabetes is that there is conflict between maternal and paternal genes over the production of insulin/ insulin-like growth factors. As such, gestational diabetes was included in

Study 2 to continue investigating the associations between changes in eating behavior and this pregnancy complication. I hypothesized that stronger cravings for or aversions towards fetal-favoring foods should be associated with experiencing high blood pressure. I also hypothesized that stronger cravings for antimicrobial foods should be associated with experiencing infections during pregnancy. Finally, I hypothesized that stronger aversions towards or nausea and vomiting in response to fetal-favoring foods should be associated with experiencing preeclampsia.

In addition to these hypotheses, I also wanted to confirm that the categories of foods I created accurately represented the variance in eating behavior. This led me to perform confirmatory factor analyses to document that these food categories were appropriate to use in these studies. To the best of my knowledge, these analyses represent the first factor analytic assessment of food categories in the literature on changes in eating behavior during pregnancy.

Method

Participants. To ensure that the sample collected for this study would be large enough to detect the predicted effects, I ran *a priori* power analyses using the pregnancy complications of interest (i.e., gestational diabetes, high blood pressure, infections during pregnancy, and preeclampsia). These complications were chosen as the complications of interest for the current study based on previous literature (e.g., Haig, 1993) and my previous research documenting that these complications are influenced by genetic conflict. As there are no prior empirical investigations that link changes in eating behavior to the likelihood of experiencing pregnancy complications, I used the effect

sizes from Study 1 as the basis for these power analyses. The calculated effect sizes (transformed from chi-square associations to *r*) were between a small and medium effect size using large using Cohen's (1988) classifications. But, like stated earlier, it is possible that the effect sizes may be overestimated due to small sample size of women who reported experiencing each complication. As such, I use the required sample sizes as the minimum number of participants that need to be recruited.

Using a traditional significant level of alpha = 0.05, power = 0.95, and average estimated precision (an estimate of the margin of error in the likelihood of experiencing the complication), the power analyses suggest that I needed a minimum sample size of 208 women to detect the effects of genetic conflict on high blood pressure, 194 women to detect the effects of genetic conflict on infections during pregnancy, and 191 women to detect the effects of genetic conflict on preeclampsia. Because the effects for detecting genetic conflict on gestational diabetes were not detected in Study 1 but the complication was included in this investigation for theoretical reasons, I do not have an estimate for the effect size needed to detect this hypothesized effect. However, since pregnancy complications can occur at any time during pregnancy in women even if they have no history of complications, pregnancy complications cannot be used as a priori criteria for recruitment. In order to give me the highest chance of recruiting enough women to detect these effects, I decided to recruit a minimum of 300 currently pregnant women to increase the chances of recruiting a sufficient sample of women who have experienced each complication.

Data for Study 2 is comprised of two samples that participated in the same survey at different time points (Spring 2019 N_1 = 249, Fall 2021 N_2 = 341; N_{total} = 590; See

Appendix B for descriptions of the samples separately). Participants were recruited through Prolific.co to participate in a 10-15 minute survey about dietary changes during pregnancy and were compensated for their time. The only inclusion criteria were that the participants must be 1) female, 2) fluent in English, 3) did not participate in Study 1, and 4) are currently pregnant (sample 2) or have been pregnant within the last two years (sample 1). All participants passed the attention checks and were included in the analyses assessing the changes in eating behavior factor structures (M_{age} = 29.89 years, SD_{age} = 5.36 years; 65.3% White; 74.2% had some college education; 360 women were currently pregnant).

Due to slight differences in the designs for the two samples (i.e., sample 2 had currently pregnant women participate at two time points to capture their current pregnancy eating behavior and pregnancy complications), not all women reported the pregnancy complications they experienced during this pregnancy. As a result, the analyses that assess the likelihood of experiencing pregnancy complications have a final sample size of 337 women (M_{age} = 30.81 years, SD_{age} = 5.46 years; 74.5% White; 74.8% had some college education; 107 women were currently pregnant). In this sample, 89 (26.4%) women reported experiencing high blood pressure, 42 (12.5%) women reported experiencing gestational diabetes, 25 (7.4%) women reported experiencing infections during pregnancy, and 29 (8.6%) women reported experiencing preeclampsia.

Materials & Procedure. The participants in this study completed the same survey as participants in Study 1 with some minor modifications. First, I added additional pregnancy complications that participants from Study 1 reported that they had experienced (i.e., placental problems) but were not captured by the existing list of

complications. Second, I added the category of "salty foods" to the antimicrobial category of foods because women in Study 1 indicated that this was a category of food that they experienced changes in eating behavior towards. I decided to add "salty foods" to the antimicrobial category as salt is commonly used to preserve meats and stop the growth of pathogenic organisms (Institute of Medicine, 2010).

Additionally, I changed the response scales for the eating behavior questions from a dichotomous option (*Yes, I experienced this craving/aversion/nausea/vomiting* or *No, I did not experience this craving/aversion/nausea/vomiting*) to a Likert-scale (1 = *I had very weak craving/aversion/nausea/vomiting for this during pregnancy* to 7 = *I had extreme craving/aversion/nausea/vomiting for this during pregnancy*) so I could use the strength of these changes to predict experiencing pregnancy complications. Finally, the nausea and vomiting category from Study 1 was split into two categories because some women reported having nausea without vomiting and vomiting without nausea which suggests that nausea and vomiting during pregnancy. The fact that not all women experience nausea and vomiting concurrently suggests that they may serve different functions and therefore provide different assessments of a woman's likelihood of experiencing pregnancy complications. After completing the survey, participants were given an open ended text box to provide any additional information about their changes in eating behavior and thanked for their time and participation.

Results

Before moving on to the focal replication analyses for this study, I wanted to ensure that the food categories I planned to use accurately captured the variance in

pregnancy eating behavior. I followed the protocol outlined by Byrne (2010), DeVellis (2016), and Mueller and Hancock (2001) for using confirmatory factor analyses instead of exploratory factor analyses to assess the factor structure because my aim was to assess the appropriateness of a specific and theoretically-derived measurement model. As previous literature does not have other theoretically-derived models for me to assess, I am not able to investigate other potential models for changes in pregnancy eating behaviors. If the five-factor model does not achieve adequate fit as derived from Hu & Bentler's (1999) cut offs, I investigated the modification indices to determine which items caused the model misspecification and if model fit could be improved with the addition of new covariance paths (Bollen, 1989; DeVillis, 2016).

Cravings confirmatory factor analyses. I performed the confirmatory factor analysis on the desirable traits in R using lavaan (Rosseel, 2012). Since chi-square statistics are known to be significant in large samples (Cheung & Rensvold, 2002), I assessed all fit measurements to determine if the theoretically derived five-factor model adequately fit the data (Hu & Bentler, 1999). The results of the confirmatory factor analysis suggested that the five-factor model of cravings during pregnancy fit the data well, χ^2 (314) = 726.42, $p < 0.001$, CFI = 0.78, TLI = 0.77, SRMR = 0.07, RMSEA = 0.066, 90% CI [0.069, 0.072]. Modification indices were not assessed due to the adequate fit of the model (Byrne, 2010). Figure 1 depicts the path diagram for this confirmatory analysis and Table 4 shows the covariances between latent factors.

Figure 1. Path model representing the structure of cravings during pregnancy.

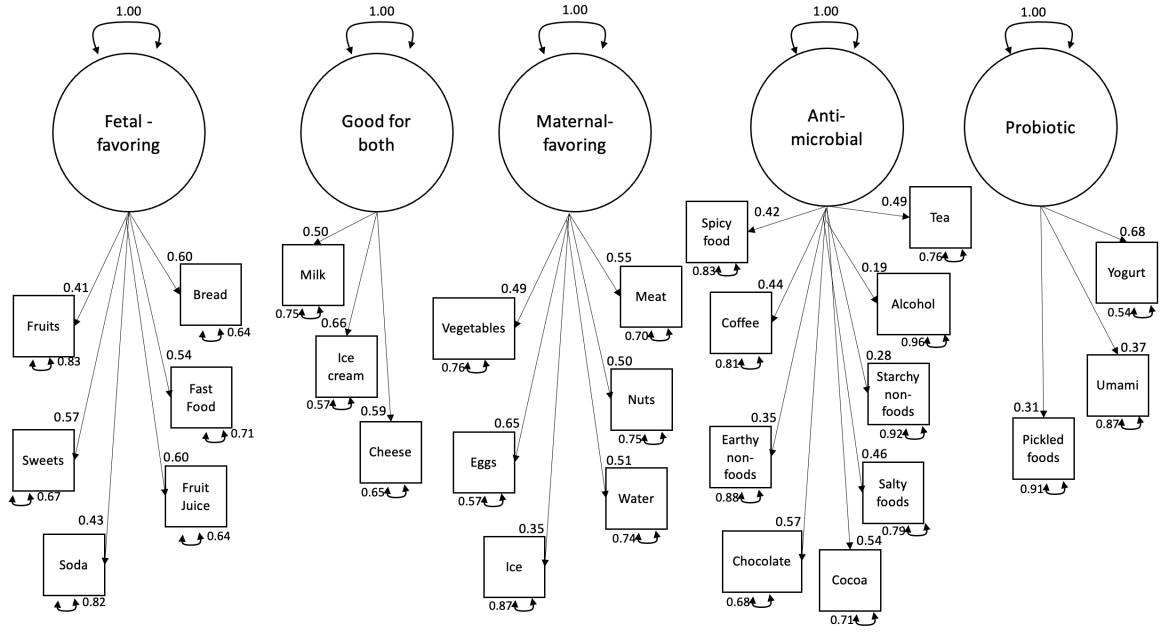

Table 4. Latent variable covariances from the cravings during pregnancy confirmatory factor analyses.

	Fetal- favoring foods	Good for both foods	Maternal-favoring foods	Antimicrobial foods	Probiotic foods
Fetal-favoring foods	--				
Good for both foods	0.90	--			
Maternal-favoring foods	0.75	0.77	--		
Antimicrobial foods	0.95	0.92	0.80	--	
Probiotic foods	0.79	0.97	1.01	0.92	--

**Note.* Covariances are not bounded within +/- 1 range (DeVellis, 2016).

Aversions confirmatory factor analyses. Next, I assessed the model fit for aversions during pregnancy. The results of the confirmatory factor analysis suggested that the five-factor model of aversions during pregnancy was slightly above the criteria for

acceptable fit, χ^2 (314) = 1042.19, p < 0.001, CFI = 0.68, TLI = 0.64, SRMR = 0.08, RMSEA = 0.09, 90% CI [0.08, 0.10]. Due to the lack of acceptable fit, I then investigated the modification indices to determine misspecification (modification indices >10) and improve model fit (Byrne, 2010). Using this criteria, I added two residual covariance paths (one between aversions towards earthy non-food substances and starchy non-food substances, and one between aversions towards sweets and chocolate) to the model and reassessed the model fit. The new model had acceptable fit, χ^2 (312) = 828.34, p < 0.001, CFI = 0.77, TLI = 0.74, SRMR = 0.08, RMSEA = 0.08, 90% CI [0.07, 0.09]. Figure 2 depicts the path diagram for this confirmatory analysis with the added covariance paths in red and Table 5 shows the covariances between latent factors.

Figure 2. Path model representing the structure of aversions during pregnancy.

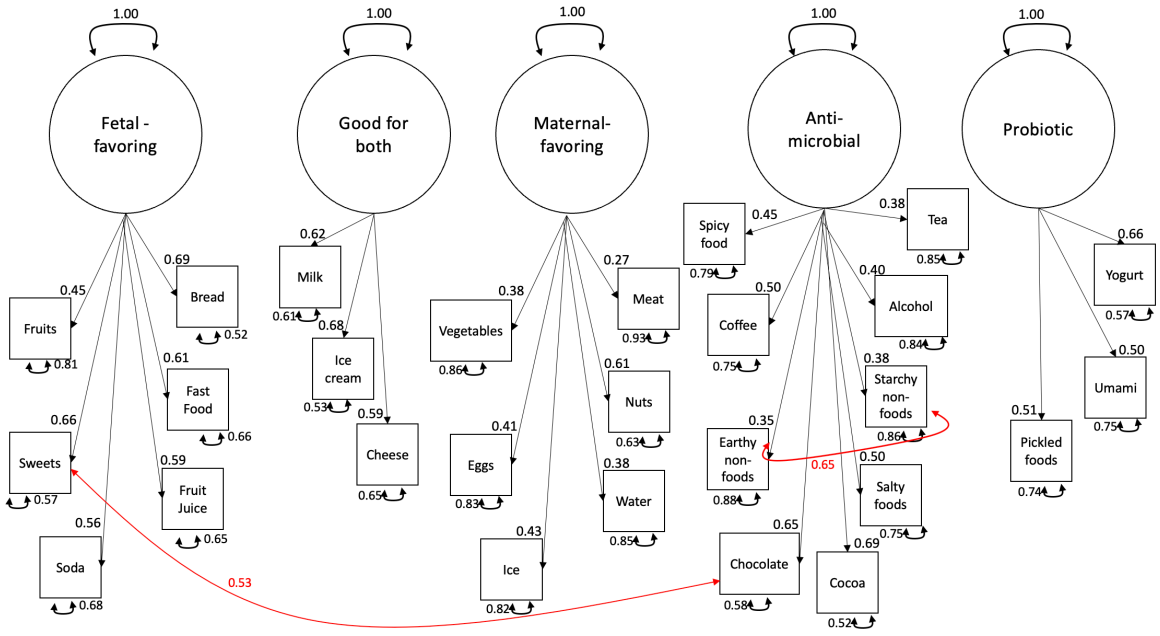

Table 5. Latent variable covariances from the aversions during pregnancy confirmatory factor analyses.

	Fetal- favoring foods	Good for both foods	Maternal-favoring foods	Antimicrobial foods	Probiotic foods
Fetal-favoring foods	--				
Good for both foods	0.76	--			
Maternal-favoring foods	0.81	0.87	--		
Antimicrobial foods	0.87	0.86	0.93	--	
Probiotic foods	0.63	0.92	0.92	0.84	--

Nausea confirmatory factor analyses. Next, I assessed the model fit for nausea during pregnancy. The results of the confirmatory factor analysis suggested that the five-factor model of aversions during pregnancy adequately fit the data, χ^2 (314) = 1002.65, p < 0.001, CFI = 0.82, TLI = 0.80, SRMR = 0.06, RMSEA = 0.08, 90% CI [0.075, 0.087]. Modification indices were not assessed due to the adequate fit of the model (Byrne, 2010). Figure 3 depicts the path diagram for this confirmatory analysis and Table 6 shows the covariances between latent factors.

Figure 3. Path model representing the structure of nausea during pregnancy.

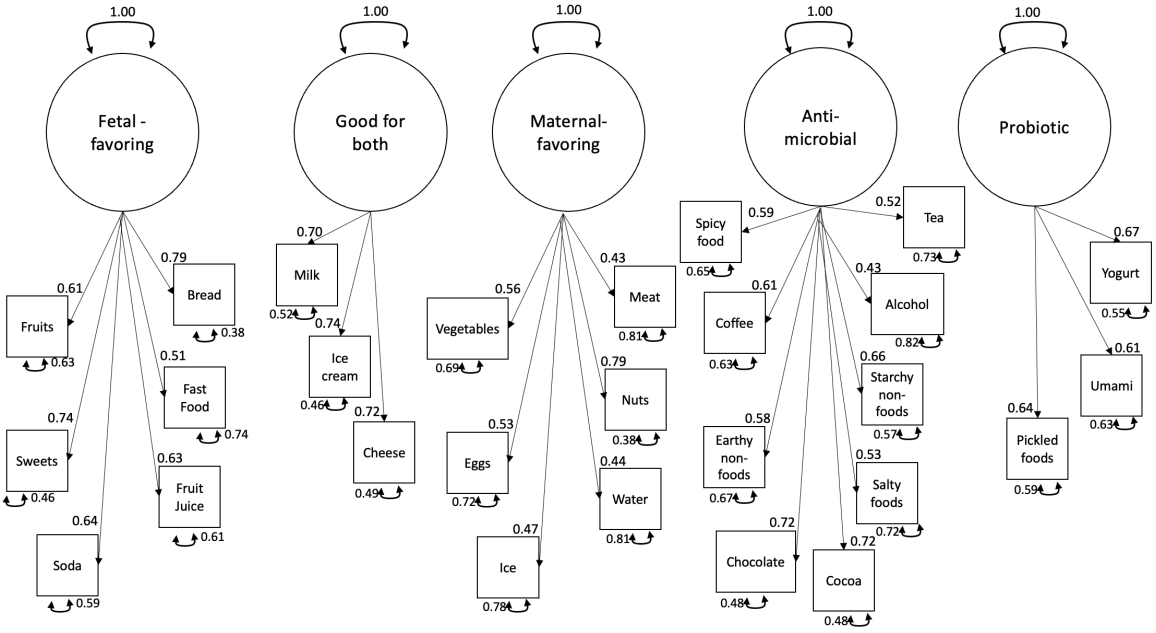

Table 6. Latent variable covariances from the nausea during pregnancy confirmatory factor analyses.

	Fetal- favoring foods	Good for both foods	Maternal-favoring foods	Antimicrobial foods	Probiotic foods
Fetal-favoring foods	--				
Good for both foods	0.89	--			
Maternal-favoring foods	0.82	0.85	--		
Antimicrobial foods	0.93	0.89	0.94	--	
Probiotic foods	0.74	0.94	0.96	0.86	--

Vomiting confirmatory factor analyses. Next, I assessed the model fit for vomiting during pregnancy. The results of the confirmatory factor analysis suggested that the five-factor model of vomiting during pregnancy was slightly above the criteria for

acceptable fit, χ^2 (314) = 909.80, p < 0.001, CFI = 0.82, TLI = 0.80, SRMR = 0.07, RMSEA = 0.096, 90% CI [0.089, 0.104]. Due to the lack of acceptable fit, I investigated the modification indices to determine misspecification. Using this criteria, I added four residual covariance paths (one between vomiting in response to meats and umami flavors, one between vomiting in response to ice and earthy non-food substances, one between vomiting in response to earthy non-food substances and starchy non-food substances, and one between vomiting in response to ice cream and chocolate) to the model and reassessed the model fit. The new model had acceptable fit, χ^2 (310) = 752.09, p < 0.001, CFI = 0.87, TLI = 0.85, SRMR = 0.08, RMSEA = 0.08, 90% CI [0.076, 0.091]. Figure 4 depicts the path diagram for this confirmatory analysis with the added covariance paths in red and Table 7 shows the covariances between latent factors.

Figure 4. Path model representing the structure of vomiting during pregnancy.

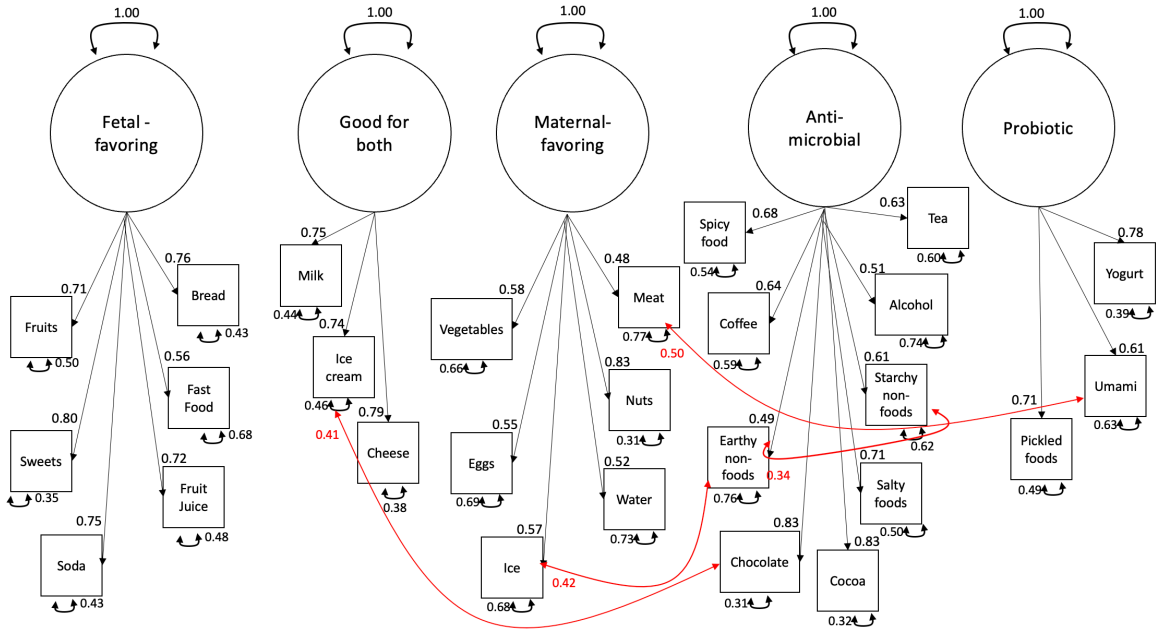

Table 7. Latent variable covariances from the vomiting during pregnancy confirmatory factor analyses.

	Fetal- favoring foods	Good for both foods	Maternal-favoring foods	Antimicrobial foods	Probiotic foods
Fetal-favoring foods	--				
Good for both foods	0.90	--			
Maternal-favoring foods	0.92	0.90	--		
Antimicrobial foods	0.94	0.94	0.96	--	
Probiotic foods	0.82	1.00	0.91	0.97	--

While the results of these analyses show that the five-factor model satisfactorily captures the variances in changes in eating behavior during pregnancy (with minor modifications) for this sample, there is an important consideration that should be discussed with these results. Across all for confirmatory factor analyses, there were high covariances between the latent factors (shown in Tables 4 – 7) and high residual variances of the measured variables (shown in Figures 1 – 4 with the small curved black arrows). These attributes of the models provide two interesting insights into the nature of changes in eating behavior during pregnancy. First, the high covariances between latent factors suggests that these changes in eating behavior do not happen independently (see Appendix C for a correlation matrix of the latent variables). While this lack of independence has been assumed within the prophylaxis hypothesis framework (e.g., between aversion, nausea, and vomiting), these results provide novel information that this lack of independence can be observed across different functional categories of foods.

Second, the high residual variances at the measured variable level suggest that while some of the variance of changes in cravings, aversions, nausea and vomiting during pregnancy are captured by these food categories, there is additional variance in these changes that is not due to these categories.

Interestingly, the results showed that cravings for these food categories were not independent and were negatively correlated with the food categories that caused aversions, nausea, and vomiting. The nature of these relationships supports the compensatory model of cravings during pregnancy and suggests that cravings may be designed to "make up" for any missing nutrients in the maternal diet during pregnancy. Previous research has only investigated the compensatory model of cravings in the context of pica during pregnancy (Mills, 2007), so future research should consider utilizing this model to understand under what circumstances the compensatory model explains changes in eating behavior.

Changes in eating behavior predicting pregnancy complications. After confirming that the theoretically derived food categories captured the variability in my participants' eating behavior, I then performed the focal logistic regressions to investigate if the strength of the changes in eating behaviors influenced the likelihood of experiencing pregnancy complications. I focused these analyses on the pregnancy complications from Study 1 that appeared to be influenced by genetic conflict (infections, high blood pressure, preeclampsia, and gestational diabetes). To control for the fact that changes in cravings, aversions, nausea, and vomiting towards these food categories are

highly correlated, the effects of each change in eating behavior are reported controlling for all other changes in eating behavior in that analysis [2].

First, I investigated which changes in eating behavior were associated with experiencing high blood pressure (see Table 8 for coefficient estimates). Given the results of the previous studies, I predicted that stronger cravings for or aversions towards fetal-favoring foods should be associated with experiencing high blood pressure. Women who reported having stronger cravings for maternal-favoring foods were more likely to experience high blood pressure during pregnancy after controlling for all other reported changes in cravings. There were no effects of aversions, nausea, or vomiting in response to these foods categories on the likelihood of experiencing high blood pressure during pregnancy.

Table 8. Changes in eating behavior unstandardized coefficients that were associated with experiencing high blood pressure during pregnancy in Study 2.

	Fetal-favoring foods	Foods that were good for both	Maternal-favoring foods	Antimicrobial foods	Probiotic foods
Cravings	-0.29	0.02	0.46*	0.17	-0.18
Aversions	0.23	0.06	-0.17	-0.03	-0.07
Nausea	0.10	-0.13	-0.08	-0.17	0.07
Vomiting	-0.20	0.13	0.06	0.22	-0.11

**Note.* $^{+}$ $p < 0.10$. *, $p < 0.05$. ** $p < 0.01$. *** $p < 0.001$.

I then investigated which changes in eating behavior were associated with experiencing gestational diabetes (see Table 9 for coefficient estimates). As this

[2] See Appendix D for logistic regressions with age included as a covariate to control for the possibility that age may be associated with and increased likelihood of experiencing these pregnancy complications. See Appendix E for logistic regressions where a within participants correction was applied before the analysis so the changes in eating behavior in the analysis removed the participant's average change in eating behavior to control for the possibility that participants reported more changes in eating behavior simply because they ate more during pregnancy.

complication did not show effects in Study 1, I did not have a priori hypotheses. Women who reported having weaker cravings for fetal-favoring foods were marginally more likely to experience gestational diabetes after controlling for all other reported changes in cravings. Women who reported stronger aversions towards fetal-favoring foods were marginally more likely to experience gestational diabetes after controlling for all other reported changes in aversions. Finally, women who reported stronger vomiting reactions in response to fetal-favoring foods were more likely to experience gestational diabetes after controlling for all other reported changes in vomiting. There were no effects of nausea in response to these food categories on the likelihood of experiencing gestational diabetes.

Table 9. Changes in eating behavior unstandardized coefficients that were associated with experiencing gestational diabetes during pregnancy in Study 2.

	Fetal-favoring foods	Foods that were good for both	Maternal-favoring foods	Antimicrobial foods	Probiotic foods
Cravings	-0.46^{+}	-0.06	0.24	0.07	0.03
Aversions	0.53^{+}	-0.14	-0.14	-0.43	0.26
Nausea	0.33	-0.04	-0.04	-0.35	0.08
Vomiting	0.83*	-0.54	0.39	-0.99	0.14

**Note.* $^{+}$ $p < 0.10$. *, $p < 0.05$. ** $p < 0.01$. *** $p < 0.001$.

Next, I investigated which changes in eating behavior were associated with experiencing infections during pregnancy (see Table 10 for coefficient estimates). Given the results of the previous studies, I hypothesized that stronger cravings for antimicrobial foods or aversions towards probiotic foods should be associated with experiencing infections during pregnancy. Unexpectedly, women who reported weaker cravings for foods that are good for both were more likely to experience infections during pregnancy after controlling for all other cravings. In addition, women who reported weaker vomiting

in response to maternal-favoring foods were more likely to experience infections during pregnancy after controlling for all other changes in vomiting. There were no effects of aversions or nausea in response to these food categories on the likelihood of experiencing infections during pregnancy.

Table 10. Changes in eating behavior unstandardized coefficients that were associated with experiencing infections during pregnancy in Study 2.

	Fetal-favoring foods	Foods that were good for both	Maternal-favoring foods	Antimicrobial foods	Probiotic foods
Cravings	0.33	-0.57*	0.20	0.67	-0.42
Aversions	0.04	0.16	0.24	-0.46	0.27
Nausea	-0.15	-0.12	0.20	0.25	0.02
Vomiting	0.26	0.42	-1.74*	0.48	0.33

**Note.* $^{+}$ $p < 0.10$. *, $p < 0.05$. ** $p < 0.01$. *** $p < 0.001$.

Finally, I investigated which changes in eating behavior were associated with experiencing preeclampsia during pregnancy (see Table 11 for coefficient estimates). Given the results of the previous studies, I hypothesized that stronger aversions towards maternal-favoring foods or vomiting in response to maternal-favoring foods should be associated with experiencing preeclampsia during pregnancy. I found partial support for this hypothesis. Women who reported weaker vomiting in response to fetal-favoring foods were marginally more likely to experience preeclampsia during pregnancy after controlling for all changes in vomiting towards the other food categories. Women who reported stronger vomiting in response to maternal-favoring foods were more likely to experience preeclampsia during pregnancy after controlling for all other changes in vomiting. There were no effects of cravings, aversions, or nausea in response to these food categories on the likelihood of experiencing preeclampsia during pregnancy.

Table 11. Changes in eating behavior unstandardized coefficients that were associated with experiencing preeclampsia during pregnancy in Study 2.

	Fetal-favoring foods	Foods that were good for both	Maternal-favoring foods	Antimicrobial foods	Probiotic foods
Cravings	-0.13	0.19	-0.04	-0.23	-0.16
Aversions	-0.07	-0.23	0.16	-0.06	0.08
Nausea	-0.06	0.13	-0.13	-0.07	-0.08
Vomiting	-0.92^{+}	0.16	1.54**	-0.30	-0.13

**Note*. $^{+}$ $p < 0.10$. *, $p < 0.05$. ** $p < 0.01$. *** $p < 0.001$.

Taken together, these results provide partial support for my hypotheses. Specifically, Study 2 replicated the relationship between experiencing stronger vomiting in response to maternal-favoring foods and experiencing preeclampsia. Study 2 did not replicate the relationships between experiencing stronger cravings for fetal-favoring foods and experiencing high blood pressure, stronger cravings for antimicrobial foods and experiencing infections, stronger aversions to probiotic foods and experiencing infections, and stronger aversions towards maternal-favoring foods and experiencing preeclampsia.

In addition, Study 2 documented preliminary evidence of the influence of genetic conflict on additional pregnancy complications. Specifically, Study 2 documented that weaker cravings for fetal-favoring foods, stronger aversions towards fetal-favoring foods, or stronger vomiting in response to fetal-favoring foods were associated with experiencing gestational diabetes. These relationships suggests that maternal interests may be 'winning' in the eating behavior manipulation, which perhaps causes the fetus to manipulate the insulin regulation system to get the resources it needs. Stronger cravings for maternal-favoring foods were associated with experiencing high blood pressure during pregnancy. Weaker cravings towards foods that are good for both or weaker

vomiting in response to maternal-favoring foods were associated with experiencing infections during pregnancy, and weaker vomiting in response to fetal-favoring foods was associated with experiencing preeclampsia. In all, these results suggest that when maternal interests are prioritized in resource transfers during pregnancy, women are more likely to experience pregnancy complications such as high blood pressure, gestational diabetes, infections, and preeclampsia during pregnancy.

CHAPTER 4

DISCUSSION

Summary of Findings

This investigation presents two studies that use a genetic conflict framework to explore the connections between changes in eating behavior and pregnancy complications. Taken together, the results of these studies provide the first empirical investigation of genetic conflict expressed through changes in eating behavior, and document that conflict over resource transfers through changes in eating behavior during pregnancy can be used to predict a woman's likelihood of experiencing pregnancy complications.

In Study 1, I tested between predictions derived from the prophylaxis hypothesis which is the currently accepted explanation for changes in eating behavior during pregnancy, such as 1) cravings should be associated with experiencing pregnancy complications and 2) cravings for fetal-favoring foods, but not other food categories, should be associated with experiencing pregnancy complications. I then tested predictions derived from the genetic conflict framework for changes in pregnancy eating behavior by 3) exploring the relationships between specific changes is eating behavior and pregnancy complications. The results from this study largely suggested that broad generalizations about changes in eating behavior having a uniform influence on the likelihood of experiencing pregnancy complications are overstated (i.e., that nausea and vomiting are protective). Additionally, pregnancy complications such as high blood pressure, gestational diabetes, infections during pregnancy, and preeclampsia were influenced by changes in eating behavior towards specific functional categories of foods.

In Study 2, I aimed to replicate the associations observed in Study 1. Specifically, I wanted to provide the first factor analytic assessment of changes in eating behavior during pregnancy and replicate the relationships between 1) stronger cravings for fetal-favoring foods and high blood pressure, 2) stronger cravings for antimicrobial foods and infections, and 3) stronger aversions, nausea, and vomiting in response to fetal-favoring foods and preeclampsia. The results from this study partially supported these predictions. First, the five-factor model adequately described the categories of foods towards which pregnant women reported experiencing changes in eating behavior (with some minor modifications). Additionally, I replicated the relationship showing that stronger vomiting in response to maternal-favoring foods predicting an increased likelihood of experiencing preeclampsia.

However, Study 2 failed to replicate some of the effects from Study 1, namely that stronger cravings for or aversions towards fetal-favoring foods predicted high blood pressure, stronger cravings for antimicrobial foods predicted infections, and stronger aversions towards or nausea and vomiting in response to fetal-favoring foods predicted preeclampsia. Study 2 also provided preliminary evidence that changes in eating behaviors (specifically, 1) decreased cravings for foods that are good for both and decreased vomiting in response to maternal-favoring foods, and 2) decreased cravings for fetal-favoring foods, increased aversions to fetal favoring foods, and increased vomiting in response to fetal-favoring foods) were associated with increased likelihoods of experiencing gestational diabetes and infections during pregnancy, respectively. The relationship between genetic conflict (via changes in eating behaviors) and gestational diabetes is not surprising given that the underlying cause of gestational diabetes is

conflict between maternal and paternal genes over blood sugar (Haig, 1993), but the link between changes in eating behavior and infections is novel as infections are not generally thought to be the consequence of genetic conflict.

Taken together, the results of these studies provide preliminary empirical evidence that changes in eating behaviors can be used as an index of underlying genetic conflict during pregnancy. This investigation also provides the first factor analytic assessment of conflict over resource transfers during pregnancy through changes in eating behavior and documented that the five-factor model of food categories can be used to capture the variance in changes in eating behavior during pregnancy. This investigation also provides evidence that preeclampsia is associated with changes in eating behavior that I hypothesized to be indexes of genetic conflict over resources transfers during pregnancy. When fetal interests compensate for the maternal body trying to prioritize maternal interests during pregnancy by causing vomiting in response to maternal-favoring foods during pregnancy, women are at an increased likelihood of experiencing preeclampsia.

Future research on genetic conflict during pregnancy would benefit from incorporating this perspective of conflict over resource transfers to understand how conflict over resource transfers plays out in other areas of maternal behavior during pregnancy. Additionally, research on pregnancy complications in general would benefit from using this genetic conflict framework of changes in eating behavior because changes in eating behavior and food preferences are a promising way to measure changes during pregnancy that are hard to see externally but can have a large impact on pregnancy

outcomes (e.g., which side is being prioritized in resources transfers, if conflict escalation has occurred).

Limitations

While this investigation has many strengths, there are some limitations that need to be addressed. The first limitation of these studies deals with potentially biased recall of changes in eating behaviors. While this is a limitation of my investigations, it should be noted that post-pregnancy dietary recalls are commonly used in research on dietary changes (Hook, 1976; 1978; though see Krall et al., 1988, for pitfalls of this method). Study 2 aimed to address the reliance on participant recall of changes in eating behaviors by recruiting a larger sample of pregnant women to recall these changes as they happened, but it is possible that participants still had biased reporting of these changes due to the social norms surrounding eating behaviors during pregnancy. For example, I asked participants to report their cravings for (but not consumption of) alcohol and coffee, so it is possible that participants did not accurately report their cravings, aversions, nausea, or vomiting in response substances such as these because they did not want to feel embarrassed or judged for consuming substances that pregnant women are told to actively avoid or consume in moderation. Additionally, since pregnancy weight gain is a source of shame for many women and predisposes them for negative mental health outcomes postpartum (Incollingo Rodriquez et al., 2019; 2020), it is possible that women may have reported socially desirable changes in eating behavior while pregnant in order to avoid those feelings. Future studies could address this limitation by engaging in longitudinal experience sampling of pregnancy eating behaviors to increase the

accuracy of these reported changes. This longitudinal approach would also allow researchers to assess if the trimester that these changes in eating behavior occur in is important for predicting pregnancy outcomes. Additionally, future research should incorporate survey measures that help decrease the stigmatization of pregnancy eating behavior changes and weight gain or connect participants to counseling services after the survey to ensure that participants do not feel judged for their responses.

Another limitation is that these studies did not assess potential moderators that may influence the relationship between changes in eating behaviors and experiencing pregnancy complications. For example, previous research has documented individual differences in socioeconomic status influences pregnant women's diet quality (Li et al., 2019) so it is possible that these differences in diet quality also interact with changes in eating behavior and mitigate the extent of genetic conflict over resource transfers that women with better quality diets face. To assess this possibility, I reran the analyses including educational attainment (a proxy for socioeconomic status) as a potential predictor. When I controlled for educational attainment, the results remained largely unchanged[3]. Additionally, women facing nutritional transitions (such as immigrating to a Westernized culture or transitioning from a traditional to Western diet; Higginbottom et al., 2011) may experience more conflict over pregnancy eating behavior since they may no longer have access to their traditional foods and therefore may have to rely on heavily processed and fetal-favoring foods such as fast foods. This reliance on non-nutritious food may increase the likelihood that these women experience pregnancy complications

[3] See Appendix F for logistic regressions with educational attainment included as a covariate to assess if educational attainment influenced the likelihood of experiencing these pregnancy complications.

as well since it could increase the likelihood that there is conflict over resource transfers during pregnancy. Similarly, women who take additional supplements such as prenatal vitamins may experience fewer conflicts over resource transfers during pregnancy because these supplements are designed to ensure that there are sufficient limiting nutrients to sustain the pregnancy. It is not clear how technological advances such as this impact the extent of underlying genetic conflict and, by extension, the likelihood of experiencing pregnancy complications.

The final limitation of these studies is a theoretical limitation. While the current investigations have incorporated the genetic conflict framework into the study of changes in eating behaviors and pregnancy compilations, the current studies are unable to assess if the mere existence of genetic conflict is what drives this relationship or if it is the escalation of conflict between maternal and fetal interests that drives this relationship. While this may not seem like a large limitation given that I was able to detect the effects of genetic conflict on the likelihood of experiencing pregnancy complications, this may limit the situations to which these results can be generalize.

For example, pregnancy is characterized as a time of insulin-resistance and elevated blood pressure (Bello et al., 2021; Sonagra, Biradar, K, & Murthy, 2014). This implies that there is always some conflict over these systems across the course of pregnancy. But long-term insulin resistance from insulin-like growth factors can lead to gestational diabetes (Zhu et al., 2016), and increased blood flow through insufficiently developed spiral arteries can lead to preeclampsia (Redman & Sargent, 2005). These biological realities lead to two potential interpretations of how genetic conflict relates to pregnancy complications. On one side, it could be interpreted that the negotiations

between maternal and fetal interests during pregnancy are analogous to a one-shot prisoner's dilemma where both sides must make the decision between behaving selfishly or cooperating for mutually beneficial outcomes (Axelrod, 1980). In this case, whether or not a woman experiences pregnancy complications is dependent on the strategies she and the fetus use in this one shot interaction.

On the other hand, it could be interpreted that the negotiations between maternal and fetal interests during pregnancy are more analogous to an iterated prisoner's dilemma. Both sides have to balance the short-term benefits of selfish behavior with the longer-term benefits of cooperation (Stephens et al., 2002). Pregnancy complications could then be thought of as the consequence of the mother or fetus choosing to defect across multiple rounds of negotiations. This perspective would imply that the severity of the complications experienced during pregnancy is the direct result of the number of negotiations where defection was favored over cooperation. This explanation is the accepted explanation for preeclampsia, such that preeclampsia is caused by the escalation of conflict between maternal and fetal interests over blood pressure across the course of pregnancy. Future research on other pregnancy complications could assess these alternative explanations by incorporating longitudinal assessment of biological markers in pregnancy where these negotiations take place (e.g., placental invasion, remodeling of spiral arteries, elevated blood pressure, elevated blood sugar) to assess how cooperation and conflict during these negotiations impacts a mother's likelihood of developing these pregnancy complications.

Pregnancy Complications and Postpartum Health

The fact that cooperation exists during pregnancy when mother and fetus have different fitness interests speaks to the fact that pregnancy is a largely cooperative venture peppered with instances of conflict. While these conflictual interactions are ubiquitous and normally result in typical growth and development (Ruvinsky, 1999), these conflictual interactions are also implicated in pregnancy complications. However, the current medical assessment of pregnancy complications is *reactive* – doctors can only detect complications once they already exist and are potentially dangerous for both mother and fetus. There is very little *proactive* assessment and prevention of potential complications.

The lack of *proactive* assessment and prevention of pregnancy complications is surprising as experiencing pregnancy complications is associated with negative psychological outcomes such as increased risk of postpartum depression (Burger et al., 1993), post-traumatic stress (Ryding et al., 1997), problems breastfeeding (Kozhimannil et al., 2014), and problems bonding with (Kokubu et al., 2012; Reading et al., 1984) and caring for newborns (Ashford, 2002; Lai et al., 2015; Tully & Ball, 2014). In addition, experiencing pregnancy complications such as gestational diabetes is associated with more self-reported negative perceptions of the one's own health, well-being, depression, and anxiety, and an increased risk of congenital abnormalities in the newborn (Daniells et al., 2003; Romon et al., 2001; Rumbold & Crowther, 2004; Wu et al., 2020).

Food preference monitoring during pregnancy is a promising *proactive* assessment that could help medical professionals screen pregnant women earlier in their pregnancy to prevent severe pregnancy complications before they occur. Specifically,

using changes in pregnancy eating behavior could help medical professionals engage in proactive risk stratification during pregnancy. This strategy may be particularly promising for pregnancy complications such as preeclampsia and gestational diabetes as the current study showed that changes in eating behaviors indexed the underlying genetic conflict over blood sugar and blood pressure that is associated with these pregnancy complications. However, as more research on changes in eating behavior during pregnancy is conducted, it is possible that these proactive risk stratifications can be extended to other pregnancy complications.

However, the proactive potential of assessing changes in pregnancy eating behavior does not end once the infant is born. Mothers who suffer from pregnancy complications also experience many long-lasting negative psychological outcomes. For example, we see that mothers who experience postpartum depression have a harder time interacting with and caring for their infants. The increased difficulty interacting with and caring for infants may, in turn, result in an insecure attachment between the infant and its mother (Carter et al., 2001). While previous research has robustly documented that infant attachment security is altered by the mother's ability to be attentive to the infant's needs (Bowlby, 1969, 1982), the novel connection here is that genetic conflict over resources and investment continues long after birth, influencing attachment (Crespi, 2011) and bonding between mother and infant (Davies, Isles, et al., 2008; Davies, Lynn, et al., 2008; MacDonald & MacDonald, 2010; Ross & Young, 2009). Extending this, the relationships between genetic conflict and infant attachment also implies that genetic conflict influences the infant's social interactions across its lifetime since attachment to primary

caregivers is believed to determine how the infant will relate to others across its lifetime (Bowlby, 1969; 1982).

Conclusion

This research assessed the possibility that changes in pregnancy eating behavior (which I hypothesized could act as an index of genetic conflict over resource transfers during pregnancy) could predict a woman's likelihood of experiencing pregnancy complications that are known to be related to genetic conflict and resource conflict. The results of these studies are consistent with some of the predictions from this framework. Across two studies, women who reported increased vomiting in response to maternal-favoring foods reported an increased likelihood of experiencing preeclampsia. Preliminary results also suggested that changes in eating behaviors were associated with an increased likelihood of experiencing high blood pressure, gestational diabetes, and infections during pregnancy, though these associations need to be replicated in future studies. Taken together, these results provide a first step towards providing a *proactive* assessment of pregnancy complications that can improve the long-term health and well-being of both the mother and the infant.

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APPENDIX A

DEFINITIONS OF CHANGES IN EATING BEHAVIOR

"**Cravings**" are a strong feeling or **desire** for specific foods or drinks, even if you do not consume these foods or drinks.

"**Aversions**" are a strong feeling of **dislike** or **desire** to avoid specific foods or drinks.

"**Nausea**" is a **feeling of sickness**, dizziness, and needing to expel the cause of the sickness from your body.

"**Vomiting**" is the act of **expelling** the cause of the sickness orally from your body.

APPENDIX B

DESCRIPTIONS OF THE TWO SAMPLES IN STUDY 2

Study 2A sample description

Participants. Participants (N = 249) were recruited through Prolific.co to participate in a 10-15 minute survey about dietary changes during pregnancy and were compensated $1.00 USD for their time. The only inclusion criteria were that the participants must be 1) female, 2) fluent in English, 3) did not participate in pilot Study 1, and 4) have been pregnant within the last two years, or currently are pregnant. All participants passed the attention checks and were included in the analyses (M_{age} = 31.25 years, SD_{age} = 5.30 years; 80.7% White; 76.3% had some college education; an average of 1 to 2 years since their most recent pregnancy).

Study 2B sample description.

Participants. Currently pregnant women (N = 382)[4] were recruited through Prolific.co to participate in time point 1 for this study and compensated $2.00 for 10 minutes of their time. The only inclusion criteria were that the participants must be 1) female, 2) fluent in English, 3) currently pregnant, and 4) agreed to be contacted in 4-6 months for a follow up study. After they had given birth, participants were asked to report their eating behavior right before giving birth and any pregnancy complications they experienced in this pregnancy. Of the 382 participants who completed the survey, 41 were removed from the analyses because they did not complete the survey. This left a final sample of 341 women (M_{age} = 28.91 years, SD_{age} = 5.19 years; 54.0% White; 72.8% had some college education; only 88 had given birth by the time of data collection).

[4] Due to an error on Prolific's end, women how were not currently pregnancy were allowed to participate in time point 1. This resulted in 551 women taking the first survey. Once women who were not currently pregnant were removed from the data, I was left with a total of 382 participants.

APPENDIX C

CORRELATION MATRIX OF CHANGES IN EATING BEHAVIOR

Figure S1. Correlation coefficients documenting the relationships changes in pregnancy eating behavior.

APPENDIX D

CHANGES IN EATING BEHAVIORS PREDICTING PREGNANCY COMPLICATIONS CONTROLLING FOR AGE

The analyses presented here test the possibility that the likelihood of experiencing pregnancy complications is influenced by participant age, such that older participants are more likely to experience pregnancy complications compared to younger participant. As a result, the effects of changes in eating behavior on the likelihood of experiencing pregnancy complications may be obscured by age. To assess this possibility, I conducted logistic regressions including age as a covariate in addition to the changes in eating behavior.

First, I investigated which changes in eating behavior were associated with experiencing high blood pressure after controlling for age (see Table S1 for coefficient estimates). Including age as a covariate did not change the observed results. Women who reported stronger cravings for maternal-favoring foods were more likely to experience high blood pressure after controlling for age and all other cravings. There was no effect of changes in aversions, nausea, and vomiting in response to these food categories on the likelihood of experiencing high blood pressure.

Table S1. Changes in eating behavior unstandardized coefficients that were associated with experiencing high blood pressure during pregnancy.

	Age	Fetal-favoring foods	Foods that were good for both	Maternal-favoring foods	Antimicrobial foods	Probiotic foods
Cravings	0.03	-0.29	0.01	0.48*	0.18	-0.17
Aversions	0.01	0.22	0.08	-0.15	-0.02	-0.09
Nausea	0.04	0.11	-0.12	-0.05	-0.14	0.04
Vomiting	-0.02	-0.21	0.14	0.07	0.19	-0.11

**Note.* $^{+}$ $p < 0.10$. *, $p < 0.05$. ** $p < 0.01$. *** $p < 0.001$.

Next, I investigated which changes in eating behaviors were associated with experiencing gestational diabetes after controlling for age (see Table S2 for coefficients

estimates). Including age as a covariate did not change the observed results. Women who reported weaker cravings for fetal-favoring foods or stronger aversions towards fetal-favoring foods were marginally more likely to experience gestational diabetes after controlling for age and all other cravings and aversions, respectively. Women who reported stronger vomiting in response to fetal-favoring foods were more likely to experience gestational diabetes after controlling for age and all other vomiting. There was no effect of changes in nausea in response to these food categories on the likelihood of experiencing gestational diabetes.

Table S2. Changes in eating behavior unstandardized coefficients that were associated with experiencing gestational diabetes during pregnancy.

	Age	Fetal-favoring foods	Foods that were good for both	Maternal-favoring foods	Antimicrobial foods	Probiotic foods
Cravings	0.01	-0.46^{+}	-0.06	0.15	0.08	0.03
Aversions	-0.01	0.52^{+}	-0.15	-0.15	-0.43	0.26
Nausea	0.03	0.33	-0.03	-0.01	-0.33	0.05
Vomiting	0.04	0.84*	-0.57	0.37	-0.96	0.15

**Note*. $^{+}$ $p < 0.10$. *, $p < 0.05$. ** $p < 0.01$. *** $p < 0.001$.

Then, I investigated which changes in eating behaviors were associated with experiencing infections during pregnancy after controlling for age (see Table S3 for coefficient estimates). Including age as a covariate did not change the observed results. Women who reported weaker cravings for foods that are good for both or weaker vomiting in response to maternal-favoring foods were more likely to experience infections during pregnancy after controlling for age and all other cravings and vomiting respectively. There were no effects of changes in aversions or nausea in response to these food categories on the likelihood of experiencing infections during pregnancy.

Table S3. Changes in eating behavior unstandardized coefficients that were associated with experiencing infections during pregnancy.

	Age	Fetal-favoring foods	Foods that were good for both	Maternal-favoring foods	Antimicrobial foods	Probiotic foods
Cravings	-0.02	0.33	0.58*	0.20	0.67	-0.42
Aversions	0.03	0.04	0.18	0.28	-0.47	0.26
Nausea	0.06	-0.13	-0.13	0.28	0.29	-0.03
Vomiting	-0.04	0.18	0.43	-1.64*	0.42	0.32

**Note.* $^{+}$ $p < 0.10$. *, $p < 0.05$. ** $p < 0.01$. *** $p < 0.001$.

Finally, I investigated which changes in eating behaviors were associated with experiencing preeclampsia during pregnancy after controlling for age (see Table S4 for coefficient estimates). Including age as a covariate did not change the observed results. Women who reported weaker vomiting in response to fetal-favoring foods were marginally more likely to experience preeclampsia during pregnancy after controlling for age and all other vomiting. Women who reported stronger vomiting in response to maternal-favoring foods were more likely to experience preeclampsia during pregnancy after controlling for age and all other vomiting. There were no effects of changes in cravings, aversions, or nausea in response to these food categories on the likelihood of experiencing preeclampsia during pregnancy.

Table S4. Changes in eating behavior unstandardized coefficients that were associated with experiencing preeclampsia during pregnancy.

	Age	Fetal-favoring foods	Foods that were good for both	Maternal-favoring foods	Antimicrobial foods	Probiotic foods
Cravings	0.02	-0.13	0.18	-0.03	-0.23	-0.15
Aversions	0.03	-0.07	-0.20	0.19	-0.07	0.07
Nausea	0.05	-0.05	0.14	-0.09	-0.06	-0.11
Vomiting	0.11	-0.89^{+}	0.02	1.64**	-0.23	-0.09

**Note.* $^{+}$ $p < 0.10$. *, $p < 0.05$. ** $p < 0.01$. *** $p < 0.001$.

APPENDIX E

CHANGES IN EATING BEHAVIORS PREDICTING PREGNANCY COMPLICATIONS AFTER REMOVING WITHIN PARTICIPANT AVERAGES

The analyses presented here test the possibility that the changes in eating behaviors reported by the participants in Study 2 were not caused by underlying genetic conflict but by other social forces. For example, it is possible that the changes in cravings women reported in this study were not due to genetic conflict over resource transfers during pregnancy but instead were due to it being socially acceptable for pregnant women to eat more food. If this is the case, the changes in cravings (as well as the other eating behaviors reported here) may be an artifact of the fact that pregnant women are just eating more food than when they were not pregnant.

To assess this possibility, I standardized the changes in eating behavior within-participant by subtracting the average change in reported eating behavior from the reported change in eating behavior towards each food category for each participant (i.e., fetal-favoring cravings – average change in cravings; maternal-favoring aversions – average change in aversions). These standardized changes in eating behaviors were then used as predictors in the logistic regressions. Unfortunately, this strategy introduced collinearity between the predictors and necessitated the removal of the changes in eating behaviors in response to probiotic foods to correct the collinearity. The results presented here assess changes in eating behaviors towards the remaining four categories of foods.

First, I investigated which changes in eating behavior were associated with experiencing high blood pressure after controlling for average within-subjects changes in eating behavior (see Table S5 for coefficient estimates). The results of this analysis were the same as the analysis presented in the main text. Women who reported experiencing stronger cravings for maternal-favoring foods were more likely to experience high blood pressure after controlling for all other cravings. There were no effects of changes in

aversions, nausea, or vomiting in response to these food categories on the likelihood of experiencing high blood pressure during pregnancy.

Table S5. Changes in eating behavior unstandardized coefficients that were associated with experiencing high blood pressure during pregnancy.

	Fetal-favoring foods	Foods that were good for both	Maternal-favoring foods	Antimicrobial foods
Cravings	-0.08	0.24	0.64*	0.23
Aversions	0.30	0.13	-0.11	0.04
Nausea	0.12	-0.20	-0.04	-0.16
Vomiting	-0.11	0.24	0.08	0.27

**Note*. $^{+}$ $p < 0.10$. *, $p < 0.05$. ** $p < 0.01$. *** $p < 0.001$.

Next, I investigated which changes in eating behaviors were associated with experiencing gestational diabetes after controlling for average within-subjects changes in eating behavior (see Table S6 for coefficients estimates). There were two differences in these analysis compared to the ones reported in the main text. Similar to the analyses in the main text, women who reported experiencing weaker cravings for fetal-favoring foods were more likely to experience gestational diabetes after controlling for all other cravings and there were no effects of changes in nausea in response to these food categories on the likelihood of experiencing gestational diabetes. However, unlike the analysis in the main text, there were no effects of changes in aversions and vomiting in response to these food categories on the likelihood of experiencing gestational diabetes.

Table S6. Changes in eating behavior unstandardized coefficients that were associated with experiencing gestational diabetes during pregnancy.

	Fetal-favoring foods	Foods that were good for both	Maternal-favoring foods	Antimicrobial foods
Cravings	-0.53^{+}	-0.14	0.12	0.23
Aversions	0.26	-0.42	-0.46	-0.73
Nausea	0.26	-0.11	-0.10	-0.42
Vomiting	-0.69	-0.69	0.35	-0.93

**Note*. $^{+}$ $p < 0.10$. *, $p < 0.05$. ** $p < 0.01$. *** $p < 0.001$.

Then, I investigated which changes in eating behaviors were associated with experiencing infections during pregnancy after controlling for average within-subjects changes in eating behavior (see Table S7 for coefficient estimates). There were two differences in these analysis compared to the ones reported in the main text. Similar to the analyses in the main text, women who reported experiencing weaker vomiting in response to maternal-favoring foods were more likely to experience infections during pregnancy after controlling for all other vomiting and there were no effects of changes on nausea in response to these food categories on the likelihood of experiencing infections during pregnancy. However, unlike the analysis in the main text, women who reported experiencing stronger cravings for fetal-favoring foods were more marginally likely to experience infections during pregnancy after controlling for all other cravings and women who reported experiencing weaker aversions towards antimicrobial foods were marginally more likely to experience infections during pregnancy after controlling for all other aversions.

Table S7. Changes in eating behavior unstandardized coefficients that were associated with experiencing infections during pregnancy.

	Fetal-favoring foods	Foods that were good for both	Maternal-favoring foods	Antimicrobial foods
Cravings	0.81^{+}	-0.08	0.67	1.02
Aversions	-0.30	-0.12	-0.20	-0.84^{+}
Nausea	-0.26	-0.13	0.09	0.19
Vomiting	-0.03	0.12	-1.59*	0.27

**Note.* $^{+}$ $p < 0.10$. *, $p < 0.05$. ** $p < 0.01$. *** $p < 0.001$.

Finally, I investigated which changes in eating behaviors were associated with experiencing preeclampsia during pregnancy after controlling for average within-subjects changes in eating behavior (see Table S8 for coefficient estimates). The results of this analysis were the same as the analysis presented in the main text. Women who reported weaker vomiting in responses to fetal-favoring foods were marginally more likely to experience preeclampsia after controlling for all other vomiting and women who reported stronger vomiting in response to maternal-favoring foods were marginally more likely to experience preeclampsia after controlling for all other vomiting. There were no effects of changes of cravings, aversions, and nausea in response to these food categories on the likelihood of experiencing preeclampsia.

Table S8. Changes in eating behavior unstandardized coefficients that were associated with experiencing preeclampsia during pregnancy.

	Fetal-favoring foods	Foods that were good for both	Maternal-favoring foods	Antimicrobial foods
Cravings	-0.06	0.24	0.11	0.14
Aversions	-0.11	-0.30	0.15	-0.11
Nausea	0.11	0.20	0.06	0.07
Vomiting	-0.94^{+}	0.29	1.48*	-0.45

**Note.* $^{+}$ $p < 0.10$. *, $p < 0.05$. ** $p < 0.01$. *** $p < 0.001$.

APPENDIX F

CHANGES IN EATING BEHAVIORS PREDICTING PREGNANCY COMPLICATIONS CONTROLLING FOR EDUCATIONAL ATTAINMENT

The analyses presented here test the possibility that the likelihood of experiencing pregnancy complications is influenced by participant socioeconomic status, such that participants with higher socioeconomic status are more likely to access to higher quality foods and resources during pregnancy and, therefore, less likely to experience pregnancy complications compared to lower socioeconomic status participants. As a result, the effects of changes in eating behavior on the likelihood of experiencing pregnancy complications may be obscured by socioeconomic status. Since I did not ask participant to report their socioeconomic status, I conducted logistic regressions including education (a proxy for socioeconomic status) as a covariate in addition to the changes in eating behavior to assess this possibility.

First, I investigated which changes in eating behavior were associated with experiencing high blood pressure after controlling for education (see Table S9 for coefficient estimates). Including education as a covariate slightly altered the observed effects. Women who had less education were marginally more likely to experience high blood pressure during pregnancy after controlling for all cravings. Women who reported weaker cravings for fetal-favoring foods were marginally more likely to experience high blood pressure during pregnancy after controlling for education and all other cravings. Women who reported stronger cravings for maternal-favoring foods were more likely to experience high blood pressure after controlling for education and all other cravings. There was no effect of changes in aversions, nausea, and vomiting in response to these food categories on the likelihood of experiencing high blood pressure.

Table S9. Changes in eating behavior unstandardized coefficients that were associated with experiencing high blood pressure during pregnancy.

	Education	Fetal-favoring foods	Foods that were good for both	Maternal-favoring foods	Antimicrobial foods	Probiotic foods
Cravings	-0.23^{+}	-0.35^{+}	0.05	0.44*	0.18	-0.18
Aversions	-0.10	0.21	0.06	-0.18	-0.03	-0.08
Nausea	-0.21	0.08	-0.10	-0.11	-0.17	0.06
Vomiting	-0.25	-0.23	0.13	0.14	0.19	-0.10

**Note*. $^{+}$ $p < 0.10$. *, $p < 0.05$. ** $p < 0.01$. *** $p < 0.001$.

Next, I investigated which changes in eating behaviors were associated with experiencing gestational diabetes after controlling for education (see Table S10 for coefficients estimates). Including education as a covariate did not change the observed results. Women who reported weaker cravings for fetal-favoring foods or stronger aversions towards fetal-favoring foods were marginally more likely to experience gestational diabetes after controlling for education and all other cravings and aversions. Women who reported stronger vomiting in response to fetal-favoring foods were more likely to experience gestational diabetes after controlling for education and all other vomiting. There was no effect of changes in nausea in response to these food categories on the likelihood of experiencing gestational diabetes.

Table S10. Changes in eating behavior unstandardized coefficients that were associated with experiencing gestational diabetes during pregnancy.

	Education	Fetal-favoring foods	Foods that were good for both	Maternal-favoring foods	Antimicrobial foods	Probiotic foods
Cravings	-0.09	-0.49^{+}	-0.04	0.12	0.08	0.03
Aversions	-0.03	0.52^{+}	-0.14	-0.15	-0.43	0.26
Nausea	-0.13	0.32	-0.02	-0.06	-0.34	0.07
Vomiting	0.11	0.83*	-0.55	0.37	-0.99	0.14

**Note*. $^{+}$ $p < 0.10$. *, $p < 0.05$. ** $p < 0.01$. *** $p < 0.001$.

Then, I investigated which changes in eating behaviors were associated with experiencing infections during pregnancy after controlling for education (see Table S11 for coefficient estimates). Including education as a covariate did not change the observed results. Women who reported weaker cravings for foods that are good for both or weaker vomiting in response to maternal-favoring foods were more likely to experience infections during pregnancy after controlling for education and all other cravings and vomiting. There were no effects of changes in aversions or nausea in response to these food categories on the likelihood of experiencing infections during pregnancy.

Table S11. Changes in eating behavior unstandardized coefficients that were associated with experiencing infections during pregnancy.

	Education	Fetal-favoring foods	Foods that were good for both	Maternal-favoring foods	Antimicrobial foods	Probiotic foods
Cravings	0.05	0.35	-0.58*	0.21	0.66	-0.41
Aversions	0.001	0.04	0.16	0.24	-0.46	0.27
Nausea	0.13	-0.14	-0.14	0.21	0.27	0.02
Vomiting	0.12	0.27	0.42	-1.80*	0.51	0.32

**Note*. $^{+}$ $p < 0.10$. *, $p < 0.05$. ** $p < 0.01$. *** $p < 0.001$.

Finally, I investigated which changes in eating behaviors were associated with experiencing preeclampsia during pregnancy after controlling for education (see Table S12 for coefficient estimates). Including education as a covariate did not change the observed results. Women who reported weaker vomiting in response to fetal-favoring foods were more likely to experience preeclampsia during pregnancy after controlling for education and all other vomiting. Women who reported stronger vomiting in response to maternal-favoring foods were more likely to experience preeclampsia during pregnancy after controlling for education and all other vomiting. There were no effects of changes in cravings, aversions, or nausea in response to these food categories on the likelihood of experiencing preeclampsia during pregnancy.

Table S12. Changes in eating behavior unstandardized coefficients that were associated with experiencing preeclampsia during pregnancy.

	Education	Fetal-favoring foods	Foods that were good for both	Maternal-favoring foods	Antimicrobial foods	Probiotic foods
Cravings	-0.14	-0.17	0.21	-0.07	-0.22	-0.15
Aversions	0.11	-0.04	-0.24	0.14	-0.07	0.10
Nausea	-0.07	-0.07	0.14	-0.15	-0.07	-0.08
Vomiting	-0.09	-0.95*	0.16	1.57**	-0.28	-0.13

**Note*. $^{+}$ $p < 0.10$. *, $p < 0.05$. ** $p < 0.01$. *** $p < 0.001$.